

ภาวะหยุดหายใจขณะหลับเนื่องจากทางเดินหายใจอุดตัน: รายงานผู้ป่วย 2 ราย และทบทวนวรรณกรรม

จรุงจิต ไกรวัฒนพงศ์¹

Abstract:

Obstructive sleep apnea syndrome: report of 2 cases and review of the literatures

Kraiwattanapong J.

Department of Otolaryngology Head and Neck Surgery, Faculty of Medicine,

Prince of Songkla University, Hat Yai, Songkhla, 90110, Thailand

E-mail: yjarungj@medicine.psu.ac.th

Songkla Med J 2005;23(2):117-123

Different manifestations of 2 cases who presented with obstructive sleep apnea syndrome (OSAS) are reported. The first case had minimal symptoms of OSAS, the second case was more severe. Standard overnight polysomnography (PSG) identified OSAS in both patients. Clinical diagnosis and choices of treatment were discussed. We suggest that OSAS should be considered in all snoring patients.

Key words: *Obstructive sleep apnea syndrome, OSAS, presentation, case report, literatures review*

¹M.D. Diplomate Thai Board of Otolaryngology Head and Neck Surgery, Certificate of Fellowship in Sleep Medicine, Lecturer, Department of Otolaryngology Head and Neck Surgery, Faculty of Medicine, Prince of Songkla University, Hat Yai, Songkhla, 90110, Thailand

รับต้นฉบับวันที่ 20 กรกฎาคม 2547 รับลงตีพิมพ์วันที่ 28 มีนาคม 2548

บทคัดย่อ:

ภาวะหยุดหายใจขณะหลับเป็นภาวะที่เกิดจากทางเดินหายใจอุดกั้นเป็นช่วง ๆ ตลอดทั้งคืน ทางเดินหายใจที่อุดกั้นเป็นได้ทั้งอุดกั้นบางส่วนหรืออุดกั้นทั้งหมด ซึ่งส่งผลให้ระดับออกซิเจนในกระแสเลือดลดลงทำให้สมองถูกกระตุ้น (arousal or awaken) ตลอดการนอนหลับ ผู้ป่วยจะมีอาการนอนหลับไม่เพียงพอ มีง่วงนอนในเวลากลางวัน สมาธิและความจำลดลง และมีอาการเหนื่อยง่าย ถ้ามีอาการรุนแรงผู้ป่วยอาจประสบอุบัติเหตุหรือได้รับอันตรายจากการพลอหลับระหว่างปฏิบัติหน้าที่ เช่น ขับรถ ใช้เครื่องจักรได้ ผู้แต่งรายงานภาวะหยุดหายใจขณะหลับเนื่องจากทางเดินหายใจอุดกั้นในผู้ป่วย 2 รายที่มีความรุนแรงของอาการต่างกัน และแนะนำว่า ภาวะหยุดหายใจขณะหลับเนื่องจากทางเดินหายใจอุดกั้น สามารถพบได้แม้ในผู้ป่วยที่มาด้วยปัญหาอนกรนอนกรนอย่างเดียว แพทย์ควรนึกถึงภาวะดังกล่าวร่วมด้วยในผู้ป่วยที่นอนกรนทุกคน

คำสำคัญ: ภาวะหยุดหายใจขณะหลับ, อาการ, รายงานผู้ป่วย, ทบทวนวรรณกรรม

Introduction

Obstructive sleep apnea syndrome (OSAS) is a clinical condition, with recognizable symptoms, that occurs because the upper airway collapses intermittently and repeatedly during sleep. This collapse can be complete or partial, resulting in oxygen desaturation and arousal from sleep. Repeated episodes of oxygen desaturation and arousal throughout a whole night causes unrefreshing sleep, excessive daytime sleepiness, poor concentration and becoming easily fatigued.¹⁻³ Long-term sleep apnea is associated with hypertension, myocardial infarction, congestive heart failure, cardiac arrhythmia, stroke and increased mortality.⁴⁻¹³

Prominent characteristics of OSAS patients are excessive daytime sleepiness, impaired concentration and snoring; other characteristics include unrefreshing sleep, choking episodes during sleep, witnessed apnea, restless sleep, irritability, personality change, nocturia and decreased libido.^{3, 14, 15} Patients with symptom of excessive daytime sleepiness, impaired concentration and snoring should be differentially diagnosed from other sleep disorders such as fragmented sleep, sleep deprivation, shift work, depression, narcolepsy, hypothyroidism, excess alcohol consumption and misuse of hypnotic drugs.³ Pertinent physical findings of OSAS patients are mainly overweight or obesity (body mass index (BMI) of 25-29.9 and >30, respectively), mean neck circumference of >17 inches (43 cm)³, and long and redundant of soft palate and uvula, enlarged tonsils, and enlarged base of the tongue evaluated by using Mallampati score. A dynamic evaluation of pharyngeal

collapse is done by Muller maneuver, which the patient tries to breathe in with nose and mouth close (reverse valsalva maneuver). Severity of daytime sleepiness is scaled using Epworth sleepiness scale, in which score of more than 10 is considered excessive daytime sleepiness.^{16, 17} Too gold standard for diagnosis of OSAS is polysomnography, an overnight sleep study to define episode of obstructive sleep apnea/hypopnea.¹⁸⁻²³ A score of more than 5 episodes of apnea and hypopnea per hour is diagnosed as OSAS.¹⁻³

Obstructive sleep apnea syndrome is new in the medical field and not known by a lot of general practitioners. The estimated prevalence of OSAS was 9 percent for women and 24 percent for men²⁴. We describe 2 cases who presented with different manifestations of obstructive sleep apnea. Clinical diagnosis and choices of treatment were discussed. We suggest that obstructive sleep apnea should be considered in all snoring patients.

Materials and methods

Physical examination

The physical examinations, including both a general examination and a specific ENT examination, were given at the ENT sleep clinic. The specific ENT examination includes anterior rhinoscopy, posterior rhinoscopy and indirect laryngoscopy, and is highlighted, performing Muller maneuver, using fiberoptic nasopharyngoscopy, while awake in an upright position.^{15, 25-27}

Sleep studies

Standard overnight polysomnography (Biologic System Corporation) was carried out using a full sleep study apparatus including electroencephalogram (EEG), electrooculogram (EOG), electromyogram (EMG at chin muscle and extremities muscles), electrocardiogram (ECG), pulse oximeter, nasal air flow, chest and abdominal wall movement, sleep position and snoring. Scoring of sleep stages were referred to Rechtschaffen and Kales criteria. Apnea is scored when there is cessation of nasal air flow lasting at least 10 seconds and obstructive sleep apnea (OSA) is scored when an apnea episode was encountered with attempted chest or abdominal respiration. Hypopnea is scored when an at least of 10 seconds of reduction in air flow by less than 50%. Diagnosis of OSAS is defined as an apnea/hypopnea index (AHI) of more than five events per hour. The AHI is the sum of the number of apneas plus the number of hypopneas per hour. An AHI of 5–14/h is classed as mild OSAS, 15–30 is moderate, and more than 30 is severe.^{2,3}

Consent

Appropriate informed consent was obtained from both patients at each stage of investigation and treatment.

Case reports

Case 1

A 57-yr-old overweight man (BMI 25.3) presented with a long history of snoring which had been increasing in loudness over the past 2 years. He had no history of witnessed apnea, morning headaches or falling asleep while driving. His daytime functioning was fair, with some feeling of afternoon sleepiness. The loudness of his snoring sometimes woke him up during the night, and caused distress to his wife. The loudness of the snoring had originally been associated with a supine position, but progressing to all positions during the past 2 years. He had no underlying diseases. His blood pressure was within normal limits. He smoked 7–8 cigarettes per day but did not drink. He had gained 1 kg of weight the previous year. Details of his demographic data and physical examination are shown in Tables 1 and 2 respectively. An overnight PSG identified OSAS with an apnea-hypopnea index (AHI) of 21.5 events/hour (Table 3).

Table 1 Demographic data of OSAS case history patients

General data	Case 1	Case 2
Gender	male	male
Age	57	45
BMI (kg/m ²)	25.3	29.24
Blood pressure (mmHg) at presentation	118/65	135/100
Neck circumference (cm.)	37 (14.8")	36 (14.4")
Waist circumference (cm.)	88.5 (35.4")	95.5 (38.2")
Epworth sleepiness scale	12/24	21/24

Table 2 ENT examination data of OSAS case history patients

ENT examination	Case 1	Case 2
<i>Nasal cavities</i>	right deviated nasal septum	<ul style="list-style-type: none"> ● bulging of both sides of nasal septum ● marked narrowing of nasal passages ● inferior turbinate hypertrophy ● small nasal polyps with mucopurulent discharge
<i>Nasopharyngeal space</i>	↓ AP dimension	↓ AP & lateral dimension
<i>Oral cavity & Oropharyngeal space</i>		
- Mallampati score	class 3	class 4
- uvula	elongated	hypertrophy
- soft palate	elongated	not elongated
- posterior pillar	web	narrow space
- tonsils	grade 2	grade 2
- base of the tongue	not enlarged	enlarged
<i>Hypopharynx & Larynx</i>	partially visible	not visible
<i>Muller maneuver</i>	collapse grade III (75%) AP > lateral dimension	collapse grade III (99%) AP = lateral dimension

Remark: AP=anterior-posterior, elongated uvular (>1 cm), elongated soft palate (>5 cm), posterior pillar web (>5 mm, wide)

Case 2

A 45-yr-old man presented with 10+ years of unrefreshing sleep. He had very loud snoring, a history of choking and gasping during sleep and witnessed apnea in all sleeping positions. He had no morning headache but usually felt too tired to get up in the morning. He experienced sleepiness within 2 hours after having breakfast and the symptoms persisted throughout the day. He had frequent episodes of dozing off while driving but had never had an accident. He sometimes had a social drink and had gained 8-9 kg after quitting smoking 2 years previously. At the time of initial evaluation, his blood pressure was 135/100 mmHg (repeated), but he had no previous diagnosis of hypertension. There was no history of allergy but the physical examination revealed signs of allergic rhinitis, nasal polyps and sinusitis (Table 2). He had an AHI of 72.2 events/hour from an overnight PSG (Table 3).

Table 3 Summarized polysomnographic data*

	Case 1	Case 2
Total sleep time (TST) (hours)	6.7	6.5
Sleep efficiency (%)	84.2	81.6
REM stage (%TST)	30.1	59.1
Apnea Total (events/hour)	20.9	68.1
Central (events/hour)	0.2	0.9
Obstruction (events/hour)	20.0	60.3
Mixed (events/hour)	0.3	7.1
Hypopnea (events/hour)	0.6	3.9
AHI (events/hour)	21.5	72.2
O₂ Baseline (%)	95.8	91.1
Average (%)	96	88
Lowest (%)	83	56
Desaturation index (events/hour)	17.6	66.7
HR Baseline (/minute)	60	59.3
Average (/minute)	51.8	56.6
Maximum (/minute)	85	87
Minimum (/minute)	36	31

* Manually scored and automatically calculated by polysomnography computer software.

Discussion

The patients described here presented with different clinical features suggestive of snoring and OSAS. Case 1 described only episodes of progressive loud snoring that woke him up during the night and some feeling of sleepiness during the day, while case 2 had overt symptoms of OSAS such as choking and gasping during sleep, morning headache, excessive daytime sleepiness and tiredness throughout the day.

It is noteworthy that in both of our patients, standard overnight polysomnography was essential for diagnosis of OSAS. According to the American Academy of Sleep Medicine (AASM) Task Force for diagnosis of OSAS, sleep study or polysomnography is currently the gold standard for diagnosis of OSAS. Diagnostic criteria consist of criterion A or B, plus criterion C²:

- A. Excessive daytime sleepiness that is not better explained by other factors.
- B. Two or more of the following that are not better explained by other factors:
 - Choking or gasping during sleep
 - Recurrent awakenings from sleep
 - Unrefreshing sleep
 - Daytime fatigue
 - Impaired concentration
- C. Overnight monitoring demonstrates 5 or more obstructed breathing events per hour during sleep.

In case 1, the hypnogram showed sleep fragmentation occurred mainly in non-rapid eye movement (NREM) sleep rather than in rapid eye movement (REM) sleep. Respiratory events of apnea and hypopnea related to oxygen desaturation and drop of heart rate were presented in NREM sleep, especially in a supine sleeping position (Figure 1). Apnea episodes of 20.9 events/hour in case 1 were caused by obstruction in 20 events/hour (Table 3). The severity of OSAS was classified as moderate severity (AHI 15-30), based on the AASM Task Force's severity criteria.² The case 2 hypnogram showed marked sleep fragmentation occurred in both REM and NREM sleep during the first half of the night. The patient had resumed more

REM sleep during the second half of the night from using 2 pillows to support his head, but oxygen desaturation events and drop of heart rate were going on throughout the whole night. The patient slept in almost a supine position (Figure 2). He had 60.3 obstructive sleep events out of 68.1 total sleep apnea events. The Apnea-Hypopnea Index of 72.2 events/hour demonstrated severe OSAS (AHI>30). Oxygen

desaturation occurred during every single minute of sleeping (desaturation index = 66.7 events/hour), with average and lowest oxygen saturation of 88% and 56% respectively (Table 3). Long-standing oxygen desaturation is strongly correlated with systemic hypertension, which can lead to ischemic heart disease, myocardial infarction, congestive heart failure, cardiac arrhythmia, stroke and increased mortality.⁴⁻¹³

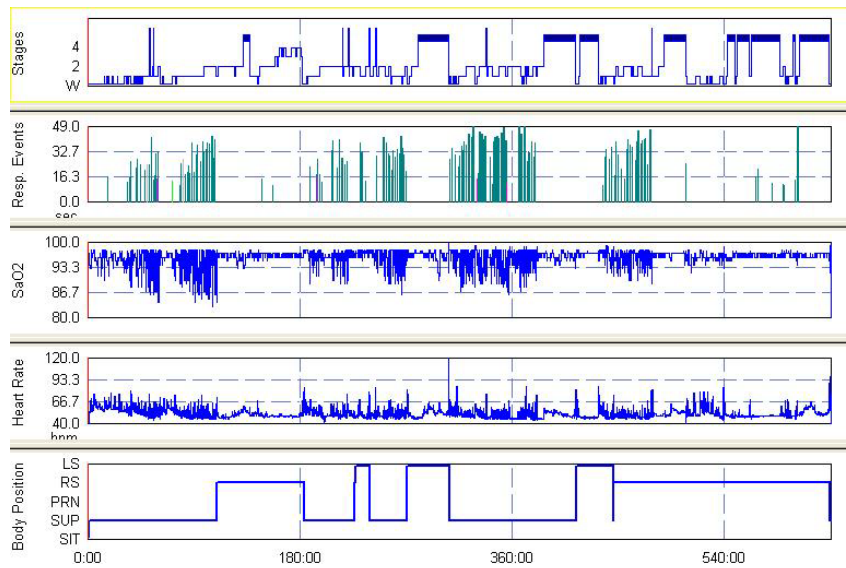


Figure 1 Case 1 hypnogram shows sleep stages, respiratory events (apnea+hypopnea), pulse oxygen saturation, ECG heart rate and body position during actual sleeping hours in the sleep lab

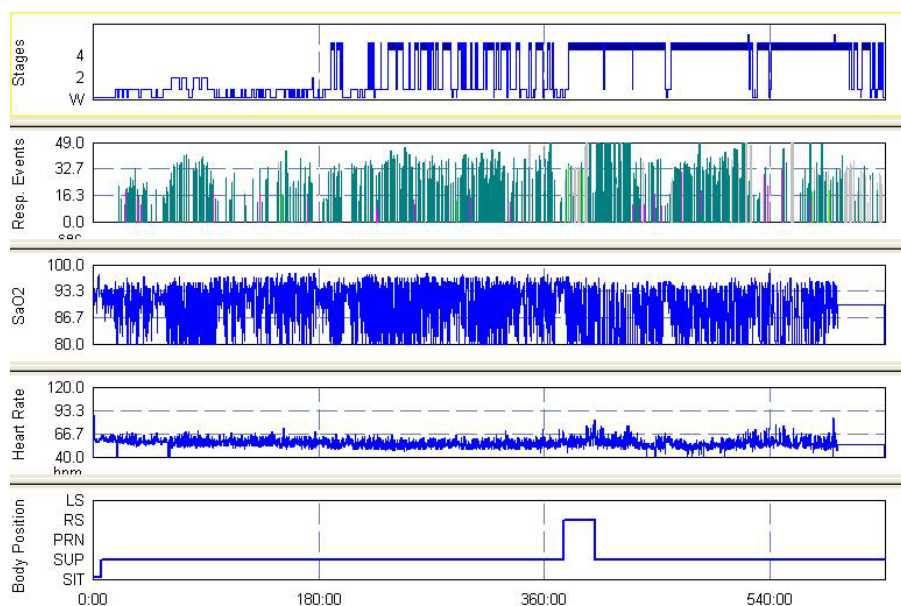


Figure 2 Case 2 hypnogram shows sleep stages, respiratory events (apnea+hypopnea), pulse oxygen saturation, ECG heart rate and body position during actual sleeping hours in the sleep lab

Factors predisposing to apnea and hypopnea^{2, 15, 23, 24} include:

- increasing age
- male gender
- obesity, particularly upper body adiposity
- sedative drugs
- smoking and alcohol consumption
- craniofacial abnormalities, including mandibulo/maxillary hypoplasia
- increased pharyngeal soft or lymphoid tissue, including tonsillar hypertrophy
- nasal obstruction
- endocrine abnormalities: hypothyroidism, acromegaly
- familial history

Treatment of OSAS consists of non-surgical and surgical treatment. Reducing weight, adjusting sleep hygiene and modifying sleep position are advised to most of the patients as a non-surgical treatment option. Nasal continuous positive airway pressure (nCPAP) machine and surgical treatment options such as tonsillectomy and uvulopalatopharyngoplasty (UPPP), uvulopalatal flap and radio-frequency volume tissue reduction (RFVTR) are considered individually. Most of the cases require a combination of treatments.

Both of our cases had several predisposing factors of OSAS, notably increasing age, male gender, narrow nasal air passage and increased pharyngeal soft tissue. Additional factors included smoking in case 1 and very close to obesity (BMI>30) in case 2. According to the ENT examination evaluation, the first patient might have benefited from surgical treatment if he refused CPAP treatment, whereas the only treatment for case 2 was CPAP with or without surgery. Also, sleep hygiene was recommended for both patients, such as stopping smoking (case 1), reducing and controlling weight and avoiding a supine position (i.e. by using the tennis ball technique). The treatment of allergic rhinitis, nasal polyps and sinusitis in case 2 was recommended to help open up the nasal air passage, making compliance with the nasal CPAP treatment easier and more effective.

Conclusion

Patients with obstructive sleep apnea can present with different manifestations, which may be as mild symptom as snoring. There are some points from the history and physical examination that can help the clinician suspect this disease. We suggest that OSAS should be considered in all snoring patients.

References

1. Guilleminault C, Tilkian A, Dement W. The sleep apnea syndrome. *Ann Rev Med* 1976;27:465-84.
2. American Academy of Sleep Medicine Task Force. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. *Sleep* 1999;22:667-89.
3. Scottish Intercollegiate Guidelines Network. Management of obstructive sleep apnoea/hypopnoea syndrome in adults: A National Clinical Guideline 2003:1-38.
4. Milleron O, Pillire R, Foucher A, Roquefeuil F, Aegerter P, Jondeau G, et al. Benefits of obstructive sleep apnea treatment in coronary artery disease: a long-term follow-up study. *Eur Heart J* 2004;25:728-34.
5. Andreas S, Schulz R, Werner GS, Kreuzer H. Prevalence of obstructive sleep apnea in patients with coronary artery disease. *Coron Artery Dis* 1996;7:541-5.
6. Peker Y, Hedner J, Kraiczi H, Loth S. Respiratory disturbance index: an independent predictor of mortality in coronary artery disease. *Am J Respir Crit Care Med* 2000;162:81-6.
7. Schafer H, Koehler U, Ewig S, Hasper E, Tasci S, Luderitz B. Obstructive sleep apnea as a risk marker in coronary artery disease. *Cardiology* 1999;92:79-84.
8. Turkington PM, Allgar V, Bamford J, Wanklyn P, Elliott MW. Effect of upper airway obstruction in acute stroke on functional outcome at 6 months. *Thorax* 2004;59:367-71.
9. Davies DP, Rodgers H, Walshaw D, James OF, Gibson GJ. Snoring, daytime sleepiness and stroke: a case-control study of first-ever stroke. *J Sleep Res* 2003;12:313-8.

10. Palomaki H. Snoring and the risk of ischemic brain infarction. *Stroke* 1991;22:1021-5.
11. Yaggi H, Mohsenin V. Obstructive sleep apnoea and stroke. *Lancet Neurol* 2004;3:333-42.
12. Pearce S, Saunders P. Obstructive sleep apnoea can directly cause death. *Thorax* 2003;58:369.
13. Marti S, Sampol G, Muñoz X, Torres F, Roca A, Lloberes P, et al. Mortality in severe sleep apnoea/hypopnoea syndrome patients: impact of treatment. *Eur Respir J* 2002;20:1511-18.
14. Monasterio C VS, Duran J, Ferrer M, Carmona C, Barbe F, et al. Effectiveness of continuous positive airway pressure in mild sleep apnea-hypopnea syndrome. *Am J Respir Crit Care Med* 2001;164:939-43.
15. Barnes M, Houston D, Worsnop CJ, Neill AM, Mykytyn IJ, Kay A, et al. A randomized controlled trial of continuous positive airway pressure in mild obstructive sleep apnea. *Am J Respir Crit Care Med* 2002;165:773-80.
16. Johns MW. Daytime sleepiness, snoring, and obstructive sleep apnea. The Epworth Sleepiness Scale. *Chest* 1993;103:30-6.
17. Uribe Echevarria EM, Alvarez D, Giobellina R, Uribe Echevarria AM. Epworth drowsiness scale value in obstructive sleep apnea syndrome. *Medicina* 2000;60:902-6.
18. Bennett LS, Stradling JR, Davies RJ. A behavioural test to assess daytime sleepiness in obstructive sleep apnoea. *J Sleep Res* 1997;6:142-5.
19. Bradley PA, Mortimore IL, Douglas NJ. Comparison of polysomnography with ResCare Autoset in the diagnosis of the sleep apnoea/hypopnoea syndrome. *Thorax* 1995;50:1201-3.
20. Whittle AT, Finch SP, Mortimore IL, Mackay TW, Douglas NJ. Use of home sleep studies for the diagnosis of the sleep apnoea/hypopnoea syndrome. *Thorax* 1997;52:1068-73.
21. Stradling J, Davies RJ. Is it necessary to record sleep? *Sleep* 1996;19:S251-4.
22. Chesson A, Jr., Ferber R, Fry JM, Grigg-Damberger MM, Hartse K, Hurwitz TD, et al. The indications for polysomnography and related procedures. *Sleep* 1997;20:423-87.
23. Douglas NJ, Thomas S, Jan MA. Clinical value of polysomnography. *Lancet* 1992;339:347-50.
24. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The Occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med* 1993;328:1230-5.
25. Shepard Jr. JW, Gefter WB, Guilleminault C, Hoffman EA, Hoffstein V, Hudgel DW, et al. Evaluation of the upper airway in patients with obstructive sleep apnea. *Sleep* 1991;14:361-71.
26. Pepin JL, Levy P, Veale D, Ferretti G. Evaluation of the upper airway in sleep apnea syndrome. *Sleep* 1992;15:S50-5.
27. Shepard JW Jr, Thawley SE. Localization of upper airway collapse during sleep in patients with obstructive sleep apnea. *Am Rev Respir Dis* 1990;141:1350-5.