CASE REPORT

An Overweight Woman with Profound Symptomatic Nocturnal Bradycardia: A Case Report

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Case Report: An overweight 48-year-old Thai female presented with syncope while she was lying down and watching television. Telemonitoring during she was unconscious demonstrated that there was long pause due to sinus arrest without escape rhythm. She was inserted with temporary transvenous pacemaker because of profound symptomatic bradycardia during nighttime. She was diagnosed as syncope due to nocturnal bradyarrhythmia. The sleep apnea syndrome (SAS) related bradycardia was the most likely diagnosis. Type 1 polysomnography was performed and compatible with moderate obstructive sleep apnea (OSA). The patient received continuous positive airway pressure (CPAP) for treatment of moderate OSA and remained no syncope or long pause during one year follow-up period.

Conclusion: This was an unusual case of an overweight woman presenting with profound symptomatic nocturnal bradycardia due to moderate OSA treated with CPAP. Patients presenting with nocturnal bradycardia should always be investigated SAS related bradycardia.

Keywords: Nocturnal bradycardia; Overweight; Obstructive sleep apnea; Sleep disordered breathing; Polysomnography

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Nocturnal bradyarrhythmias are common in clinical practice. Most of the patients have vagal-mediated asymptomatic bradycardias[1]. These conditions are common in the young and in the athlete[2]. Sinus bradycardia, sinus pauses, first-degree and Mobitz I second-degree atrioventricular block are the common bradyarrhythmias in sleep-disordered breathing (SDB)[3].

Obstructive sleep apnea (OSA) is common in patients with SDB. Its prevalence is 11.4% and most of patients with OSA are obese in Thailand[4]. The authors report an unusual case of an overweight patient presenting with profound symptomatic nocturnal bradycardia.

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Case Report

A 48-year-old Thai female presented with transient loss of consciousness while she was lying down and watching television. She had no underlying disease. She was sent to a private hospital for investigating causes of transient loss of consciousness. The physical examination was unremarkable except overweight (her height, body weight, and body mass index were 160 cm, 62 kg, and 24.22 kg/m^2, respectively). The electrocardiography and chest radiography were performed and appeared to be normal. Brain magnetic resonance imaging appeared unremarkable. She was monitored for cardiac arrhythmia. The telemonitoring during she was unconscious was shown in Figure 1. The provisional diagnosis was syncope associated with long pause due to sinus arrest without escape rhythm. She was referred to the authors' hospital for further investigation and management.

She was transferred to the cardiac care unit (CCU) and inserted with temporary transvenous pacemaker because of profound symptomatic bradycardia during nighttime. The chest radiograph showed the good position of temporary transvenous pacing lead.

She had never been prescribed any heart rate slowing medication. She had no history of pain or diarrhea before hospital admission. The

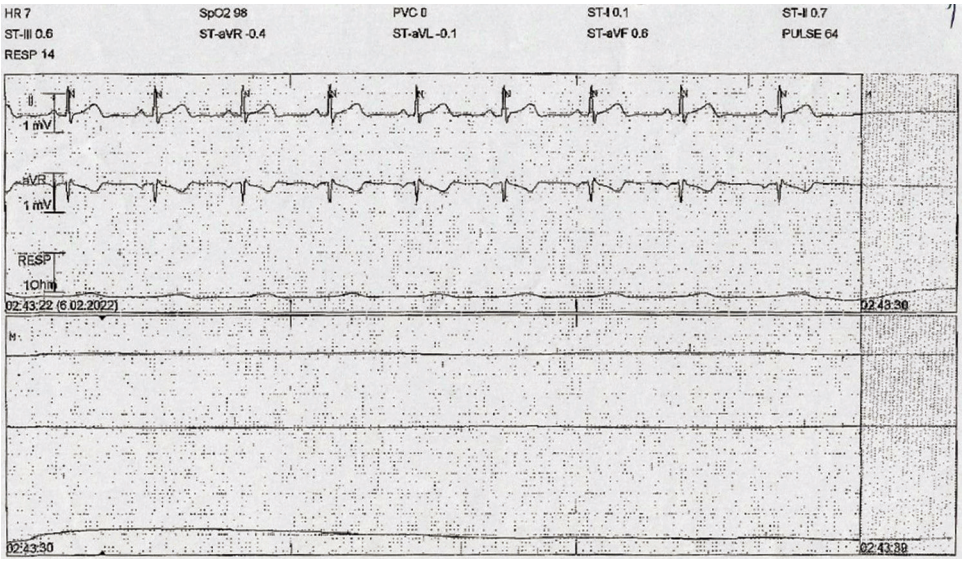

Figure 1. Telemonitoring demonstrating long pause due to sinus arrest without escape rhythm during the patient was unconscious.

results of blood testing including renal function, electrolyte and thyroid function test were normal. The echocardiography was performed and illustrated good left ventricular function, normal wall motion, no significant valvular dysfunction and no pericardial effusion. She was diagnosed as syncope due to nocturnal bradyarrhythmia. The sleep apnea syndrome (SAS) related bradycardia was the most likely diagnosis.

Sleep medicine specialist was consulted for investigating and confirming SAS. A screening history before performing polysomnography (PSG) showed that she had a history of snoring, choking during sleep and excessive daytime sleepiness. The patient used the continuous positive airway pressure (CPAP) during awaiting examination of PSG. There was no long pause after CPAP used. The type 3 PSG was performed at CCU. The result of type 3 PSG demonstrated that respiratory event index (REI) was within normal limit (REI of 3.8 events/hour) while there was significant oxygen desaturation (the lowest oxygen saturation of 89%). The type 1 PSG was recommended for confirming SAS. The temporary transvenous pacemaker was removed. The patient got CPAP during sleep at home and was scheduled for type 1 PSG.

Before type 1 PSG examination, the Epworth Sleepiness Scale was evaluated in this patient for assessment of daytime sleepiness and showed that she had a scale of 14 (score of 11 or more is interpreted as excessive daytime sleepiness). The facial profile, nasal cavity, oral mucosa including tongue, hard and soft palate were normal. She had modified Mallampati (Friedman tongue) grade 2 (higher grade is associated with the presence and severity of OSA). The result of type 1 PSG illustrated that apnea-hypopnea index was 26.0 events/hour aggravating to 42.6 events/hour during rapid eye movement (REM) sleep with significant oxygen desaturation (the lowest oxygen saturation of 89%). Moderate OSA was diagnosed.

The patient received CPAP for treatment of moderate OSA and remained no syncope or long pause during one year follow-up period.

Discussion

The present case was an unusual case of the patient presenting with profound symptomatic nocturnal bradycardia during sleep. The SAS-related bradycardia was the most likely diagnosis. OSA is common in patients with SDB and related to obese patients. However, this patient was overweight according to World Health Organization refining obesity criteria in Asia-Pacific region[5]. The anatomic OSA risk factors including craniofacial morphology and patterns of adiposity tends to increase a risk of OSA in this overweight patient[3].

To date, available data have shown that Asian OSA patients predominantly have the inferior position of the hyoid bone, retropositioning of the mandible, a small cranial base and increased craniocervical extension angle leading to have more severe OSA at lower level of obesity compared with Caucasian patients[6].

The key mechanism of SAS-related bradycardia

in this patient is SAS-induced hypoxemia leading to an increase vagal tone. However, previous study has shown that split-night positive airway pressure (PAP) titration PSG could significantly reduce isolated premature ventricular complexes and slower heart rate in OSA patients[7]. REM sleep-related bradycardia is another rare mechanism, unrelated to apnea[1]. The abnormal autonomic nervous system including vagal tone exaggeration and acute sympathetic activity withdrawal during phasic REM events may be the possible mechanism[8]. SAS-induced hypoxemia was the most likely mechanism of SAS-related bradycardia in this patient because she had no symptoms and no profound symptomatic bradycardia after CPAP use, despite there was no significant arrhythmia detection during PSG while REM sleep-related bradycardia should not be treated with CPAP.

After this patient has regularly continued using CPAP for one year, she has remained no symptom and no detection of profound symptomatic bradycardia. This supported the diagnosis in this patient.

Conclusion

This was an unusual case of an overweight woman presenting with profound symptomatic nocturnal bradycardia due to moderate OSA treated with CPAP. Patients presenting with nocturnal bradycardia should be always investigated SAS related bradycardia.

What is already known on this topic?

Vagal-mediated asymptomatic bradycardias are a common cause of nocturnal bradyarrhythmias in obese patients with OSA. The key mechanism of SAS-related bradycardia is SAS-induced hypoxemia leading to an increase vagal tone and REM sleep-related bradycardia.

What this study adds?

Profound symptomatic bradycardia was detected in an overweight patient with OSA treated with CPAP. SAS-induced hypoxemia was the most likely mechanism of SAS-related bradycardia in this patient.

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Conflicts of interest

The authors have nothing to disclose.

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