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CASE REPORT

Asystole Due to Vagal Reflex in a Patient with Obstructive Sleep Apnea during Anesthesia Intubation with Laryngoscope

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Obstructive sleep apnea (OSA) is a serious sleep disorder. The complications of OSA are respiratory and cardiovascular events, including bradycardia, tachycardia, and even cardiac arrest. A 57-year-old female with OSA was vulnerable to vagal stimulation, developing severe bradycardia and asystole during general anesthesia while undergoing intubation with a conventional direct laryngoscope. This asystole case highlights the fact that anesthetized patients with OSA may experience increased parasympathetic activity (vagal tone) and vagal stimulation with consequent severe bradycardia and asystole. Atropine is recommended to resolve such conditions.

Key words: Asystole, bradycardia, direct laryngoscope, obstructive sleep apnea, vagal reflex

INTRODUCTION

Patients with obstructive sleep apnea (OSA) experience increased sympathetic activity; however, the vagal tone decreases during sleep.[1] OSA is associated with life-threatening arrhythmias, conduction disorders, severe bradycardia, and sudden cardiac arrest during sleep.[1] We describe a patient with OSA who, during general anesthesia with conventional direct laryngoscopic endotracheal intubation, experienced increased vagal tone with consequent life-threatening bradycardia, and asystole. She also developed severe bradycardia due to the vagal stimulation during transesophageal echocardiography insertion (TEE) and colonoscopy with air insufflation. Furthermore, bradycardia occurred without vagal stimulation while undergoing polysomnography examination during sleep. These events indicate that patients with OSA tend to have high vagal tone and are vulnerable to vagal stimulation.

CASE REPORT

A 57-year-old Asian female (weight, 70 kg; height, 152 cm; body mass index, 30.3 kg/m^2) underwent a modified radical mastectomy (MRM) for breast cancer. The patient had severe snoring for 11 years and was not administered medical treatment to alleviate the problem. Before anesthesia induction, her arterial blood pressure (ABP) was 158/96 mmHg and her heart rate (HR), 99 beats/min (BPM). General anesthesia was induced with fentanyl (100 μg), lidocaine (60 mg), propofol (100 [60 + 40] mg), and cisatracurium (10 [4 + 6] mg). Routine conventional direct laryngoscope and endotracheal intubation were performed. General anesthesia was initiated with 1% sevoflurane with 100% oxygen at a flow rate of 1 L/min. Approximately 30 s after intubation, the HR dropped to 50 BPM. Ephedrine (10 mg) was administered; however, the HR continued to decrease to 20 BPM, followed by asystole within 1 min [Figure 1]. Cardiopulmonary resuscitation was performed for 10 s, and spontaneous circulation resumed (BP, 100/60 mmHg and HR, 85 BPM). Arterial blood gas and blood biochemistry were assessed within 5 min; all values were within the normal ranges, apart from pH (7.373), $PaCO_2$ (51.2 mmHg), and $HCO3^-$ (30.1 mmol/L) [Table 1]. To

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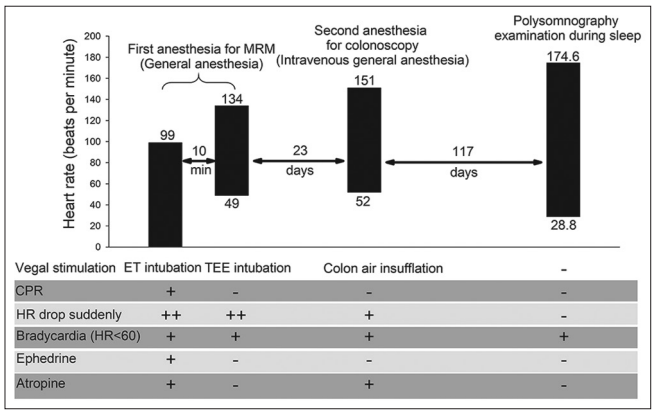

Figure 1: Bradycardia and/or asystole occurred during anesthesia and polysomnography. Vagal stimulation with endotracheal intubation with a conventional direct laryngoscope, transesophageal echocardiography insertion, and colonoscopy with air insufflation resulted in severe bradycardia and cardiac arrest during anesthesia. The patient also developed bradycardia while undergoing polysomnography during sleep. CPR = Cardiopulmonary resuscitation; HR = Heart rate; MRM = Modified radical mastectomy

Table 1: Arterial blood gas and chemistry measurements within 5 min after cardiopulmonary resuscitation and return of spontaneous circulation

Arterial blood gas		Blood chemistry	
FiO_2 (%)	100	Na^+ (mmol/L)	140.3
pH	7.373	K^+ (mmol/L)	3.51
pCO_2 (mmHg)	51.2	Ca^{++} (mmol/L)	1.13
pO_2 (mmHg)	403.2	Mg^{++} (mmol/L)	0.41
SO_2 (%)	99.9	Glucose (mg/dL)	223
HcT (%)	37	Lactate (mmol/L)	1.2
Hb (g/dL)	12.4		
$HCO3^-$ (mmol/L)	30.1		
BE (mmol/L)	4.4		

HcT=Hematocrit; Hb=Hemoglobin; BE=Base excess

exclude cardiogenic shock, TEE was performed; bradycardia subsequently recurred with a nadir HR of 49 BPM [Figure 1]. Therefore, TEE was terminated and the HR gradually increased to 80 BPM. Subsequently, TEE was performed smoothly and revealed normal heart function. MRM was then initiated. Ten minutes after the skin incision, the HR dropped from 80 to 60 BPM within 1 min. To prevent a continuous drop in HR, ephedrine (5 mg) was administered but without success. Therefore, atropine (0.5 mg) was administered intravenously; this restored her ABP and HR to 130-170/70-90 mmHg and 90–110 BPM, respectively. After MRM, the cardiologist was consulted, who suggested echocardiography; this revealed no specific abnormalities. Coronary angiography and Thallium-201 myocardial perfusion imaging were not suggested. Holter monitoring of the cardiac rhythm was also not suggested, as the scheduled polysomnography would record a continuous cardiac rhythm for at least 8 h.

After 25 days, colonoscopy was performed under intravenous general anesthesia. Before anesthesia induction, 0.9% sodium chloride (250 mL) was administered, followed by fentanyl (50 μg) and propofol (40 mg), with slow-pump intravenous infusion over 2 min. Colonoscopy commenced with air insufflation; however, 3 min later, her HR decreased from 99 to 52 BPM [Figure 1]. Atropine (0.5 mg) restored the HR to 90-99 BPM.

Polysomnography was performed 117 days after the colonoscopy. According to the American Academy of Sleep Medicine criteria, the apnea-hypopnea index (AHI) is calculated according to the number of apnea (hypopnea) episodes per hour, with an AHI >30 classified as severe OSA. The patient's AHI was 102.1, indicating very severe OSA. During the rapid eye movement stage of the test, her HR decreased to 28.8 BPM [Table 2]. Thereafter, she was prescribed continuous positive airway pressure during sleep for the management of OSA. She currently has good quality of life and is able to perform her activities of daily living with no evidence of cardiovascular problems. This case report was approved by the institutional review board of Tri-Service General Hospital (TSGHIRB No.: C202105096).

DISCUSSION

The possible explanations for the high vagal tone and low sympathetic tone before vagal stimulation are as follows. First, anesthesia interferes with the sympathetic neural outflow and cardiovascular regulation.[2] These effects are more pronounced in states of chronically elevated sympathetic activity,[2] such as OSA. Second, fentanyl may increase the vagal tone, and propofol can inhibit the sympathetic tone.[3] Third, OSA increases the vagal tone during sleep, which may result in life-threatening nocturnal bradycardia and paroxysmal asystole[1] as evidenced by the extremely low HR of 28.8 BPM assessed by polysomnography during sleep in our patient.

High vagal stimulation resulting from endotracheal intubation, TEE insertion, and colonoscopy with air insufflation all resulted in bradycardia in this patient. First, during general anesthesia, in addition to anesthetic agents, vagal stimulation from the intubation resulted in a sudden decrease in HR with no response to atropine or ephedrine. This resulted in asystole requiring cardiopulmonary resuscitation. Second, with vagal stimulation due to the TEE insertion, the HR decreased suddenly but responded to atropine. Third, during the colonoscopy with air insufflation under intravenous general anesthesia, the patient's HR decreased suddenly (with a response to atropine). In view of these experiences, we

conclude that this patient was vulnerable to vagal stimulation. Treatment using atropine, and possibly cardiopulmonary resuscitation, should be considered in similar situations of severe bradycardia or asystole [Figure 1].

This case should motivate surgeons and anesthesiologists to identify OSA in their patients preoperatively. This patient had a long history (11 years) of severe snoring. However, she was not diagnosed with OSA preoperatively. Singh *et al*. reported that, of 267 patients with moderate-to-severe OSA, 92% (n = 245) and 60% (n = 159) remained undiagnosed by surgeons and anesthesiologists, respectively.[4] If polysomnography is not available preoperatively, the STOP-Bang questionnaire and serum HCO^{3-} levels can be valuable tools for identifying OSA.[5] This patient's STOP-Bang score of 5 indicated a high risk of OSA [Table 3].[5] Immediately after cardiopulmonary resuscitation, arterial blood gas assessment revealed an HCO^{3-} of 30.1 mmol/L.

Table 2: Polysomnography report of apnea-hypopnea (a), bradycardia and tachycardia (b) data

	Number of events	Mean duration (s)	Maximum duration (s)	Total duration (min)
A. Apnea-hypopnea				
Obstructive	561	19.3	46.2	180.0
Central	0	-	-	0.0
Mixed	0	-	-	0.0
Hypopneas	148	17.2	36.3	42.3
Total	709	18.8	46.2	222.3
	REM + NREM		REM	NREM
AHI (#/h)	102.1		92.4	103.5
	Awake		REM	NREM
B. Bradycardia and tachycardia				
Mean heart rate (bpm)	80.6		83.0	78.3
Lowest heart rate (bpm)	29.8		28.8	36.8
Highest heart rate (bpm)	145.8		140.5	123.9

AHI=Apnea-hypopnea index; #/hour=Apnea and hypopnea events per hour of sleep; REM=Rapid eye movement; NREM=Nonrapid eye movement; BPM=Beats per minute

Table 3: STOP-Bang scores and predicted probabilities of obstructive sleep apnea

	Yes
STOP	
Snoring?	■
Do you snore loudly (loud enough to be heard through closed doors, or your bed partner elbows you for snoring at night)?	
Tired?	■
Do you often feel tired, fatigued, or sleepy during the daytime (such as falling asleep during driving)?	
Observed?	■
Has anyone observed you stop breathing or choking/gasping during your sleep?	
Pressure?	
Do you have or are being treated for high blood pressure?	
Bang	
Body mass index >35 kg/m^2?	
Age >50 years?	■
Neck size large? (measured around Adams apple)	■
For male, is your shirt collar 17 inches/43 cm or larger?	
For female, is your shirt collar 16 inches/41 cm or larger?	
Gender: Male?	

This patient's STOP-Bang questionnaire revealed positive findings, including a history of loud snoring, a feeling of tiredness during the daytime, cessation of breathing during sleep, an age of >50 years and a neck circumference of 41 cm. ■=Indicate positive findings. Fewer than 3 Yes=Low risk of OSA; 3 or more Yes=Intermediate risk of OSA; 5-8 Yes=High risk of OSA. OSA=Obstructive sleep apnea

Chung *et al*. reported that serum HCO^{3-} levels ≥30 mmol/L plus STOP-Bang scores of ≥3 have a 95% predictive specificity for OSA.[5] Therefore, using the STOP-Bang questionnaire and serum HCO^{3-} level measurements should be considered in patients with suspected OSA.

CONCLUSION

This case highlights the importance of preoperative workup for patients with suspected OSA. Bradycardia and asystole after vagal stimulation during anesthesia can be expected in patients with a history of severe OSA. Therefore, OSA should be considered a significant risk factor for multiple adverse events, including bradycardia and cardiac arrest, because of vulnerability to vagal stimulation. Screening for OSA is important to prevent cardiovascular morbidity and mortality, especially during anesthesia.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that her name and initials will not be published and due efforts will be made to conceal her identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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