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Obstructive sleep apnea as a potentially reversible cause of nighttime bradyarrhythmias. Clinical case

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ABSTRACT

Obstructive sleep apnea syndrome is a common condition, especially among obese patients. Patients with obstructive sleep apnea syndrome have an increased risk of developing arterial hypertension and cardiovascular events, as well as cardiac arrhythmias, which include reflexively occurring bradyarrhythmias and episodes of asystole at night. Treatment of obstructive sleep apnea syndrome leads to an improvement in the patient's quality of life and also reduces cardiovascular risk and eliminates associated bradyarrhythmias during night sleep.

Keywords: obstructive sleep apnea syndrome; respiratory therapy; non-invasive ventilation; bradyarrhythmias.

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Синдром обструктивного апноэ сна как потенциально обратимая причина брадиаритмий в ночные часы. Клинический случай

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АННОТАЦИЯ

Синдром обструктивного апноэ сна является распространенным заболеванием, особенно среди больных ожирением. У пациентов с синдром обструктивного апноэ сна повышаются риски развития артериальной гипертензии и сердечно-сосудистых событий, чаще возникают нарушения ритма сердца, к которым относятся в том числе рефлекторно возникающие брадиаритмии и эпизоды асистолии в ночные часы. Лечение синдрома обструктивного апноэ сна не только приводит к улучшению качества жизни пациента, но и позволяет снизить сердечно-сосудистые риски, а также устранить ассоциированные с ним брадиаритмии во время ночного сна.

Ключевые слова: синдром обструктивного апноэ сна; респираторная терапия; неинвазивная вентиляция легких; брадиаритмии.

Как цитировать

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INTRODUCTION

Obstructive sleep apnea syndrome (OSAS) is characterized by recurrent episodes of airway obstruction at the pharyngeal level, cessation of pulmonary ventilation with persistent respiratory effort, decreased blood oxygen saturation, gross sleep fragmentation, and excessive daytime sleepiness [1].

The estimated prevalence of OSAS in individuals aged >30 years is between 5% and 7%. This condition affects approximately one billion people worldwide, with OSAS risk being correlated with body mass index (BMI) [2, 3]. OSAS is more prevalent in middle-aged and older men and postmenopausal women, and central obesity is the most significant and potentially modifiable risk factor for its development. Many patients with BMI ⩾ 30 kg/m^2 suffer from OSAS [4]. The dynamic airway lumen obstruction in OSAS can be caused by anatomical and/or functional factors. The most prevalent cause is the narrowing of the upper airway lumen associated with adipose tissue accumulation, which establishes the conditions for their collapse and obstruction during sleep [5, 6, 7].

The severity of OSAS is typically quantified by the apnea–hypopnea index (AHI). Diagnostic procedures (nocturnal cardiorespiratory/respiratory monitoring and polysomnography) during sleep involve assessing the frequency of obstructive events. Apnea is defined as the cessation of airflow for at least 10 s. Hypopnea is defined as a decrease in airflow of at least 30% for at least 10 s, accompanied by a reduction in oxygen saturation of at least 4%. According to the American Academy of Sleep Medicine, OSAS can be classified according to the AHI, which defines 5–15/h mild OSAS, 15–30/h as moderate, and ⩾30/h as severe [8].

Pathological daytime sleepiness, which may not always be perceived by the patient and is often described as fatigue, lassitude, or decreased energy, is a significant and prevalent consequence of sleep disturbance. Sleepiness leads to decreased social engagement and cognitive abilities and mediates the risk of accidents and motor vehicle accidents. Up to 20% of traffic accidents are thought to be related to falling asleep at the wheel. In clinical settings, OSAS may present with various additional symptoms, although none are diagnostic [4].

An increase in cardiovascular risks is another unfavorable consequence of sleep apnea. Consequently, a pathogenic link between OSAS and several cardiovascular diseases, including arterial hypertension (AH), heart rhythm disorders, heart failure, ischemic heart disease, and acute cerebrovascular disorders, has been established. OSAS can be a cause of pulmonary hypertension and plays a role in the development of metabolic syndrome and insulin resistance [9].

AH is a common comorbidity of OSAS. In approximately half of patients with OSAS, AH is accompanied by peculiarities in the blood pressure profile, including non-reduction or an increase in blood pressure at night. In >80% of cases, AH that is resistant to therapy with ⩾3 drugs is accompanied by OSAS.

Hypoxemia, autonomic dysregulation, and changes in intrathoracic pressure can lead to structural and functional remodeling of the atria and fibrosis development. This process increases the risk of cardiac rhythm disturbances, which are more frequent in individuals with more severe OSAS and hypoxemia. The mechanisms underlying arrhythmogenesis are based on changes in myocardial automatism, trigger activity, and re-entry mechanisms [10]. Abnormal automaticity can be associated with multiple factors, such as changes in sympathetic and parasympathetic tone, acid–base balance, and electrolyte disturbances at the membrane and submembrane levels [11]. OSAS causes repetitive, cyclic changes in sympathetic tone. During apnea attacks, an increase in the tone of the vagus nerve results in bradycardia, which is then followed by a sympathetic discharge caused by hypoxemia and hypercapnia. This, in turn, contributes to the formation of arrhythmias due to beta-adrenergic stimulation [12, 13].

OSAS increases the risk of atrial fibrillation by four times [14]. Arrhythmogenic effects of OSAS are also realized in the increased risk of atrial fibrillation recurrence after cardioversion, a twofold increased risk of recurrence after radiofrequency ablation, and decreased effectiveness of antiarrhythmic therapy [15–17].

OSAS is detected in 68% of patients with sleep-related bradyarrhythmias [20]. The most frequently recorded features at night are sinoatrial block, grade II atrioventricular block, ventricular extrasystole, and unstable ventricular tachycardia. At night, the incidence of arrhythmias can reach 50% [18–20]. The cyclic nature of heart block in OSAS is attributed to the occurrence of apnea episodes [21–25]. Nevertheless, bradyarrhythmias related to OSAS frequently do not indicate heart diseases and are reflexive. This occurs during ineffective respiratory efforts when hypoxemia in the absence of pulmonary ventilation causes bradycardia. In such cases, bradyarrhythmias manifest solely during sleep and dissipate following OSAS therapy [26]. According to C. Zwillich et al., the duration and severity of bradycardia correlate with the degree of hypoxemia during apnea [26].

H.F. Becker et al. demonstrated a resolution or reduction in the frequency of grade II–III atrioventricular blockade

and/or sinus node arrest with the effective treatment of OSAS [27].

Non-invasive ventilation (NIV), an effective method of respiratory support, involves creating positive airway pressure using nasal, oronasal, or face masks [28, 29]. The choice of the NIV regimen depends on the nature of respiratory disorders. Continuous positive airway pressure (CPAP) therapy is an NIV with continuous positive airway pressure throughout the respiratory cycle (inhalation and exhalation). CPAP therapy primarily maintains upper airway patency during sleep and prevents airway collapse. This treatment is considered the "gold standard" for treating OSAS. CPAP therapy is extremely effective in eliminating apnea and hypopnea. Although various treatment options are available for this condition, positive airway pressure therapy remains the mainstay of OSAS treatment since its introduction into practice in 1981 [30, 31]. CPAP therapy is initiated only after instrumental confirmation of the disease (mainly in moderate and severe OSAS). An individualized interface should be selected for comfort, and different masks may be better suited for people with different facial structures. To be effective, CPAP therapy should be used for at least 4 h per day for at least five nights per week. Currently, both the CPAP mode (which employs individually selected constant pressure) and the automatic positive airway pressure (APAP, auto-CPAP, an automated mode that employs algorithms to increase the pressure when episodes of sleep apnea are registered and to decrease it when they are absent) [28, 32].

CPAP therapy for OSAS results in clinically significant improvements in daytime sleepiness, ability to maintain wakefulness, and sleep-related quality of life indicators. CPAP therapy improves the AH course, including a reduction in blood pressure in resistant AH. A reduction in the risk of cardiovascular events was also established [28]. Continuous positive pressure NIV eliminates nocturnal bradyarrhythmias, which points to OSAS as the cause of these disorders [26, 27, 33].

CLINICAL CASE

Patient N., a 54-year-old man, presented with complaints of dyspnea during moderate physical activity and an associated decrease in tolerance to physical activity, daytime sleepiness, difficulty in nasal breathing, and snoring, which was corroborated by others. The patient also reported episodes of pressing sensations in the chest lasting up to 2 min during exercise and subsiding at rest. He attributed the appearance and progression of these symptoms to an increase in body weight over several years.

The medical history included hypertension, atherosclerosis of the brachiocephalic arteries with hemodynamically insignificant (35%–40%) stenosis of the common carotid arteries on both sides, grade 3 obesity, liver steatosis, and dyslipidemia. The patient was consistently taking antihypertensive drugs (sartans, diuretics, and calcium blockers).

The outpatient daily electrocardiogram (ECG) Holter monitoring revealed 33 pauses of >2000 ms, with a maximum duration of 3646 s, in sinus rhythm, which occurred during nocturnal sleep. No atrioventricular conduction disorder was identified. In addition, episodes of accelerated supraventricular rhythm with a heart rate of 75 beats/min were observed following a pause of 2114 ms during sleep. The calculated circadian index was 1.4. A total of 584 single supraventricular extrasystoles were observed, in addition to 9 paired and 12 group extrasystoles. The examination also revealed single polymorphic polytopic ventricular extrasystoles (48 in total), including insertion and bigeminy type. One paired monomorphic ventricular extrasystole was observed. No clinically significant repolarization disorders were observed at rest or during exercise.

The patient was referred to the L.G. Sokolov North-West District Research and Clinical Center of the Federal Medical and Biological Agency for further examination, including the exclusion of nocturnal respiratory disorders.

Upon examination, the patient was found to be in a satisfactory condition. The patient was conscious and alert. The skin had a normal color and moderate moisture. The patient exhibited excessive subcutaneous adipose tissue development, with a height of 1.72 m, body weight of 134 kg, and body mass index of 45.3 kg/m^2. The abdominal region exhibited characteristics of obesity. His pulse was rhythmic and satisfactory in terms of filling and tension, with a rate of 72 beats per minute, and his blood pressure was 150/100 mm Hg. The heart tones were muffled. Both chest sides were involved in breathing. The percussion sound was clear pulmonary. Auscultation revealed vesicular breathing, with no rales. The frequency of respiratory movements was 18/min. The abdominal volume increased because of subcutaneous adipose tissue accumulation and was soft and painless. There was no edema.

In the therapeutic department, the patient underwent respiratory monitoring at night. The results indicated severe OSAS. The AHI, desaturations index, average blood oxygen saturation, and minimum were 64.6/h, 62.3/h, 89%, and 69%, respectively.

Based on the findings of the respiratory study conducted at night, a course of respiratory therapy for OSAS was initiated. The patient underwent CPAP therapy at night using a Prisma 20A device (Loewenstein Medical (Weinmann), Germany) in the APAP mode through the oronasal mask. The patient reported a notable improvement in sleep quality,

reduction in daytime sleepiness, and enhancement in general well-being. The AHI during CPAP therapy was 5/h.

Daily Holter ECG monitoring of the ECG confirmed the absence of signs of conduction disturbances and pauses in respiratory therapy for OSAS. Sinus rhythm with a normal circadian profile and normal total variability was recorded during the study. A single, paired, and group ventricular extrasystole were recorded, along with a few supraventricular extrasystoles, including 35 single and 1 paired. No ischemic repolarization changes were found at rest or on exertion.

In light of the clinical presentation and diagnostic testing results, a comprehensive search for coronary heart disease was initiated. Coronary angiography revealed significant stenosis of the anterior interventricular artery, prompting subsequent angioplasty with stenting.

In this patient on CPAP therapy, severe OSAS and the elimination of heart rhythm pauses permitted the consideration of these changes in Holter ECG monitoring as secondary (reflex) to apnea and hypopnea episodes and the exclusion of contraindications to beta-adrenoblockers such as sinus node dysfunction. This included prescribing optimal drug therapy for ischemic heart disease.

CONCLUSIONS

1. In clinical practice, OSAS must be urgently verified, given its high prevalence and the increased risk of cardiovascular disease.

2. The provision of respiratory therapy at night eliminates OSAS as a potentially reversible cause of heart rhythm abnormalities and a risk factor for cardiovascular disease.

ADDITIONAL INFORMATION

Ethics approval. The study was approved by North-Western state medical university named after I.I. Mechnikov of Sciences Ethics Committee, protocol No. 10, 11.10.2023.

Author contribution. Thereby, all authors confirm that their authorship complies with the international ICMJE criteria (all authors have made a significant contribution to the development of the concept, research, and preparation of the article, as well as read and approved the final version before its publication).

Personal contribution of the authors. N.G. Kucherenko, A.N. Bebekh — data analysis, writing the main part of the text; I.A. Umarova — data analysis; A.R. Abukova — data analysis, literature review.

Competing interests. The authors declare that they have no competing interests.

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ДОПОЛНИТЕЛЬНАЯ ИНФОРМАЦИЯ

Заключение этического комитета. Исследование было одобрено этическим комитетом Северо-Западного государственного университета им. И.И. Мечникова (протокол № 10 от 11.10.2023).

Вклад авторов. Все авторы внесли существенный вклад в разработку концепции, проведение исследования и подготовку статьи, прочли и одобрили финальную версию перед публикацией.

Вклад каждого автора. Н.Г. Кучеренко, А Н. Бебех — анализ полученных данных, написание текста; И.А. Умарова — анализ полученных данных; А.Р. Абукова — анализ полученных данных, обзор литературы.

Конфликт интересов. Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи.

Источник финансирования. Авторы заявляют об отсутствии внешнего финансирования при проведении исследования.

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