VIA MEDICA

CASE REPORT

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Diagnosis and treatment of obstructive sleep apnoea in patients with pauses in Holter ECG monitoring: case series

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ABSTRACT

Obstructive sleep apnoea (OSA) is a common disease in today's society and may affect even more of the population in the near future. Ailments and complications of OSA result from a decrease in the muscle tone of the soft palate, which causes shallow breathing or complete apnoea. The consequences of such episodes may be the development of arterial hypertension, the occurrence of cardiac arrhythmias, and also the deterioration of the quality of life. Diagnosis is based on polysomnography in people with suspected OSA. Continuous Positive Airway Pressure (CPAP) therapy is the most effective treatment for OSA. A case series is presented in which patients with OSA risk factors and nocturnal pauses in Holter ECG monitoring (HEM) were diagnosed due to suspected OSA. In the polysomnographic test, severe OSA was diagnosed and CPAP therapy was introduced. Follow-up HEM performed during treatment showed complete resolution or significant reduction in the number and length of nocturnal pauses. Based on the case series, current medical knowledge and guidelines for pacemaker implantation, it was concluded that in the event of pauses in the HEM, the diagnosis of OSA should be considered in each patient, especially if the pauses occur predominantly at night and the patient is at high risk of OSA. Such a procedure may protect the patient from serious complications related to the pacemaker implantation. It should be emphasized, however, that CPAP therapy requires close cooperation of the patient because it brings effects only when it is used for each sleep.

Keywords: obstructive sleep apnoea, bradyarrhythmia, CPAP, nocturnal pauses, pacemaker implantation

Introduction

Obstructive sleep apnoea (OSA) is a disease associated with impaired breathing during sleep [1]. It is a disorder that is common in the population, but also underdiagnosed [2, 3]. It affects 2–24% of the population in middle age, but in people over 65 years old, OSA symptoms are already present in at least 50% [4, 5]. Overweight and obesity are the main risk factors [3, 6]. Nowadays, it is talked about an epidemic of obesity [7], especially in young people, so the diagnosis and treatment of OSA may become even more important in the near future. In a patient with OSA, there is a decrease in muscle tone of the soft palate and consequent reduction (hypopnea) or closure of airflow (apnoea) [8].

This can be followed by fragmented sleep, a decrease in saturation, brady- and tachyarrhythmia, increased blood pressure, but also subjective sensations that affect the comfort of life, such as excessive daytime sleepiness, nighttime awakening with feelings of anxiety, excessive sweating, problems maintaining concentration, irritability or decreased libido [6].

The diagnosis and severity of OSA are determined by polysomnography [9] — multiple indicators such as apnoea-hypopnea index (AHI), apnoea index (AI), hypopnea index (HI), oxygen desaturation index (ODI) are assessed during the overnight study. According to the recommendations of the Polish Society of Respiratory Diseases (PTChP) on the diagnosis and treatment of sleep-disordered breathing (SDB) in adults and the American Academy of Sleep Medicine (AASM), OSA is divided into mild (AHI 5–15), moderate (AHI 16–30) and severe (AHI > 30) [9, 10].

Questionnaires such as the Epworth sleepiness scale (ESS) (Tab. 1) [11] or STOP-Bang (Tab. 2) [12] help to assess the risk of OSA, but it is polysomnography that is the basis for diagnosis.

The polysomnographic test consists of recording the bioelectrical activity (e.g. electroencephalography) and the polygraphic part (movements of the abdomen and chest, measurement of pulse, saturation, blood flow, body position, presence of snoring). Often, only a polygraphic examination without bioelectrical analysis is sufficient to make the diagnosis [13]. A method that provides immediate improvement in OSA is Continuous Positive Airway Pressure (CPAP) treatment [14–16]. The therapy involves the use of a special mask worn over the face, which is connected to a device that, by means of maintaining positive airway pressure, prevents the soft palate from collapsing [15]. This is currently the most commonly used method, however, each patient should be treated individually. Other methods that may reduce OSA-related symptoms include intraoral appliances, stimulation of the sublingual nerve, laryngological operations, positional therapy, and myofunctional training [10]. In addition, weight reduction is recommended for each overweight and obese person, which may have a positive effect on reducing AHI [17].

This study presents a clinical case series of patients admitted for SDB diagnosis at the Department of Diagnostic Medicine at the St. John Paul II Specialized Hospital in Krakow, Poland, who had nighttime pauses in Holter ECG monitoring (HEM) performed before hospitalization.

Table 1. Epworth Sleepiness Scale (0 = would never doze, 1 = slight chance of dozing, 2 = moderate chance of dozing, 3 = high chance of dozing; total score ≥ 10 suggests presence of excessive sleepiness)

How likely are you to doze off or fall asleep in the following situations, in contrast to feeling just tired? This refers to your usual way of life in recent times. Even if you have not done some of these things recently try to work out how they would have affected you.	**0**	**1**	**2**	**3**
Sitting and reading				
Watching TV				
Sitting, inactive in a public place (e.g. a theatre or a meeting)				
As a passenger in a car for an hour without a break				
Lying down to rest in the afternoon when circumstances permit				
Sitting and talking to someone				
Sitting quietly after lunch without alcohol				
In a car, while stopped for a few minutes in traffic				

Table 2. STOP-Bang Scoring Model (high risk: answering yes to three or more items, low risk: answering yes to less than three items; BMI — body mass index)

Question	**Yes**	**No**
Do you snore loudly (louder than talking or loud enough to be heard through closed doors)?		
Do you often feel tired, fatigued, or sleepy during the daytime?		
Has anyone observed you stop breathing during your sleep?		
Do you have or are you being treated for high blood pressure?		
BMI more than 35 kg/m^2?		
Age over 50 years old?		
Neck circumference greater than 40 cm?		
Gender male?		

Case series

Case 1

A 69-year-old patient was admitted to the Cardiology Department for the diagnosis of SDB. The main complaints, suggesting the diagnosis of OSA, included snoring, episodes of pause in breathing during sleep observed by relatives of the patient and nycturia (SSE = 5, STOP-Bang = 5). In addition, the patient had a history of dyslipidaemia, hypertension and obesity. The ambulatory HEM showed an average sinus rhythm of 73/min, in the range of 44–120/min, additionally 25 episodes of bradycardia (at least 16/min at night) and 71 pauses at night in the mechanism of sinus arrest (the longest lasting 5962 ms). The patient was taking Bellapan — a natural tropine alkaloid. In measurements, the BMI was 32.36.

Basic laboratory tests detected hypertriglyceridemia, elevated creatinine, decreased renal function (eGFR = 55) and mildly elevated B-type natriuretic propeptide (pBNP) (212 pg/mL).

A polysomnographic test was performed at night. Based on the overall clinical picture, according to the PTChP recommendations for the diagnosis and treatment of SDB in adults and the AASM, a diagnosis of severe SDB in obstructive, central and mixed mechanisms was made (AHI = 78.0/h; AI = 60.8/h; HI = 17.3/h. Apnoeas in obstructive (21.4/h), central (16.9/h) and mixed (22.5/h) mechanisms. Mean apnoea duration: 25.4 s, longest: 50,8 s. Shallow respirations with an average duration of 18.0 s. Saturation on average: 91.4%. ODI: 78.2/h. Lowest saturation: 76.0%). There were pauses of up to 4 seconds in the sinus inhibition mechanism correlated with respiratory events. The patient was qualified for CPAP therapy. A trial of CPAP therapy was conducted with good results and good tolerance of the therapy.

In the control HEM during the application of CPAP therapy, the leading rhythm was sinus rhythm with an average rate of 59/min, (46–91/min) with one episode of bradycardia (min. 36/min), without pauses. The patient's past treatment was modified — Bellapan was discontinued.

Case 2

A 75-year-old patient was admitted to the Cardiology Department for the diagnosis of SDB. The main complaints, suggesting a diagnosis of OSA, included loud snoring, episodes of pause in breathing noticed by the patient's family, increased sweating during sleep, a feeling of awakening with shortness of breath, nycturia, morning headaches, sleep not bringing rest (SSE = 6, STOP-Bang = 6). In addition, the patient had hypertension, hypercholesterolemia and obesity. An ambulatory HEM recorded a leading sinus rhythm with an average rate of 68/min and in the range from 53 to 100/min, additionally 9 pauses during the night - the longest lasting 3.11s, quite numerous additional ventricular beats (3154 additional polymorphic ventricular beats including 1 episode of accelerated ventricular rhythm/ventricular arrhythmia, 19 pairs, 91 bigeminiesand 3 trigeminies). The patient was taking long-acting beta-blockers (BB).

In measurements, the BMI was 56.39.

Basic laboratory tests detected normal pBNP, elevated D-dimer levels, hyperuricemia and hypercholesterolemia. Due to exertional dyspnoea additional tests were performed to expand the diagnosis. The echocardiography, chest X-ray, ultrasound of the deep veins and CT angiography of the pulmonary arteries found no significant pathology.

A polysomnographic test was performed at night. Based on the entire clinical picture, according to the PTChP recommendations for the diagnosis and treatment of SDB in adults and the AASM, a diagnosis of severe OSA was made (AHI = 110.7/h, AI = 91.7/h, HI = 19.1/h. Apnoeas in obstructive (90.8/h), central (0.1/h) and mixed (0.7/h) mechanism. Mean apnoea duration: 15.6 s, longest: 29.0 s. Shallow breathing with an average duration of 12.0 s. Saturation on average: 88.7%. ODI = 78.2/h. Lowest saturation: 67.0%). The patient was qualified for CPAP therapy. A trial of treatment with CPAP was carried out with good results and tolerance of the therapy.

In the control HEM during CPAP therapy, sinus rhythm with an average rate of 63/min (50–85/min) was leading, in addition, 1 pause of borderline length (2.01 seconds) and few ventricular and supraventricular beats (polymorphic ventricular beats of 832 including 3 bigeminies and 4 trigeminies, extra supraventricular beats: 149 including 6 salvos and 6 pairs).

Case 3

A 69-year-old patient was admitted to the Cardiology Department for the diagnosis of SDB. The main complaints, suggesting a diagnosis of OSA, included loud snoring, momentary pauses in breathing during sleep observed by the patient's daughter, and nycturia (SSE = 3, STOP-Bang = 5). In addition, the patient's history included chronic heart failure with preserved ejection fraction, persistent atrial fibrillation,

hypertension, hypercholesterolemia, and obesity. An ambulatory HEM recorded atrial fibrillation with an average rate of 71/min and ranging from 31–170/min, 95 tachycardias, 160 bradycardias, 410 pauses at night, the longest of 4450 ms. On admission, the patient was using long-acting BB.

In measurements, the BMI was 31.63.

Basic laboratory tests detected hypercholesterolemia, hyperuricemia, slightly elevated pBNP (557 pg/mL) and slight hyperglycaemia. The oral glucose tolerance test (OGTT) performed gave a normal result. The patient was consulted pulmonologically due to chronic moist cough with expectoration of mucous secretions, numerous furcations, wheezes over the lung fields, and crackles at the base of the lungs. A body plethysmography study was performed, which diagnosed obstructive-type ventilation disorder, with high airway resistance and chronic inhaled treatment was started. Echocardiography showed enlarged both atria of the heart, thickened left ventricular myocardium, preserved global left ventricular contractility [left ventricular ejection fraction (LVEF) 55%], without obvious segmental abnormalities, moderate mitral regurgitation.

A polysomnographic test was performed. Based on the entire clinical picture, according to the PTChP recommendations for the diagnosis and treatment of SDB in adults and the AASM, a diagnosis of severe OSA was made (AHI = 58.8/h; AI = 12.5/h; HI = 46.3/h. Apnoeas in obstructive (12.1/h) and mixed (0.4/h) mechanism. Mean apnoea duration: 19.1 s, longest: 40,5 s. Shallow breathing with an average duration of 21.7 s. Saturation on average: 89.0%. ODI: 61.6/h. Lowest saturation: 73.0%). The patient was qualified for CPAP therapy. A trial of CPAP therapy was conducted with good results and a good tolerance of therapy. A control HEM during CPAP showed atrial fibrillation with a mean rate of 78/min (62–147/min), with no pauses or bradycardia in the recording.

Due to the occurrence of pauses and bradycardia at night, a long-acting BB (bisoprolol — half-life 10–12 h, duration of action up to 24 h) was additionally changed to a short-acting BB administered in the morning (metoprolol — half-life 3–5 h, duration of action up to 9 h).

Case 4

A 71-year-old patient was admitted to the Cardiology Department for the diagnosis of SDB. Additionally, the patient had a history of persistent atrial fibrillation, hypertension and obesity.

On admission, the patient denied typical symptoms that might suggest the diagnosis of OSA (SSE = 0, STOP-Bang = 3). In ambulatory HEM atrial fibrillation was recorded with an average ventricular rate of 79/min, (34–169/min) with approximately 300 pauses of over 2 seconds, the longest 2972 ms, occurring mainly at night. On admission, the patient was taking short-acting BBs and theophylline, a natural methylxanthine derivative.

In measurements, the BMI was 34.35.

Basic laboratory tests detected hyperglycaemia and mixed hyperlipidaemia. The diagnosis was expanded with an OGTT, which revealed impaired glucose tolerance and a chest X-ray that showed no significant abnormalities.

Polysomnography was performed at night. Based on the overall clinical picture, in accordance with the PTChP recommendations for the diagnosis and treatment of SDB in adults and the AASM, a diagnosis of severe OSA was made (AHI = 34.6/h; AI = 18.3/h; HI = 16.3/h. Apnoeas in obstructive (9.8/h), central (2.2/h) and mixed (6.4/h) mechanisms. Mean apnoea duration: 24.1 s, longest: 44,2 s. Shallow breathing with an average duration of 22.4 s. Saturation on average: 93.5%. ODI: 35,6/h. Lowest saturation: 80.0%.).

A trial of treatment with CPAP was carried out with good results and good tolerance of the therapy.

In the control HEM during CPAP therapy, atrial fibrillation was recorded with an average ventricular rate of 66/min, (46–101/min), without pauses in the recording. Treatment was modified — theophylline was discontinued.

Discussion

OSA is a risk factor for many cardiovascular diseases such as hypertension, heart failure, coronary artery disease, arrhythmias (brady- and tachyarrhythmias) and sudden cardiac death [18–20]. Hypoxemia and hypercapnia occurring during an apnoeic episode cause chemoreflex activation and consequently increased sympathetic vascular nerve activity and catecholamine release [18]. Tachycardia and an increase in blood pressure occurring at the end of apnoea coincides with the lowest saturation. Such a situation causes an imbalance between the increased oxygen demand of the heart muscle and the decreased oxygen supply of the blood [18]. An additional factor contributing to greater cardiovascular risk is increased prothrombotic activity associated with increased platelet activation and aggregation [18]. In addition, hypoxemia can affect renal and adrenal receptors that are responsible for the secretion of hormones such as catecholamines, renin, and angiotensin II [19]. This may result in the

persistence of increased tension of the sympathetic system also during the daytime [18, 19] and consequently increase vascular resistance and vascular remodelling [19]. Arrhythmias that occur at night are associated with the chemoreceptor reflex [21] and are often reversible with effective therapy, especially CPAP [22, 23]. Bradyarrhythmias in OSA occur through the effects of hypoxemia on chemoreceptors and parasympathetic stimulation [21]. They are closely associated with the onset of apnoea in a person who cannot compensate for hypoxemia through hyperventilation. Baroreceptors, in contrast, act antagonistically and inhibit parasympathetic action, however, patients with long-term hypertension may have baroreflex impairment and consequently a predominance of chemoreceptor action [21]. In conclusion, in patients with OSA, depending on the situation, the chemoreceptor reflex can cause both tachy- and bradyarrhythmias at night. It is important to consider performing a diagnosis of OSA before deciding to implant a cardiac pacemaker for bradyarrhythmia. The selected clinical cases were characterized by pauses in HEM prior to the diagnosis of OSA with subsequent complete resolution or significant reduction of abnormalities on follow-up during CPAP therapy. In a study of 443 patients who were burdened with cardiovascular disease, 282 of them were classified as people at high risk of SDB, and OSA was diagnosed in 158 study participants. Pauses longer than 3 seconds were diagnosed in 12 patients — with a higher frequency in those with atrial fibrillation — 8 cases [24]. Another study of 51 patients showed a significant reduction in the number (from 148.58 ± 379.44 to 16.07 ± 58.5) and length of pauses (4.38 ± 2.95 seconds to 0.57 ± 1.05 seconds) after implementation of CPAP therapy [25]. In studies, the incidence of bradyarrhythmia and sinus pauses is characterized by wide variability and ranges from 5–50% [20, 26, 27]. Studies present a significant reduction in the incidence of bradyarrhythmias and sinus pauses during CPAP therapy [18, 22, 23]. When analysing the HEM, special attention should be paid to the timing of bradyarrhythmias and relate these situations to the patient's history. As previously pointed out, OSA-induced bradyarrhythmias are associated with the chemoreceptor response to hypoxemia associated with the onset of apnoea during sleep [18, 19]. Usually, such situations occur at night, however individuals with untreated OSA often sleep during the day in the hospital setting, and this can result in sleep-related arrhythmias during the day.

According to the European Society of Cardiology pacing guidelines and cardiac resynchronization therapy from 2021 mention that pacemaker implantation should be considered for symptomatic bradyarrhythmia [28]. Establishing a link between symptoms and bradyarrhythmia can often be difficult due to the numerous symptoms of comorbidities. According to the guidelines, in the presence of asymptomatic bradyarrhythmias during the night and symptoms associated with SDB, a diagnosis of OSA should be made [29]. On the other hand, difficulties in management may arise in case of daytime bradyarrhythmia or absence of OSA symptoms. The ESC guidelines detail the part of bradyarrhythmia that is a pause, which should be considered in the context of the patient's clinical data. Due to insufficient scientific data at this time, no clear recommendations can be made regarding pauses found in asymptomatic patients [29].

In addition, pacemaker implantation is associated with the risk of complications such as lead-related re-intervention, infections (superficial, within the locus and systemic), pneumothorax, pleural haematoma, brachial plexus injury, cardiac perforation, coronary sinus dissection/perforation, haematoma, tricuspid regurgitation and deep vein thrombosis [29]. The rate of any complication is in the range of 5–15%, and the mortality rate up to 30 days is 0.8–1.4% [29]. In comparison, CPAP therapy is associated with a milder degree and fewer complications and there are no studies in the available medical literature on the complications of using CPAP to treat OSA. One of the limitations in connection with CPAP therapy is the patient's cooperation because only when used does sleep apnoea disappear — a large proportion of patients do not use the therapy regularly [30].

Conclusions

OSA is a disease that can cause bradyarrhythmias, including pauses, while sleeping. Such a situation was observed in the patients presented in the case series. In patients who are observed to have multiple pauses in the HEM, especially during the nighttime hours, and/or with a high risk of OSA, it is advisable to consider polysomnography for OSA, before deciding on implantation of a cardiac pacemaker. The use of CPAP therapy is an effective and recommended method of treating OSA and resulting bradyarrhythmias, however, it requires the patient's cooperation and regular use of the device.

Article information

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