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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

# Obstructive sleep apnoea in patients with atrial fibrillation: prevalence, determinants and clinical characteristics of patients in Polish population

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## Abstract

**Background:** Obstructive sleep apnoea (OSA) and atrial fibrillation (AF) are two conditions highly prevalent in the general population. OSA is known to cause haemodynamic changes, oxidative stress, and endothelial damage, and therefore promote vascular and heart remodelling which results in AF triggering and exacerbation. Coexistence of OSA and AF influences the course of both diseases, and therefore should be taken into consideration in patient management strategy planning.

**Aim:** To assess the prevalence of OSA in Polish AF patients, and to describe the clinical characteristics of patients with concomitant OSA and AF.

**Methods:** We enrolled into the study 289 consecutive patients hospitalised in a tertiary, high-volume Cardiology Department with a primary diagnosis of AF. In addition to standard examination, all patients underwent an overnight sleep study to diagnose OSA, which was defined as apnoea–hypopnoea index (AHI) ≥ 5 per hour.

**Results:** After applying exclusion criteria, the final analysis covered 266 patients (65.0% male, mean age 57.6 ± 10.1 years). OSA was present in 121 (45.49%) patients. Patients with OSA were older (59.6 ± 8.0 vs. 56.0 ± 11.4 years; $p = 0.02$), had higher body mass index (BMI; 30.9 ± 5.4 vs. 28.7 ± 4.4 kg/m$^2$; $p < 0.01$) larger neck size (41.2 ± 3.8 vs. 39.3 ± 3.3 cm; $p = 0.0001$) and waist circumference (108.5 ± 13.1 vs. 107.7 ± 85.4 cm; $p < 0.0001$) than patients without OSA. There were no significant differences between the groups in terms of systolic and diastolic blood pressure or history of comorbidities ($p > 0.05$). OSA patients were less likely than non-OSA patients to have paroxysmal AF (62.0% vs. 75.9%; $p = 0.02$). Dividing newly diagnosed OSA patients according to the disease severity showed that mild OSA (AHI ≥ 5/h and < 15/h) was present in 27.82% of the study population, moderate OSA (AHI ≤ 15/h and ≥ 30/h) in 13.16% of patients, and severe OSA (> 30/h) in 4.51% of patients. No significant differences in terms of comorbidities and anthropometric features were seen between mild and moderate, between moderate and severe, and between mild and severe OSA.

**Conclusions:** OSA is highly prevalent in patients with AF in the Polish population, and affects approximately half of the patients. OSA patients are more likely to be older, have higher BMI, and greater waist and neck circumference. Persistent AF is the most common form of the arrhythmia in patients with OSA, while patients without OSA are more likely to have paroxysmal AF.

**Key words:** obstructive sleep apnoea, atrial fibrillation, epidemiology

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## INTRODUCTION

Obstructive sleep apnoea (OSA) is a disorder of the edges of pulmonology, laryngology, cardiology and other medical specialties. This disease of a seemingly simple aetiology, associated with repeating episodes of apnoeas and hypopnoeas caused by collapse of the upper respiratory tract, is in fact responsible for a number of complications in organs and systems other that airways and lungs. Recently, the relationship between OSA and cardiovascular (CV) disorders has been widely discussed. Most of the common CV disorders

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can be somehow linked with this type of sleep disordered breathing. Hypoxia and inflammation associated with OSA cause endothelial damage and a state of increased coagulation [1]. It is also responsible for haemodynamic changes caused by repetitive microarousals following desaturation, associated with rapid increases in sympathetic activity, heart rate and blood pressure [2]. Therefore, OSA changes healthy vasculature, promotes endothelial dysfunction and subsequent atherosclerosis, and causes remodelling of blood vessel walls and heart chambers. It results in increased risk of resistant hypertension, heart failure, coronary artery disease, myocardial infarction (MI), stroke, sexual dysfunction, and thromboembolic diseases [1, 3–9].

The described mechanisms also play a role in the development of atrial fibrillation (AF), one of the most common types of arrhythmia. Electrophysiology and echocardiographic studies show that OSA patients have significant atrial remodelling associated with atrial enlargement, reduction in voltage, conduction abnormalities, and prolonged sinus node recovery time — factors associated with increased AF risk [10]. In most of the AF cases, a single cause of arrhythmia is impossible to describe, but determining potentially reversible or treatable causes is helpful in long-term patient management [11]. Identification of OSA patients nowadays should be an integral part of cardiology practice, not limited to hypertensive or MI patients [12]. The aim of the present study was to determine OSA prevalence, and describe the clinical characteristics of OSA patients in an AF cohort.

## METHODS

### *Study population*

The study population consisted of 289 consecutive patients hospitalised in a tertiary, high-volume Cardiology Department as part of the pre-qualification process for catheter ablation of AF. We included in the study patients with AF documented in at least one 24-h electrocardiogram (ECG) Holter examination performed in the six months prior to the enrollment. Exclusion criteria were age $< 18$ or $\geq 75$ years, prior ablation of AF, MI, stroke or decompensation of heart failure within the last six months, untreated overt hyper- or hypothyroidism, current treatment with a continuous positive airway pressure device, and absence of written informed consent.

### *Obstructive sleep apnoea diagnosis*

During the hospitalisation, apart from a standard ablation pre-qualification examination (including taking medical history, biochemical evaluation, or transoesophageal echocardiography, if needed), an overnight sleep study using a portable device for diagnosing sleep disorders (Embletta Gold, Flaga, Reykjavik, Iceland) was performed within one day following admission in every patient. The polygraphy device used was a level 3 sleep monitoring tool, according to the recommendations of the European Respiratory Society and the European Society of Hypertension, with $> 4$ channels including channels to detect respiratory movements or respiratory effort, airflow, heart rate, ECG, and oxygen saturation [13]. According to the recommendations, level 3 sleep monitoring tools are sufficient to objectively confirm an OSA diagnosis.

The data was scored manually according to the recommendations of the American Academy of Sleep Medicine [14]. Apnoea was defined as a cessation of airflow lasting 10 s. Hypopnoea was defined as a recognisable transient reduction (but not complete cessation) of breathing for 10 s or longer, a decrease of greater than 50% in the amplitude of a validated measure of breathing, or a reduction in amplitude of less than 50% associated with oxygen desaturation of 4% or more. Total desaturation time was defined as the period during which blood oxygen saturation was 90% or lower. OSA was diagnosed when the apnoea–hypopnoea index (AHI) exceeded the threshold of $\geq 5$ per hour. Patients with confirmed OSA were subsequently grouped according to the disease severity into a AHI $\geq 5$/h and $< 15$/h (mild OSA group); a AHI $\leq 15$/h and $\geq 30$/h (moderate OSA group); and a $> 30$/h (severe OSA group).

### *Atrial fibrillation evaluation*

Type of AF was determined in accordance with the definitions contained in the 2010 and 2012 European Society of Cardiology Guidelines for the management of AF [11, 15]. Since permanent AF is diagnosed only if it is decided not to pursue rhythm control strategy, and the cohort consisted of patients prequalified for AF ablation, permanent AF patients were not included in the study. Paroxysmal AF was defined as self-terminating, usually within 48 h, which may continue for up to seven days. Persistent AF was defined when an AF episode either lasted longer than seven days or required termination by cardioversion, either with drugs or by direct current [11]. AF symptoms severity was classified according to EHRA score, EHRA I being patients who did not experience any symptoms of AF. In EHRA II were patients with only mild symptoms, not affecting normal daily activity. In EHRA III were patients with severe AF symptoms, affecting daytime activity, while EHRA IV contained patients with disabling symptoms, forcing them to discontinue normal daily activity [11].

### *Statistical analysis*

Continuous data are presented as mean ± standard deviation (SD) and were compared using the Mann-Whitney test or Student's t-test. Categorical variables were compared using either the $\chi^2$ or Fisher exact tests. A p value of less than 0.05 was considered statistically significant, whereas the confidence intervals (CI) were 95%. All analyses were performed using SAS statistical software version 8.02 (SAS Institute, Inc., Cary, NC, USA).

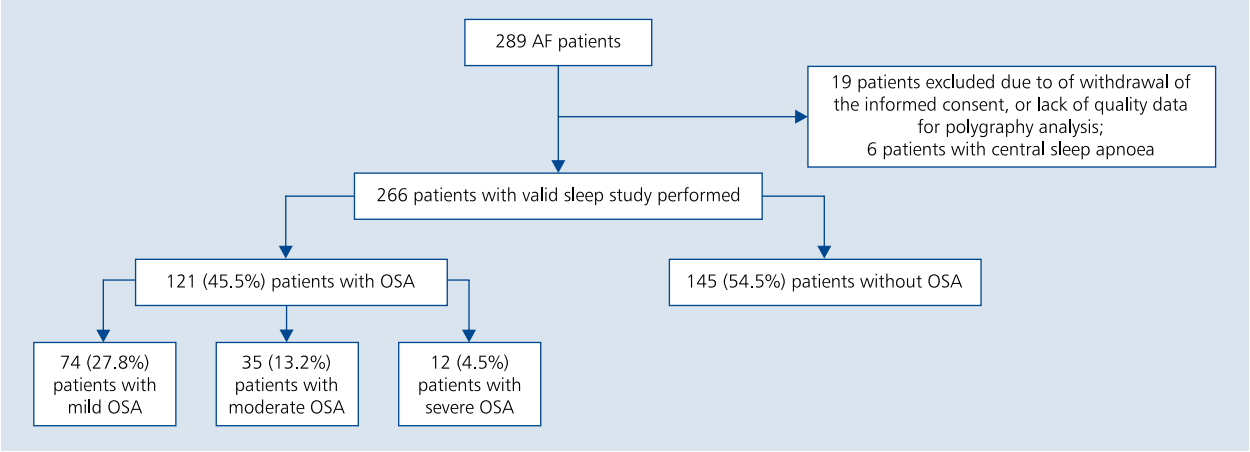


Figure 1. Study design flow chart; AF — atrial fibrillation; OSA — obstructive sleep apnoea

## RESULTS

The final analysis covered 266 patients. In 19 cases, patients were excluded because of withdrawal of informed consent, lack of data required for analysis, or polygraphy result quality insufficient for the analysis. Six patients were excluded from the study because of central sleep apnoea diagnosed in the polygraphy (Fig. 1).

The study group was predominantly male (173 patients, 65.0%) with a mean age of 57.6 ± 10.1 years. Mean body mass index (BMI) was 29.7 ± 5.0 kg/m$^2$ which meets the criteria for overweight. On admission, mean systolic and diastolic blood pressure were 131.7 ± 16.7 and 80.7 ± 11.1 mm Hg, respectively, but hypertension was previously diagnosed in as many as 194 (72.9%) patients. Dyslipidaemia was diagnosed in 167 (62.8%) patients. Other comorbidities were not highly prevalent in the study population. Diabetes mellitus was present in 22 (8.3%) patients, the same percentage as those with a history of MI (8.3%).

As for the AF, paroxysmal form of the arrhythmia was present in the majority of patients (185 patients, 69.5% of the study population), while the remaining patients had persistent AF (30.5%). Dividing patients according to AF severity showed that 33 (12.4%) of them had symptoms in EHRA class I, 104 (39.1%) patients in EHRA II, 110 (41.4%) patients in EHRA III, and 19 (7.1%) patients in EHRA IV. Other baseline characteristics of the study group are shown in Table 1.

Analysis of the sleep study results showed that OSA was present in 121 (45.49%) patients. Figure 2 illustrates an example of a polygraphy result of a patient with OSA. As expected, OSA patients had higher mean AHI (15.6 ± 12.1 vs. 1.9 ± 1.4/min) and their mean lowest recorded oxygen saturation was lower than in non-OSA patients (81.6 ± 7.2 vs. 86.5 ± 5.0%; $p < 0.0001$). Patients with OSA were older (59.6 ± 8.0 vs. 56.0 ± 11.4 years; $p = 0.02$), had higher BMI (30.9 ± 5.4 vs. 28.7 ± 4.4 kg/m$^2$; $p < 0.01$) and great-

Table 1. Baseline characteristics of the study population

| Parameter | Mean ± SD or n (%) |
|---|---|
| Male sex | 173 (65.0%) |
| Age [years] | 57.6 ± 10.1 |
| Height [cm] | 172.0 ± 10.4 |
| Weight [kg] | 87.9 ± 17.0 |
| Body mass index [kg/m$^2$] | 29.7 ± 5.0 |
| Neck circumference [cm] | 40.2 ± 3.6 |
| Waist circumference [cm] | 108.1 ± 64.2 |
| Systolic BP [mm Hg] | 131.7 ± 16.7 |
| Diastolic BP [mm Hg] | 80.7 ± 11.1 |
| Heart rate [bpm] | 75.1 ± 15.8 |
| Prior MI | 22 (8.3%) |
| Diabetes mellitus | 22 (8.3%) |
| Hypertension | 194 (72.9%) |
| Gout | 18 (6.8%) |
| Smoking | 28 (10.5%) |
| Alcohol consumption | 23 (8.6%) |
| Prior stroke | 22 (8.3%) |
| Peptic ulcer disease | 44 (16.5%) |
| Dyslipidaemia | 167 (62.8%) |
| Thyroid disease | 68 (25.6%) |
| Family history of CVD | 104 (39.1%) |
| EHRA class I | 33 (12.4%) |
| EHRA class II | 104 (39.1%) |
| EHRA class III | 110 (41.4%) |
| EHRA class IV | 19 (7.1%) |
| Paroxysmal AF | 185 (69.5%) |

SD — standard deviation; BP — blood pressure; MI — myocardial infarction; CVD — cardiovascular disease; AF — atrial fibrillation; EHRA — atrial fibrillation symptoms severity according to European Heart Rhythm Association score

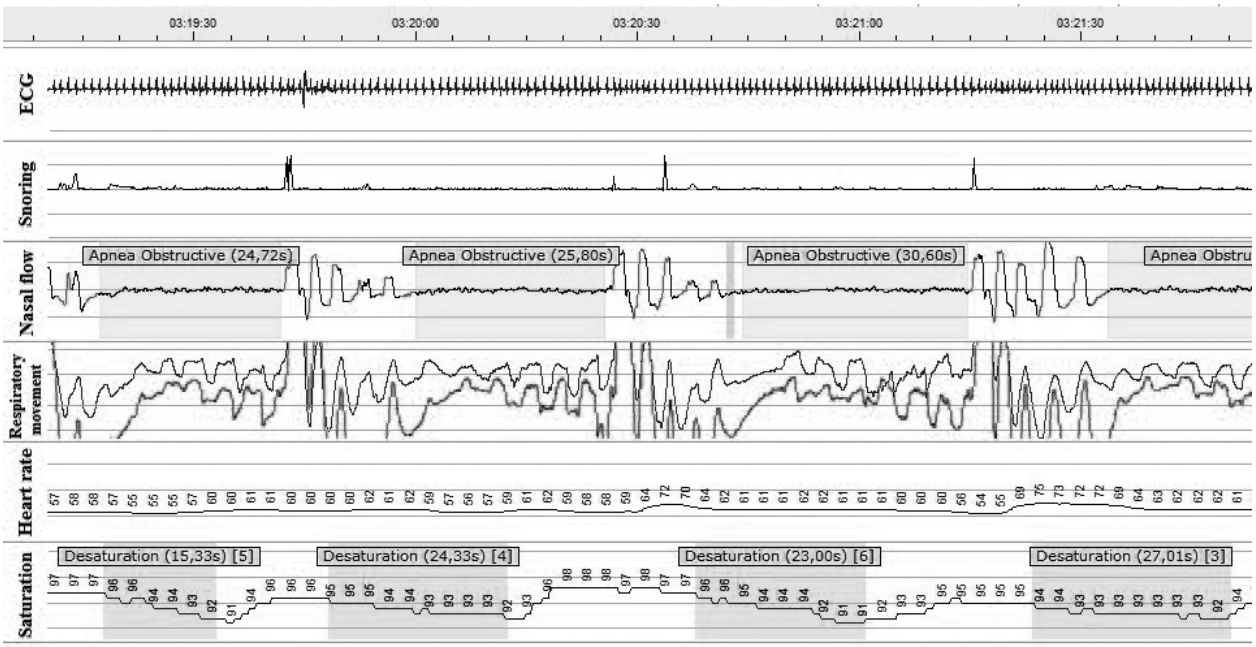


Figure 2. Example of polygraphy results of patient with obstructive sleep apnoea

er neck circumference (41.2 ± 3.8 vs. 39.3 ± 3.3 cm; $p < 0.01$) and waist circumference (108.5 ± 13.1 vs. 107.7 ± 85.4 cm; $p < 0.0001$) than patients without OSA. There were no differences between the groups in terms of systolic and diastolic blood pressure or history of comorbidities ($p > 0.05$). A trend towards higher heart rate in OSA patients was observed, but it did not reach the level of statistical significance (77.3 ± 17.9 vs. 73.3 ± 13.5 bpm; $p = 0.0596$). Importantly, the paroxysmal form of arrhythmia was present in fewer of the OSA patients than the non-OSA patients (62.0% vs. 75.9%; $p = 0.02$) (Table 2).

Dividing newly diagnosed OSA patients according to the disease severity showed that mild OSA (AHI ≥ 5/h and < 15/h) was present in 27.82% of the study population patients, moderate OSA (AHI ≤ 15/h and ≥ 30/h) in 13.16% of patients, and severe OSA (> 30/h) in 4.51% of patients. So the mild form of sleep-disordered breathing was the most common in the study population. Significant differences in terms of comorbidities and anthropometric features were seen between mild and moderate, between moderate and severe, and between mild and severe OSA. Again, a non-significant trend in heart rate being lower in mild than in moderate OSA was observed (75.4 ± 18.7 vs. 80.7 ± 16.7; $p = 0.06$).

In terms of sleep study parameters, we observed that mean AHI was 8.6 ± 2.5/h, 20.9 ± 3.7/h, and 43.4 ± 15.8/h in the mild, moderate and severe OSA groups, respectively. Mean oxygen saturation was higher in the mild vs. the severe group (93.1 ± 1.7 vs. 91.2 ± 2.8%; $p = 0.07$), and was higher in moderate vs. severe group (93.4 ± 1.6 vs. 91.2 ± 2.8%; $p = 0.04$). Also total desaturation time during the whole sleep study was shorter in mild vs. severe group (20.4 ± 32.4 vs. 105.8 ± 87.0 min; $p < 0.01$), and moderate vs. severe group (22.5 ± 28.6 vs. 105.8 ± 87.0 min; $p < 0.01$). Similarly, lowest registered oxygen saturation was found in mild vs. severe group (83.1 ± 5.4 vs. 65.9 ± 11.4%; $p < 0.01$), and moderate vs. severe group (81.6 ± 5.5 vs. 65.9 ± 11.4; $p < 0.01$). There were no further differences between the three groups observed (Table 3).

## DISCUSSION

The prevalence of OSA in the general population is estimated to be approximately 24% in men and 9% in women between 30 and 60 years of age [16]. In Poland, epidemiology data in this area is limited, but it seems that OSA prevalence is oscillating in a similar range. In a slightly older group of patients at 41 to 72 years of age, sleep disordered breathing was present in 36.5% and 18.5% of screened men and women, respectively. As for the AF prevalence, most sources state that it is about 2% in the general population and rises with age. In people of 40 to 50 years, it is only 0.5%, while in 80-year-olds it is between 5% and 15% [17, 18]. Recently, mostly due to a greater accessibility of devices for long-term heart rate monitoring, it has been suggested that in fact AF prevalence is considerably higher than previously thought, but there is a lack of consensus as to whether lone, asymptomatic, device-detected AF should be treated similarly to 'classical' AF [19].

In patients with AF, OSA occurs more often than in the general population. Different analyses show OSA to be present in from 32% to 49% of AF patients [20]. In our study, OSA

Table 2. Comparison of patients with and without obstructive sleep apnoea (OSA)

| Parameter | OSA (n = 121) | No OSA (n = 145) | P |
|---|---|---|---|
| Apnoea–hypopnoea index [/h] | 15.6 ± 12.1 | 1.9 ± 1.4 | – |
| Lowest $SpO_2$ [%] | 81.6 ± 7.2 | 86.5 ± 5.0 | **< 0.0001** |
| Male sex | 85 (70.2%) | 88 (60.7%) | 0.13 |
| Age [years] | 59.6 ± 8.0 | 56.0 ± 11.4 | **0.02** |
| Body mass index [kg/m$^2$] | 30.9 ± 5.4 | 28.7 ± 4.4 | **< 0.01** |
| Neck circumference [cm] | 41.2 ± 3.8 | 39.3 ± 3.3 | **< 0.01** |
| Waist circumference [cm] | 108.5 ± 13.1 | 107.7 ± 85.4 | **< 0.0001** |
| Systolic BP [mm Hg] | 132.8 ± 16.8 | 130.9 ± 16.7 | 0.36 |
| Diastolic BP [mm Hg] | 82.0 ± 11.5 | 79.7 ± 10.7 | 0.10 |
| Heart rate [bpm] | 77.3 ± 17.9 | 73.3 ± 13.5 | 0.06 |
| Prior MI | 11 (9.1%) | 11 (7.6%) | 0.83 |
| Diabetes mellitus | 12 (9.9%) | 10 (6.9%) | 0.50 |
| Hypertension | 94 (77.7%) | 100 (69.0%) | 0.15 |
| Gout | 12 (9.9%) | 6 (4.1%) | 0.10 |
| Smoking | 10 (8.3%) | 18 (12.4%) | 0.37 |
| Alcohol consumption | 13 (10.7%) | 10 (6.9%) | 0.37 |
| Prior stroke | 14 (11.6%) | 8 (5.5%) | 0.12 |
| Dyslipidaemia | 77 (63.6%) | 90 (62.1%) | 0.90 |
| Thyroid disease | 26 (21.5%) | 42 (29.0%) | 0.21 |
| Family history of CVD | 43 (35.5%) | 61 (42.1%) | 0.40 |
| Paroxysmal atrial fibrillation | 75 (62.0%) | 110 (75.9%) | **0.02** |

$SpO_2$ — blood oxygen saturation; BP — blood pressure; MI — myocardial infarction; CVD — cardiovascular disease

occurred in 45.49% of patients: in 27.82% the disorder was classified as mild, in 13.16% as moderate, and in 4.51% as severe. This correlates with most of the data on European populations [20]. Importantly, OSA was mild in most of the patients, but our study cohort is relatively young, which may reflect progression of OSA severity with age. The increased prevalence of OSA in AF patients seems to be associated with common pathophysiology and concomitant risk factors for both diseases.

Due to the abovementioned increased sympathetic activation, oxidative stresses, endothelial dysfunction, and left atrial overload and stretch, AF is partially a consequence of OSA, and hypertension secondary to it [21, 22]. Also apnoea-induced hypoxia, intrathoracic pressure shifts, inflammation, and autonomic instability leading to diastolic dysfunction, left atrial enlargement, and electrical remodelling are thought to cause aberrant conduction in atria, and as a consequence AF [20]. The described relationship is so strong, and important in the clinical practice, that we recently postulated OSA and AF to be not only concomitant diseases, but in fact part of a clinical syndrome [23]. The present study was not designed to establish which mechanisms were responsible for OSA and AF comorbidity in the described cohort, therefore data on oxidative stress, endothelial function markers or echocardiographic parameters are not presented and were not analysed. Nevertheless, we showed that OSA patients are more likely to suffer from persistent rather than paroxysmal AF. Transition from paroxysmal to persistent AF is thought to be a part of the natural course of arrhythmia [11]. Therefore we can speculate that in OSA patients, atrial remodelling was more advanced and severe than in patients without the disease, which caused the higher prevalence of persistent AF.

Apart from these mechanisms, OSA and AF are simply likely to coexist for example because of very common factors such as obesity and older age playing crucial roles in the pathogeneses of both diseases. Both AF and OSA are more likely to occur in older patients. Our study population was fairly young (mean age, 57.6 ± 10.1 years), but nevertheless OSA affected almost half of the patients, which shows that in the general population, the OSA — AF coexistence can be much more frequent, and a larger scale problem than it was thought to be [16–18].

Body mass is another factor positively correlated with AF and OSA prevalence. It has been suggested that up to one in four AF patients should be diagnosed with obesity, and large population trials show the mean BMI in AF to be approxi-

Table 3. Differences between the groups according to obstructive sleep apnoea (OSA) severity

| Parameter | Mild OSA (n = 74) | Moderate OSA (n = 35) | Severe OSA (n = 12) |
|---|---|---|---|
| Apnoea–hypopnoea index [/h] | 8.6 ± 2.5 | 20.9 ± 3.7 | 43.4 ± 15.8 |
| Mean $SpO_2$ [%] | 93.1 ± 1.7 | 93.4 ± 1.6 § | 91.2 ± 2.8 § |
| Total desaturation time [min] | 20.4 ± 32.4 # | 22.5 ± 28.6 § | 105.8 ± 87.0 #, § |
| Lowest $SpO_2$ [%] | 83.1 ± 5.4 # | 81.6 ± 5.5 § | 65.9 ± 11.4 #, § |
| Male sex | 55 (74.3%) | 24 (68.6%) | 6 (50.0%) |
| Age [years] | 59.6 ± 8.5 | 60.0 ± 7.2 | 58.6 ± 7.0 |
| Height [cm] | 172.0 ± 9.0 | 173.2 ± 11.6 | 169.8 ± 11.3 |
| Weight [kg] | 89.2 ± 18.1 | 93.8 ± 15.1 | 99.4 ± 22.0 |
| Body mass index [kg/m$^2$] | 30.1 ± 5.5 | 31.3 ± 4.6 | 34.4 ± 6.3 |
| Neck circumference [cm] | 41.1 ± 3.9 | 41.1 ± 3.4 | 42.0 ± 4.5 |
| Waist circumference [cm] | 106.9 ± 13.2 | 109.4 ± 11.2 | 116.9 ± 15.8 |
| Systolic BP [mm Hg] | 133.2 ± 16.6 | 132.1 ± 18.3 | 131.7 ± 15.1 |
| Diastolic BP [mm Hg] | 82.8 ± 10.7 | 81.0 ± 12.9 | 79.6 ± 12.1 |
| Heart rate [bpm] | 75.4 ± 18.7 | 80.7 ± 16.7 | 79.6 ± 15.8 |
| Prior MI | 6 (8.1%) | 3 (8.6%) | 2 (16.7%) |
| Diabetes mellitus | 4 (5.4%) | 6 (17.1%) | 2 (16.7%) |
| Hypertension | 55 (74.3%) | 30 (85.7%) | 9 (75.0%) |
| Gout | 8 (10.8%) | 1 (2.9%) | 3 (25.0%) |
| Smoking | 6 (8.1%) | 3 (8.6%) | 1 (8.3%) |
| Alcohol consumption | 6 (8.1%) | 6 (17.1%) | 1 (8.3%) |
| Prior stroke | 9 (12.2%) | 4 (11.4%) | 1 (8.3%) |
| Peptic ulcer disease | 16 (21.6%) | 4 (11.4%) | 2 (16.7%) |
| Dyslipidaemia | 46 (62.2%) | 22 (62.9%) | 9 (75.0%) |
| Thyroid disease | 17 (23.0%) | 5 (14.3%) | 4 (33.3%) |
| Family history of CVD | 24 (32.4%) | 15 (42.9%) | 4 (33.3%) |

Statistical significance ($p < 0.05$): *significant difference between mild and moderate OSA group; #significant difference between mild and severe OSA group; §significant difference between moderate and severe OSA group; $SpO_2$ — blood oxygen saturation; Total desaturation time — time of blood oxygen saturation < 90%; BP — blood pressure; MI — myocardial infarction; CVD — cardiovascular disease

mately 27.5 kg/m$^2$ with translates to overweight [24]. Even more data is available on the association between OSA and obesity. It has even been described in detail how substantial weight gain causes increases in OSA risk and severity. With every additional weight gain equal to standard deviation from the population mean, the prevalence of OSA rises fourfold, to reach a prevalence of over 90% in patients with a BMI of ≥ 60 kg/m$^2$ [16, 25]. Also every weight gain of 10% of the initial body weight results in a 32% increase in the AHI, and causes a six-fold increase in the risk for developing OSA of moderate or higher intensity [26].

These findings are consistent with findings from the present study. Our study population's mean BMI was high (29.7 ± 5.0 kg/m$^2$) which meets the criteria for an overweight diagnosis, and is almost at the cut-off point for obesity. Moreover, we showed that BMI was significantly higher in OSA than in non-OSA patients, along with other parameters associated with body mass and fat deposition patterns. Patients with OSA had also a larger waist circumference and neck circumference. This second parameter has particular prognostic significance in screening patients for OSA. It has been established that patients with larger (and shorter) necks are more likely to suffer from OSA [27]. This is attributable to fat deposited around the airways, which narrows their lumen, makes them limp and susceptible to collapse, and causes apnoea or hypopnoea episodes.

After dividing OSA patients according to disease severity, we no longer observed differences in the baseline characteristics including age, BMI, and waist or neck circumference. On the other hand, differences in parameters like total desaturation time or lowest registered blood oxygen saturation became especially apparent between severe OSA and the other two groups. This indicates that clinical features of patients can be useful in establishing the risk of OSA, but cannot different-

iate whether the disease will be severe or only mild. based on a clinical manifestation of the disease. Eventhough apart from tradicional OSA-screening tool like Epworth Sleepiness Scale or the Berlin Questionnaire, we now have novel tools independent of patient-caused bias and based only on clinical parameters, such as OSACS score, polysomnography remains the only gold standard method for OSA diagnosis confirmation [28].

Another important issue is the fact that OSA not only contributes to AF development, but also interferes with its treatment. Firstly, as described above, OSA promotes a hypercoagulative state and is associated with an increased risk of stroke, which is especially important in AF patients due to their increased residual stroke risk [29]. Secondly, in patients with rhythm control strategy, OSA diminishes the response to anti-arrhythmic drugs and promotes AF recurrence after successful AF ablation [30, 31]. An important issue not yet implemented in clinical practice guidelines is a strategy for ablation scheduling in OSA patients. Given that OSA treatment may reduce AF burden and/or symptoms, making ablation unnecessary, or in the case of ablation improve procedure success rates, it is reasonable to consider treating OSA prior to ablation [32]. Therefore, early detection and treatment of OSA in AF patients is crucial for proper management planning.

### *Limitations of the study*

The present study concerns a topic about which there is not a lot of data in the literature. However, there are some limitations to the study. Firstly, it is limited only to the experience of a single centre, therefore cannot be predictive for the situation in the whole population. Secondly, the study population consisted of patients pre-qualified to AF ablation. This group of patients is relatively small and differs in many aspects from the general AF patient population. Patients qualified for ablation in general will be younger and have fewer comorbidities than most other AF patients. As the prevalence of OSA is well known to rise with age, the OSA burden in the current study may be lower than in the general AF population.

## CONCLUSIONS

OSA is highly prevalent in patients with AF, and affects approximately half of this population. OSA patients are more likely to be older, to have higher BMI, and greater waist and neck circumferences. Persistent AF is the more common form of the arrhythmia in patients with OSA, while patients without OSA are more likely to have paroxysmal AF, which can be a partial indicator that atrial remodelling is more advanced in OSA patients. The present study highlights the possibility of massive underdiagnosing of OSA in AF patients. Clinicians should focus on screening for OSA in AF patients, because early diagnosis and treatment helps in choosing more suitable therapeutic options and better controlling both diseases.

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# Obturacyjny bezdech senny u pacjentów z migotaniem przedsionków: częstość występowania i charakterystyka kliniczna polskiej populacji

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**Streszczenie**

**Wstęp:** Obturacyjny bezdech senny (OSA) i migotanie przedsionków (AF) to dwa schorzenia szeroko rozpowszechnione w populacji ogólnej. Występowanie OSA wiąże się z narastaniem zmian hemodynamicznych, stresem oksydacyjnym i powstawaniem uszkodzeń śródbłonka, co wiąże się z postępowaniem remodelingu serca i naczyń krwionośnych. Wszystkie te procesy mogą, poprzez propagowanie niestabilności elektrycznej miokardium, przyczyniać się do powstawania AF, wyzwalać jego napady lub pogarszać przebieg już istniejącej choroby. Współwystępowanie OSA i AF wpływa negatywnie na przebieg obu chorób, powodując ich wzajemne zaostrzanie się. Dlatego też pacjenci z AF powinni być rutynowo kontrolowani pod względem występowania zaburzeń oddychania podczas snu, takich jak OSA, ponieważ będą one wpływać na wybór strategii postępowania z leczonymi pacjentami.

**Cel:** Celem badania było określenie częstości występowania OSA w populacji osób z AF i opisanie charakterystyki klinicznej pacjentów ze współistniejącymi OSA i AF.

**Metody:** Do badania włączono 289 kolejnych pacjentów hospitalizowanych w Klinice Kardiologii trzeciego stopnia referencyjności, z powodu AF. Oprócz typowych badań klinicznych i biochemicznych wykonywanych standardowo u każdego pacjenta kwalifikowanego do ablacji podłoża AF u wszystkich osób przeprowadzono dodatkowo całonocne badanie snu mające na celu wykrycie OSA. Zgodnie z obowiązującymi obecnie rekomendacjami bezdech definiowano jako przerwę w przepływie powietrza trwającą 10 s, natomiast spłycenie oznaczało przejściowy spadek przepływu powietrza w drogach oddechowych o > 50% w stosunku do wartości wyjściowych, trwający co najmniej 10 s z następczym spadkiem saturacji krwi o co najmniej 4%. Rozpoznanie OSA ustalano w momencie zarejestrowania u pacjenta wskaźnika AHI (*apnoea–hypopnoea index*) ≥ 5 na godzinę.

**Wyniki:** Kryteria włączenia i wyłączenia z badania spełniło 266 pacjentów (65,0% mężczyzn w średnim wieku 57,6 ± 10,1 roku). OSA zarejestrowano u 121 (45,49%) chorych. Pacjenci z OSA byli istotnie starsi (59,6 ± 8,0 *vs*. 56,0 ± 11,4 roku; p = 0,02), mieli wyższy wskaźnik masy ciała (BMI; 30,9 ± 5,4 *vs*. 28,7 ± 4,4 kg/m$^2$; p < 0,01) oraz większy obwód szyi (41,2 ± 3,8 *vs*. 39,3 ± 3,3 cm; p = 0,0001) i obwód talii (108,5 ± 13,1 *vs*. 107,7 ± 85,4 cm; p < 0,0001) niż pacjenci, u których nie występował OSA. Nie stwierdzono różnic między grupami w zakresie skurczowego i rozkurczowego ciśnienia tętniczego oraz częstości występowania chorób współistniejących (p > 0,05). Napadowe AF rzadziej stwierdzano u chorych z OSA niż u osób bez tego schorzenia (62,0% vs. 75,9%; p = 0,02). Przyporządkowanie pacjentów ze zdiagnozowanym OSA do grup w zależności od natężenia choroby pokazało, że osoby z postacią łagodną OSA (AHI ≥ 5/h i < 15/h) stanowili 27,82% badanej populacji, z postacią umiarkowaną OSA (AHI ≤ 15/h i ≥ 30/h) — 13,16% populacji, a z postacią ciężką (> 30/h) — 4,51% populacji. Nie zanotowano istotnych statystycznie różnic w zakresie częstości występowania chorób współistniejących oraz rozkładu innych cech klinicznych między pacjentami z postacią łagodną i umiarkowaną, między umiarkowaną i ciężką oraz między postacią łagodną i ciężką OSA.

**Wnioski:** Częstość występowania OSA w grupie pacjentów z AF w populacji polskiej jest bardzo wysoka — dotyczy ok. 50% chorych. Pacjenci z OSA są starsi, charakteryzują się wyższym BMI oraz większym obwodem talii i szyi niż osoby bez OSA. U pacjentów z OSA AF częściej przyjmuje formę przetrwałą niż w grupie bez zaburzeń oddychania w czasie snu, w której dominuje napadowa forma arytmii.

**Słowa kluczowe:** obturacyjny bezdech senny, migotanie przedsionków, epidemiologia

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