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Predictive factors for severe obstructive sleep apnea – a single-hospital retrospective study

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Abstract

Introduction and Objective. Severe obstructive sleep apnea (OSA), defined by the Apnea Hypopnea Index as AHI ≥30, is associated with increased cardiovascular, metabolic, and neurocognitive risks but is frequently overlooked in primary care. The aim of this study is to identify the predictors of severe OSA, and to derive a simple, easy-to-use a clinical model as a guide for pre-specialist triage.

Materials and Method. A retrospective single-centre study was carried out of 278 consecutively hospitalized adults assessed for sleep-disordered breathing (Lublin, 2018–2020). Polygraphy/polysomnography provided AHI and oximetry indices. Receiver operating characteristics (ROC) analyses identified thresholds for continuous variables; predictors were tested in univariate and multivariable logistic regression. Primary outcome: severe OSA (AHI ≥30).

Results. Severe OSA occurred in 56.8% of patients (158/278). ROC analysis suggested cut-offs at: age ≥55 years, body mass index (BMI) ≥30 kg/m^2, Epworth Sleepiness Scale (ESS) ≥10, and High Dense Lipoprotein (HDL) ≤53 mg/dL (best performance for BMI: area under curve (AUC) 0.756; sensitivity 70.9%, specificity 70.0%). In multivariable models, independent predictors included: BMI ≥30 (OR 5.37, 95% CI 2.70–10.70), age ≥55 (OR 2.66, 95% CI 1.36–5.20), and ESS ≥10 (OR 2.77, 95% CI 1.46–5.21). A three-item rule (age, BMI, ESS) produced predicted probabilities over 50% for several combinations, and overall discrimination was approximately 0.75.

Conclusions. Obesity is the strongest predictor of severe OSA, with additional influences from older age and daytime sleepiness. A simple three-item rule may assist primary care in triaging referrals for sleep testing; external validation is necessary.

Key words

primary care, obstructive sleep apnea, BMI, Epworth Sleepiness Scale, risk prediction, severe OSA

INTRODUCTION AND OBJECTIVE

Obstructive sleep apnea (OSA) is a common condition which remains under-diagnosed in many countries. An early epidemiological study by Punjabi et al. estimated its prevalence at 3–7% in the general adult population, with an Apnea-Hypopnea Index (AHI) ≥ 5 [1]. Recent meta-analyses show an overall prevalence of 9–38%, with moderate-to-severe OSA (AHI ≥ 15) affecting 6–17%, and increasing sharply among older adults (up to 49%) [2]. A significant indicator of OSA is the AHI, which measures the number of apneas per hour of sleep. Another key issue is the necessity of screening tests to detect individuals with severe OSA (AHI ≥ 30), as this condition is associated with significantly more complications than mild or moderate conditions (AHI < 30) [1].

OSA is a significant risk factor for motor vehicle accidents, with data showing a 1.2- to 4.9-fold increased risk of accidents compared to healthy individuals [3]. In a large population-based study of over 800,000 people, OSA was associated with about a 17% higher risk of accidents [4]. Additionally, OSA is an independent risk factor for cardiovascular disease. A 2022 meta-analysis indicated increased risks of cardiovascular disease (OR≈1.71), stroke (OR≈1.86), coronary heart disease (OR≈1.48), and overall mortality (OR≈1.77) [5]. In another study, severe OSA was linked to a 1.9-fold increase in all-cause mortality and a 2.65-fold increase in cardiovascular death [6].

The main challenge is that diagnosing OSA usually requires type 3 polygraphy (PG) or polysomnography (PSG) in a hospital, which often leads to long waiting times and delays. A potential solution is a commercial home-based polygraph test that patients can perform themselves. However, since it is expensive for patients, this option is less frequently used. Additionally, in uncertain cases, a polysomnography in a hospital is still necessary to confirm the diagnosis [7].

Another problem is that primary care doctors often struggle to identify patients with OSA because they frequently lack accessible tools to assess OSA risk, and determine who needs a referral for sleep testing. From the patient's perspective, the primary care physician plays a key role in diagnosis. Different specialists often treat patients with severe obesity, resistant hypertension, or depression, without realizing that OSA could be the underlying cause of the issues. Early detection of patients at risk for severe OSA is crucial, especially in family medicine settings where most patients first present with non-specific symptoms like fatigue, morning headaches, or poor sleep

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The aim of the study is to identify predictors of severe OSA and to derive a simple, easy-to-use clinical model to guide pre-specialist triage.

MATERIALS AND METHOD

Study design. A retrospective study was conducted using the medical records of patients hospitalized at the Institute of Rural Health in Lublin, Poland, from 2018 – 2020, due to suspected sleep-disordered breathing. Of over 300 available medical histories, 278 cases which met the diagnostic criteria for OSA and had complete medical documentation were included in the final analysis. The items most frequently missing from the medical records were selected laboratory results (haematology and inflammation markers) and numerical AHI values.

Patients were included if they had a diagnosis of OSA based on an AHI greater than 15 or 5 with clinical symptoms. Special focus was given to patients with severe OSA, defined as AHI ≥ 30. Inclusion and exclusion criteria are shown in Table 1. The study received ethical approval from the Bioethics Committee of the Medical University of Lublin (Approval No. KE-0254/57/03/2022).

Table 1. Inclusion and exclusion criteria for the study

Inclusion criteria	Exclusion criteria from the study
Age >= 18 years	Age < 18 years
Confirmed diagnosis of OSA by polysomnography or polygraphy	No confirmation of OSA diagnosis
Hospitalization in 2018 – 2020	Hospitalization in a different time period
Availability of all necessary information in the documentation	Lack of complete medical records

Collected data included demographic factors (age, gender, rural vs. urban residence), anthropometric measurements: height, weight, body mass index (BMI); comorbidities: hypertension, type 2 diabetes, chronic obturative pulmonary disease (COPD), asthma, coronary artery disease, atrial fibrillation, psychiatric disorders, tobacco use; pharmacological treatments (use of ACE inhibitors/sartans, beta-blockers, acetylsalicylic acid); laboratory results: haematocrit, haemoglobin, RBC, WBC, neutrophils, monocytes, CRP, creatinine, lipid profile, serum sodium, and potassium; and the score on the Epworth Sleepiness Scale (ESS). All patients underwent PG or PSG, providing detailed information about OSA, including AHI, Oxygen Desaturation Index, and time spent with oxygen saturation below 90%. The focus was on whether the values of individual quantitative variables can predict the occurrence of AHI ≥ 30 and, additionally, with the aim of identifying the cut-off points of these variables that best predict AHI ≥ 30.

Sleep studies followed the American Academy of Sleep Medicine Guidelines. PSG included EEG, EOG, chin EMG, airflow, respiratory effort, pulse oximetry, and body position; type-3 PG included airflow, respiratory effort, and pulse oximetry. The research database lacked a structured field for modality (PSG/PG), therefore counts by modality are unavailable.

Statistical analysis was performed in Statistica 9.1 and PQStat 1.8.2. In the first stage, receiver operating characteristic (ROC) analysis was performed to identify cutoff points for continuous variables (age, BMI, ESS), beyond which the risk of severe OSA significantly increased. In the second stage, univariate and multivariate logistic regression analyses were used to determine significant predictors, reported as odds ratios (ORs) with 95% confidence intervals (CIs). A p-value < 0.05 was regarded as statistically significant.

RESULTS

Socio-demographic characteristics. A total of 278 patients were included in the analysis: 67 women (24.1% of respondents) and 211 men (75.9% of respondents); mean height – 1.73 m ± 0.10; mean weight – 94.47 kg ± 21.65; average age – 55.3 ± 11.7 years; and mean BMI – 31.7 ± 6.1 kg/m^2. Severe OSA was diagnosed in 158 patients (56.8%), moderate OSA (AHI 15–29) in 69 (24.8%), and mild OSA (AHI 5–14) in 51 (18.4%).

The most common comorbidities included arterial hypertension (59%), dyslipidaemia (73%), type 2 diabetes (17%), coronary artery disease (13.3%), atrial fibrillation (7.9%), asthma (7.2%), chronic obstructive pulmonary disease (COPD) (5%), and deviated nasal septum (past or present) (18.4%). The vast majority of patients (around 90%) reported snoring as one of their main symptoms.

Among the examined patients, 32 (11.5%) were of normal body weight, 98 (35,3%) were overweight, and 148 (53.2%) were obese. Impaired glucose metabolism (type 2 diabetes, impaired fasting glucose, impaired glucose tolerance) was found in 29% of the patients. Current tobacco use was reported by 21.6%, previous use by 20.9%, and 57.6% were non-smokers. In the study cohort, the most frequently used medications were angiotensin-converting inhibitors (39.2%), β-blockers (34.5%), and acetylsalicylic acid (18.7%). The most common medication used in patients with diabetes was metformin. According to the available records, none of the patients received pharmacological treatment for obesity. However, the medication taken by participants did not show a statistical difference in analyses ($p > 0.05$) and was therefore not included in subsequent analyses.

Optimal predictive thresholds for severe OSA-ROC analysis. Receiver operating characteristic (ROC) analysis was conducted for continuous variables, using an AHI of 30 or higher as the dependent variable (Tab. 2). Statistical significance was observed for age, BMI, CRP, HDL level, and ESS score. The optimal cutoff points with the highest diagnostic performance were: age ≥ 55 years, BMI ≥ 30 kg/m^2, ESS ≥ 10 points, CRP ≥ 1,26 and HDL ≤ 53. The highest sensitivity and specificity were achieved for the BMI parameter, sensitivity 70.89% and specificity 70.00%.

The analysis of CRP (in ROC curves) was statistically significant, but the cut-off point was 1.26, which falls within the normal range (0–5 mg/dL). Therefore, this variable was not included in further analyses.

Univariate logistic regression analysis of risk factors for severe OSA. In univariate logistic regression (Tab. 3) several factors were associated with higher odds of severe OSA (Tab. 3). Obesity showed the strongest effect: overall for BMI ≥30 kg/m^2 the OR was 5.681 (95% CI 3.378–9.554), with a clear dose–response across classes: class I – 4.687 (1.912–11.491), class II – 9.533 (3.366–27.004), and class III – 13.200 (3.612–48.233). Age ≥55 years was associated with

Table 2. ROC curve analysis results for the occurrence of AHI ≥ 30, considering age, BMI, ESS, inflammatory markers, haemoglobin concentration, hematocrit, lipid profile and others

	AUC	95% CI	p	cut-off point	sensitivity	specificity
Age	**0.599**	**0.532–0.666**	**0,005**	**55**	**62.82%**	**54.17%**
BMI	**0.756**	**0.699–0.813**	**<0.001**	**30.02**	**70.89%**	**70.00%**
CRP (mg/l)	**0.615**	**0.506–0.725**	**0.046**	**1.26**	**57.14%**	**67.39%**
Hb (g/dl)	0.520	0.447–0.592	0.594			
RBC (T/L)	0.537	0.463–0.611	0.321			
WBC (G/L)	0.549	0.477–0.620	0.184			
Neut (G/L)	0.566	0.495–0.637	0.073			
Limf (G/L)*	0.519	0.447–0.591	0.613			
Mono (G/L)	0.559	0.487–0.630	0.111			
PLT (G/L)*	0.547	0.475–0.619	0.201			
Na (mmol/l)	0.502	0.430–0.574	0.955			
K (mmol/l)	0.516	0.443–0.590	0.660			
Total cholesterol*	0.534	0.460–0.607	0.375			
HDL*	**0.590**	**0.516–0.665**	**0.017**	**53**	**77.62%**	**43.43%**
LDL*	0.547	0.473–0.621	0.219			
nie-HDL*	0.513	0.439–0.586	0.735			
Triglyceride	0.569	0.495–0.644	0.069			
ESS	**0.579**	**0.511–0.648**	**0.025**	**10**	**55.48%**	**61.21%**

* explanatory variable as a destimulant

nearly twice the odds (1.997, 1.231–3.240). Cardiometabolic comorbidities were also associated with severe OSA: hypertension (2.542, 1.554–4.159) and type 2 diabetes (2.286, 1.153–4.534). Daytime sleepiness (ESS ≥10) increased the odds (1.874, 1.151–3.052), and low HDL (≤53) did as well (2.664, 1.523–4.659). The remaining variables were not statistically significant (Reference Categories: BMI <30 kg/m^2; age <55 years; no hypertension; ESS <10; HDL >53; no diabetes).

Multivariable analysis of clinical and metabolic predictors of severe OSA. A multivariate model was constructed which included only the predictors that proved to be statistically significant in the previously presented univariate analysis (Tab. 4). Statistical significance was demonstrated for age of 55 years or older, OR 2.661 (95% CI: 1.362–5.198); obesity, OR 5.371 (95% CI: 2.696–10.702); and an ESS score of 10 points or higher, OR 2.763 (95% CI: 1.466–5.209). Statistical significance was observed for each degree of obesity separately, but the confidence intervals were larger when comparing individuals with a BMI >= 30 to those with a BMI < 30. Obesity class I – OR 3,964 (95% CI: 1,240–12,674), Obesity class II – OR 6,221 (95% CI: 1,681–23,025) and Obesity class III – OR 19,331 (95% CI: 2,907–128,545) Hypertension, type 2 diabetes, and HDL levels did not show statistical significance in the multivariate model.

A simplified predictive model was created using the three strongest predictors (age ≥ 55, BMI ≥ 30 kg/m^2, ESS ≥ 10). Estimated probabilities of AHI ≥ 30 for different combinations are shown in Table 5.

The highest likelihood of developing severe OSA was seen in patients who met all three criteria: age of 55 years or older, BMI of 30 or higher, and ESS score of 10 or higher. A greater than 50% probability was also found for patients who met the criteria for age and BMI (but not ESS), and for those who met the criteria for BMI and ESS (but not age).

DISCUSSION

The study found that several clinical and anthropometric factors were significantly and independently associated with a higher risk of severe OSA, defined by an AHI of 30 or higher. Obesity (BMI ≥ 30 kg/m^2) emerged as the most significant predictor, linked to a more than a fivefold increased likelihood of severe OSA. This finding is consistent with recent meta-analyses showing that obesity remains a major risk factor for OSA, directly affecting the structure and collapsibility of the upper airway during sleep [8]. In the current study, it was also important to demonstrate the link between different levels of obesity and the risk of developing severe OSA. The risk was 3.96 times higher for class I obesity, 6.22 times higher for class II, and as much as 19.33 times higher for class III; however, confidence intervals, especially for class III obesity, were high (95% CI: 2,907–128,545), which makes it difficult to draw clear conclusions.

A recent Individual Participant Data meta-analysis that combined data from four population-based cohorts in the USA and Switzerland (total n = 12,860; average age 66.6 ± 7.3 years) found that 56.2% of participants had OSA (AHI ≥ 5/h). It also reported that 25.7% met the criteria for obesity (BMI ≥ 30 kg/m^2). Among those with OSA, 31.5% were obese, 44.4% were overweight (25 ≤ BMI < 30), and 23.5% had BMI < 25. Being overweight was associated with about twice the likelihood of having OSA (OR ≈ 2.18), while obesity raised the odds nearly five times (OR ≈ 4.84). The study also found that obesity was more common among women and those aged under 65 within the OSA group.

The presented cohort study from a single centre targeting severe OSA (AHI ≥ 30) showed, through multivariable analysis, an odds ratio (OR) of 0.99 for BMI 25–29.9 and an OR of 5.37 for BMI ≥ 30, using BMI < 25 as the reference. The overweight group was not statistically significant (p = 0.982), suggesting no independent risk increase after adjustment, whereas obesity showed a strong correlation. Compared to the population-based IPD meta-analysis (overweight OR ≈ 2.18, obesity OR ≈ 4.84), the findings obtained suggest a sharper risk increase, mostly at BMI ≥ 30. This difference in the overweight risk likely results from a higher concentration of severe cases in the referral cohort and covariate adjustments, such as age, gender, and cardio-metabolic factors, which may absorb BMI's effect.

Importantly, in a study from 2025, up to 23.5% of OSA patients were of normal weight or underweight, emphasizing the complex nature of the pathophysiology of OSA, and the importance of a personalized approach to diagnosis and treatment. As noted by the authors, their samples were collected from 1995 – 2015. Since obesity prevalence has increased since then, the reported rates may not reflect current prevalence of either OSA or obesity [9].

Alongside standard lifelong Continuous Positive Airway Pressure therapy, which effectively reduces symptoms in most patients but does not target the root cause of the disease (e.g. obesity), an important aspect of OSA treatment involves modern techniques for weight reduction. These methods decrease episodes of sleep apnea,that and can sometimes result in complete remission.

Table 3. Results of logistic regression analysis (univariate model) for the variable AHI ≥ 30

Analyzed variable		p	OR	95% CI	sensitivity	specificity
Sex	men (reference group: women)	0.249	1.383	0.797–2.402	78.48	27.50
Age	**55 years and more**	**0.005**	**1.997**	**1.231–3.240**	**62.82**	**54.17**
habitation	rural (reference group: city)	0.825	1.058	0.643–1.742	35.44	65.83
BMI level (reference group: normal weight)	overweight (25–29.99)	0.574	1.277	0.544–2.997	93.67	18.33
	Obesity I st. (30–34.99)	**0.001**	**4.687**	**1.912–11.491**	**70.89**	**70.00**
	Obesity II st. (35–39.99)	**<0.001**	**9.533**	**3.366–27.004**	**39.87**	**89.17**
	Obesity III st. (40+)	**<0.001**	**13.200**	**3.612–48.233**	**15.19**	**96.67**
BMI level	**≥ 30 (reference group: <30)**	**<0.001**	**5.681**	**3.378–9.554**	**70.89**	**70.00**
Diseases	**Hypertension**	**<0.001**	**2.542**	**1.554–4.159**	**68.99**	**53.33**
	Type 2 diabetes *	**0.018**	**2.286**	**1.153–4.534**	**36.71**	**80.83**
	impaired fasting glucose *	0.108	2.263	0.836–6.130	27.85	85.83
	impaired glucose tolerance *	0.057	3.557	0.963–13.140	6.96	97.50
	ischemic heart disease	0.483	1.290	0.633–2.627	14.56	88.33
	atrial fibrillation	0.267	1.693	0.668–4.294	9.49	94.17
	bronchial asthma	0.864	0.923	0.370–2.304	93.04	7.50
	COPD	0.106	2.918	0.796–10.703	6.96	97.50
	nasal septum deviation	0.751	1.105	0.597–2.046	18.99	82.50
	chronic inflammation in the respiratory tract	0.596	1.230	0.572–2.644	12.03	90.00
	mental disorders	0.930	0.963	0.421–2.205	91.14	9.17
	eGFR < 60	0.979	0.986	0.332–2.928	94.52	5.56
	hyperlipidaemia	0.150	1.624	0.839–3.144	85.52	21.57
	snoring	0.427	2.661	0.238–29.696	99.37	1.67
Smoking cigarettes	smokers (reference group: non-smokers and ex-smokers)	0.394	1.290	0.718–2.315	23.42	80.83
	ex-smokers (reference group: non-smokers)	0.577	1.189	0.647–2.184	44.94	60.83
	smokers (reference group: non-smokers)	0.332	1.350	0.736–2.475	23.42	80.83
	ex-smokers and smokers together (reference group: non-smokers)	0.335	1.268	0.783–2.053	44.94	60.83
ESS	**≥ 10 (reference group: under 10)**	**0.012**	**1.874**	**1.151–3.052**	**54.84**	**60.68**
HDL ≤ 53	**≤ 53 (reference group: over 53)**	**0.001**	**2.664**	**1.523–4.659**	**77.62**	**43.43**

*compared to people without diabetes and without prediabetes (no impaired fasting glucose, no impaired glucose tolerance)

Table 4. Results of logistic regression analysis (multivariate model) for the variable AHI ≥ 30

Analyzed variable	P	OR	95% CI
Age 55 and over	**0.004**	**2.750**	**1.388–5.445**
Overweight (BMI 25–29.99) (reference group: normal weight)	0.982	0.989	0.358–2.729
Obesity stage I (BMI 30–34.99) (reference group: normal body weight)	**0.020**	**3.964**	**1.240–12.674**
Obesity stage II (BMI 35–39.99) (reference group: normal body weight)	**0.006**	**6.221**	**1.681–23.025**
Obesity stage III (BMI 40+) (reference group: normal body weight)	**0.002**	**19.331**	**2.907–128.545**
BMI level ≥ 30 (reference group: normal body weight)	**<0.001**	**5.371**	**2.696–10.702**
Arterial hypertension	0.308	1.410	0.728–2.734
Type II diabetes *	0.307	0.598	0.223–1.602
Impaired fasting glucose *	0.752	0.831	0.263–2.625
Impaired glucose tolerance *	0.571	0.638	0.135–3.020
HDL ≤ 53	0.187	1.584	0.800–3.136
ESS ≥ 10	**0.002**	**2.770**	**1.455–5.276**

* compared to patients without diabetes and without prediabetes (no impaired fasting glucose, no impaired glucose tolerance)

Table 5. Prediction of the occurrence of AHI ≥ 30 with specific results of variables included in the model.

Analysed variable			Occurrence of AHI ≥ 30 (probability > 50%)	predicted probability
Age ≥ 55	BMI ≥ 30	ESS ≥ 10		
+	**+**	**+**	**YES**	**0.754**
+	**+**	**–**	**YES**	**0.576**
–	**+**	**+**	**YES**	**0.558**
–	+	–	NO	0.359
+	–	+	NO	0.332
+	–	–	NO	0.180
–	–	+	NO	0.170
–	–	–	NO	0.083

Recent reports show that tirzepatide, a new GLP-1 analogue approved in the USA for obesity, diabetes and OSA, marks a key step in understanding the link between obesity and OSA. A personalized approach to treating patients with OSA and concurrent hypertension may also be necessary; medications such as ARNI or SGLT2 inhibitors may be used even if these patients do not have conditions like heart failure or diabetes, for which these drugs are typically prescribed

[10]. It is important to highlight the increasing significance of new treatments and supportive interventions for patients with severe OSA. Recent clinical trials have shown that GLP-1 receptor agonists — especially tirzepatide — are effective not only in weight loss but also in significantly reducing AHI and inflammatory markers (e.g., CRP), which could lead to new possibilities for causal treatment [11].

There is a notable gap in the treatment of OSA with pharmacotherapy. Some studies involve scientists using experimental treatment protocols with drugs like atomoxetine and oxybutynin. Studies have produced some interesting results in reducing AHI, and improving sleep structure [12]. Non-pharmacological treatment is also very important in the case of OSA. Meta-analyses have demonstrated that aerobic exercise can decrease AHI and improve daytime sleepiness independently of weight loss, probably by enhancing respiratory mechanics and muscle tone [13].

Age also independently predicts severe OSA. Cohort studies have demonstrated that the prevalence of OSA rises with age even among non-obese individuals, likely due to reduced pharyngeal muscle tone, decreased chemoreceptor sensitivity, and neuroanatomical and metabolic changes associated with aging [14]. In the current study, being aged ≥ 55 years was associated with more than doubling the risk of AHI ≥ 30.

Excessive daytime sleepiness was also identified as a significant risk factor. Although the ESS is subjective and does not always correlate linearly with OSA severity, its prognostic value has been confirmed in relation to road traffic accidents, quality of life, and chronic fatigue [15].

Using these three variables (age, BMI, and ESS), a straightforward predictive model was developed by the authors of the current study that can assist in clinical decision-making, particularly in settings with limited access to polysomnography. Similar models are increasingly recognized in the literature as valuable tools in primary care and occupational medicine. In a study conducted in China in 2025, a model that incorporated gender, age, BMI, neck circumference and ESS was used to detect a link between the occurrence of hypertension and any form of OSA [16].

Interestingly, in the presented study, some variables, although clinically plausible, did not show significant links to severe OSA. Hypertension and diabetes showed statistical significance in univariate analysis, but lost this significance in multivariate analysis. However, many other studies confirm a strong link between hypertension, type 2 diabetes, and severe OSA [17 18]. Other factors did not show statistical significance already at the stage of univariate analysis. These included male gender, smoking tobacco, asthma, COPD, nasal septal deviation, and common cardiovascular medications (e.g., ACE inhibitors, sartans, beta-blockers). These negative results are important for clinical reasoning, as they suggest that although these factors are common in OSA patients, when considered alone, they may not significantly predict disease severity. Data in the literature on this topic shows inconsistent findings. A 2017 study found that age, gender, neck circumference, and ESS were not reliable tools for predicting OSA in individuals with moderate to severe chronic obstructive pulmonary disease [19]. In a 2021 study conducted with patients diagnosed with severe OSA at the Institute of Rural Medicine in Lublin, Poland, the lymphocyte count in the blood was found to be statistically significant in both univariate and multivariate analyses [20]. The current study, however, did not confirm the statistical significance of any blood count components in predicting the occurrence of severe OSA. Many OSA patients had vascular diseases, such as arterial hypertension (59% of patients), dyslipidaemia (73%), type 2 diabetes (17%), and coronary artery disease (13.3%). From the perspective of internal medicine and cardiology, it is essential to recognize that patients with OSA are at a significantly higher risk of cardiovascular events, including after acute coronary syndrome [21]. Another study has shown that increased platelet aggregation is more common in patients with OSA, and such patients may require a more individualized approach to treatment [22]. Some studies also suggest a link between severe OSA and increased cancer risk. The most common finding is that OSA may contribute to the development of lung cancer, which requires further investigation, but increased oncological vigilance in this population is advisable [23].

Limitations of the study. The study was a retrospective analysis conducted at a single hospital-based centre which limited the generalizability of the findings. The study included only symptomatic participants with a confirmed obstructive sleep apnea diagnosis, and therefore might not reflect the broader population. BMI was used to evaluate obesity and different weight categories and is a widely accepted standard measure of obesity. However, BMI may not always accurately reflect abdominal fat or fat around the neck. Unfortunately, the authors did not have access to such data in the medical records examined.

In recent years, incretin-based therapies (GLP-1 receptor agonists) for obesity and diabetes have become more common, with emerging reports of positive effects on OSA severity. During the study period (2018–2020), however, these treatments were not widely used and their impact could not be assessed. External validation of the model appears to be necessary.

Another issue was using two test types (PSG and PG) which could influence AHI. PG does not record EEG and relies on recording time, therefore AHI might be underestimated. If this mistake occurs, it probably affects patients similarly and tends to weaken the observed links rather than create false ones. No stratification by test type was possible because the data export lacked a structured 'modality' field. This field will be included in future datasets.

CONCLUSIONS

In the study, obesity (BMI ≥ 30 kg/m^2) was the strongest independent risk factor for severe OSA. Age ≥ 55 years and excessive daytime sleepiness were also significantly linked to an increased risk of severe OSA. Using these three variables, a straightforward predictive model was created that showed good diagnostic accuracy (AUC = 0.75), and can assist primary care physicians in prioritizing referrals for sleep studies, especially in settings with limited resources and limited access to PSG or PG.

Several clinical factors often associated with OSA (e.g., male gender, smoking, COPD, nasal septal deviation) were not significant predictors of severe disease in this cohort, which may inform more targeted screening approaches. This model can be used as a screening tool to aid primary care providers in identifying patients who should be referred

for further testing for sleep-disordered breathing. However, external validation in independent patient populations seems to be necessary before the model can be widely recommended for clinical use.

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