ORIGINAL ARTICLE

Pulmonary artery dilation indicates severe obstructive sleep apnea in patients with resistant hypertension: the Resist-POL Study

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KEY WORDS

echocardiography, main pulmonary artery, obstructive sleep apnea, resistant hypertension, right ventricle

ABSTRACT

INTRODUCTION The effect of obstructive sleep apnea (OSA) on right ventricular (RV) function and pulmonary circulation parameters is unclear.

OBJECTIVES The aim of this study was to determine whether newly diagnosed OSA and its severity has any impact on RV performance and echocardiographic parameters of pulmonary circulation in patients with true resistant hypertension.

PATIENTS AND METHODS The study included 155 patients (93 men and 62 women; mean age, 47.5 ±10 years). The apnea–hypopnea index (AHI), end-diastolic and end-systolic area of the right ventricle, main pulmonary artery diameter (MPAd) at diastole, acceleration time (AccT), tricuspid annular systolic velocity wave, and tricuspid annular plane systolic excursion were evaluated.

RESULTS Patients were divided into 4 groups: without OSA (AHI <5; n = 43), with mild OSA (AHI = 5–15; n = 45), moderate OSA (AHI = 15–30; n = 27), and severe OSA (AHI >30; n = 40). There were no differences in RV systolic function between the groups. Patients with severe OSA had a wider MPAd (26.0 ±2.6 vs 23.1 ±3.7 mm; P <0.0001) and shorter AccT (114.2 ±15.7 vs 133.4 ±22.1 ms; P <0.001) in comparison with patients without OSA. The cut-off for the best predictive value of severe OSA was an MPAd of 25 mm or higher with a sensitivity of 63.6% and specificity of 78.9%. The area under the receiver operating characteristic curve for severe OSA in relation to an MPAd of 25 mm or higher was 0.766 (95% confidence interval, 0.673–0.859; P <0.0001). Factors independently associated with an MPAd of 25 mm or higher were severe OSA and nighttime diastolic blood pressure levels.

CONCLUSIONS Our study showed a relationship between pulmonary artery dilation and the presence of newly diagnosed severe OSA. Among the parameters studied, an MPAd of 25 mm or higher turned out to be the most useful parameter in identifying patients with severe OSA.

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INTRODUCTION Patients with resistant hypertension have an increased risk of stroke, myocardial infarction, heart failure, and chronic kidney disease,[1] compared with patients with non-resistant hypertension. The most frequent medical condition in patients with resistant hypertension is obstructive sleep apnea (OSA).[2,3] OSA is a sleep-related breathing disorder associated with an increase in cardiovascular mortality and morbidity.[4,5] OSA and resistant hypertension are known to have a negative impact on left ventricular (LV) morphology and function,[6-9] whereas the effects of OSA and resistant hypertension on right ventricular (RV) morphology and systolic function and on the parameters associated with pressure in pulmonary circulation have not been clearly established.[10-13] The aim of this study was to determine whether newly diagnosed OSA and its severity have any impact on RV performance and pulmonary circulation studied by

echocardiography in patients with true resistant hypertension.

PATIENTS AND METHODS **Study population** A total of 204 patients have been enrolled in the Resist-POL study at the Department of Hypertension, Institute of Cardiology, Warsaw, Poland, between 2009 and 2011. After excluding patients with secondary hypertension, we analyzed 155 patients. The description of the population recruitment has been presented in Supplementary material online, *Figure S1*. The inclusion criteria have been published previously.[14] The exclusion criteria included a history of other cardiovascular diseases (ischemic heart disease, heart failure, transient ischemic attacks, and previous stroke), decreased estimated glomerular filtration rate of less than 60 ml/min/1.73 m^2, and previous diagnosis of diabetes mellitus. The study protocol conformed to the ethical guidelines of the 1975 Declaration of Helsinki and was approved by a local research ethics committee.

Office and ambulatory blood pressure measurements Blood pressure (BP) was measured by a trained nurse with a patient in the sitting position after a 5-minute rest, using an automated device (Omron 705IT, Omron Co., Kyoto, Japan). Based on the upper arm circumference, an appropriately sized cuff was placed on the arm with the lower edge of the cuff located 2 cm above the antecubital fossa. Three consecutive readings were taken. In all patients the ambulatory BP measurements were recorded, using SpaceLabs 90207 or 90217 (Redmond, Washington, United States). Readings were obtained every 15 minutes during the day (6:00 AM – 10:00 PM) and every 30 minutes during the night (10:00 PM – 6:00 AM). The average 24-hour, systolic BP (SBP), diastolic BP (DBP), and average 24-hour heart rate were analyzed.

Polysomnography The diagnosis of OSA was made by standard attended polysomnography with an Alice 5 device (Respironics Inc., Murrysville, Pennsylvania, United States). The polysomnographic recordings were scored manually using 30-second epochs, following Rechtschaffen and Kales' criteria for sleep and wake determination and sleep staging. Abnormal respiratory events were evaluated according to the standard criteria of the American Academy of Sleep Medicine Task Force.[15] The apnea–hypopnea index (AHI) indicating the number of apneic and hypopneic episodes per hour of sleep was calculated. Mean saturation was measured. OSA was diagnosed when the AHI exceeded 5 events per hour.

Echocardiography All patients underwent a complete transthoracic echocardiographic evaluation, using a GE Medical System Vivid 7 with a 2.5 MHz transducer (GE Vingmed Ultrasound, Horten, Norway). All studies were performed by one experienced researcher. The RV end-diastolic area (RVAD), RV end-systolic area (RVAS), fractional area change (FAC), and tricuspid annular plane systolic excursion (TAPSE) were measured according to the current guidelines.[16] The main pulmonary artery diameter (MPAd) at diastole was measured in the proximal pulmonary artery, in the parasternal short axis view at the level of the great vessels. The acceleration time (AccT) of the RV outflow tract was determined as the time elapsed from the onset of pulmonary arterial flow to the peak flow velocity.[17] The tricuspid lateral annular systolic velocity wave (S'RV) was measured by using a tissue Doppler imaging and placing the sample volume in the basal segment of the RV free wall in a 4-chamber view. All parameters were recorded according to the current guidelines.[18] The LV mass was calculated using the modified American Society of Echocardiography cube formula proposed by Devereux et al[19] and was indexed to the body surface area, in order to obtain the LV mass index (LVMI).

Statistical analysis All of the data were expressed as mean ± standard deviation, and the frequency was expressed as a percentage. Significant differences of the studied parameters between the 4 groups were determined by means of the analysis of variance (ANOVA). Multiple comparisons between the groups were performed by 1-way ANOVA with the Duncan post hoc test. The values of variables were compared between 2 groups; continuous and discrete variables were compared using the *t* test or Mann–Whitney test, and categorical variables were compared using the χ^2 test or Fisher exact test. Pearson's correlation was used to examine the correlation of variable factors with the echocardiographic parameters. The areas under the receiver operating characteristic (ROC) curves (AUCs) were calculated for the diagnostic test performance analysis. The ROC analysis was used to assess the predictive accuracy of the MPAd for detecting severe OSA. Parameters identified as statistically significant based on a univariate analysis were included in the multivariate logistic regression models to determine the combined effect of several variables on the evaluated echocardiographic parameters and severe OSA. A *P* value of less than 0.05 was considered to be statistically significant. All statistical analyses were performed with the commercially available computer software: PASW Statistics 18 (SPSS Inc., Chicago, Illinois, United States).

RESULTS The analyzed group consisted of 155 patients. The mean age of the patients was 47.5 ±10.5 years (range, 19–65 years); there were 92 men (59.4%) and 63 women (40.6%). Newly diagnosed OSA was present in 112 patients (72.3%). Patients were divided into 4 groups based on the AHI: without OSA (AHI <5; n = 43), with mild OSA (AHI = 5–15; n = 45), moderate OSA (AHI = 15–30; n = 27), and severe OSA (AHI >30; n = 40).

There were significant differences in the RVAD, RVAS, MPAd, and AccT between the groups (TABLE 1). Patients with severe OSA had

TABLE 1 Echocardiographic parameters in patients with sleep obstructive apnea classified according to the apnea–hypopnea index

Variables	OSA (AHI <5) (n = 43)	OSA (AHI = 5–15) (n = 45)	OSA (AHI = 15–30) (n = 27)	OSA (AHI >30) (n = 40)	*P* value (ANOVA)
RVAD, cm^2	15.0 ±3.6	15.8 ±3.2	16.7 ±3.2	17.7 ±4.0[a]	0.027
RVAS, cm^2	6.8 ±2.0	7.1 ±2.6	7.4 ±1.9	8.6 ±2.1[a]	0.01
MPAd, mm	23.1 ±3.1	23.5 ±2.3	24.2 ±4.9	26.0 ±2.6a	0.001
AccT, ms	132.4 ±22.9	124.1 ±22.3	130.4 ±24.5	113.1 ±16.5[a]	0.003
TAPSE, mm	22.8 ±3.7	24.1 ±3.5	24.2 ±5.0	24.2 ±5.4	0.4
FAC	0.50 ±0.4	0.56 ±0.1	0.56 ±0.2	0.45 ±0.3	0.3
S'RV, cm/s	14.4 ±2.6	15.5 ±4.2	14.9 ±3.6	16.1 ±3.9	0.2

Data are presented as mean ± SD.

a severe OSA vs no OSA

Abbreviations: AccT, acceleration time; AHI, apnea–hypopnea index; ANOVA, analysis of variance; FAC, fractional area change; MPAd, main pulmonary artery diameter, OSA, obstructive sleep apnea; RVAD, right ventricular end-diastolic area; RVAS, right ventricular end-systolic area; S'RV, tricuspid lateral annular systolic velocity wave; TAPSE, tricuspid annulus plane systolic excursion

significantly greater RVAD, RVAS, MPAd, and shorter AccT compared with patients without OSA. There were no differences in RV systolic function parameters assessed with TAPSE, FAC, and S'RV between the analyzed groups.

In the entire group, AccT significantly correlated with age (β = –0.26; *P* = 0.002), AHI (β = –0.25; *P* = 0.004), body mass index [BMI] (β = –0.35; *P* <0.0001), severe OSA (β = –0.28; *P* = 0.001), and mean saturation (β = 0.23; *P* = 0.01). In the linear regression model, the factors independently associated with AccT were severe OSA (β = –0.211; *P* = 001), BMI (β = –0.293; *P* = 0.001) and age (β = –0.184; *P* = 0.02). The MPAd significantly correlated with AHI (*r* = 0.42; *P* <0.0001), mean saturation (*r* = –0.27; *P* = 0.002), LVMI (*r* = 0.34; *P* <0.0001), and age (*r* = 0.19; *P* = 0.02).

The AUCs were calculated for the MPAd, AccT, RVAD, and RVAS for their ability to predict severe OSA. The MPAd was characterized by the best performance with an AUC of 0.77 (95% confidence interval [CI], 0.67–0.86; *P* <0.001). The other AUCs were 0.72 (95% CI, 0.62–0.83; *P* <0.001) for RVAS, 0.70 (95% CI, 0.60–0.80; *P* = 0.001) for AccT, and 0.68 (95% CI, 0.57–0.79; *P* = 0.004) for RVAD. Based on the AUC, an optimal cut-off value of an MPAd of 25 mm or higher for the prediction of severe OSA was selected. It was characterized by a sensitivity of 63.6% and a specificity of 78.9% (FIGURE 1).

Consequently, 2 groups were compared based on the MPAd: MPAd <25 mm (n = 106) and MPAd ≥25 mm (n = 49) (TABLE 2). Patients with an MPAd of 25 mm or higher were older and had a higher BMI compared with patients with an MPAd of less than 25 mm. Patients with an MPAd of 25 mm or higher had a higher AHI and mean saturation, compared with subjects with an MPAd of less than 25 mm. There were no significant differences in the use of antihypertensive drug classes between the analyzed groups (*P* = 0.42). Moreover, there were no severe pulmonary diseases diagnosed. There were no significant differences in office and 24-hour SBP, DBP, daytime SBP, DBP, and nighttime SBP levels between the groups. The only difference in BP value was noticed in nighttime DBP levels. The LVMI was significantly higher in patients with an MPAd of 25 mm or higher compared with the group with an MPAd of less than 25 mm (131.28 ±28.14 g/m2 vs 119.12 ±25.22 g/m^2; *P* = 0.01).

Following our previous analysis[14] of clinical variables associated with the presence of severe OSA, we performed a multivariate analysis including the variables that were included in the previous model (male sex, plasma glucose levels 2 hours after glucose administration, abdominal obesity, age, BMI ≥30 kg/m^2, newly diagnosed diabetes, non-dipping systolic BP, snoring, and Epworth Sleepiness Score) and added an MPAd of 25 mm or higher to those variables. In this logistic model, the factors associated with severe OSA were as follows: MPAd ≥25 mm, male sex, and plasma glucose levels 2 hours after glucose administration (TABLE 3).

To evaluate independent factors related to an MPAd of 25 mm or higher, a logistic regression model was applied. In this model, the factors independently associated with the MPAd were severe OSA and nighttime DBP levels (TABLE 4).

DISCUSSION With its frequency and concomitant risk factors, hypertension remains a significant worldwide public health challenge.[20] In a recent meta-analysis, the prevalence of resistant hypertension among patients with hypertension was estimated to be 13.72%.[21] Patients with resistant hypertension have shown a significant increase in the risk of cardiovascular events during follow-up, compared with patients with non-resistant hypertension. Several epidemiological studies have shown the concomitance of resistant hypertension and OSA.[22] The prevalence of OSA in patients with resistant hypertension was estimated

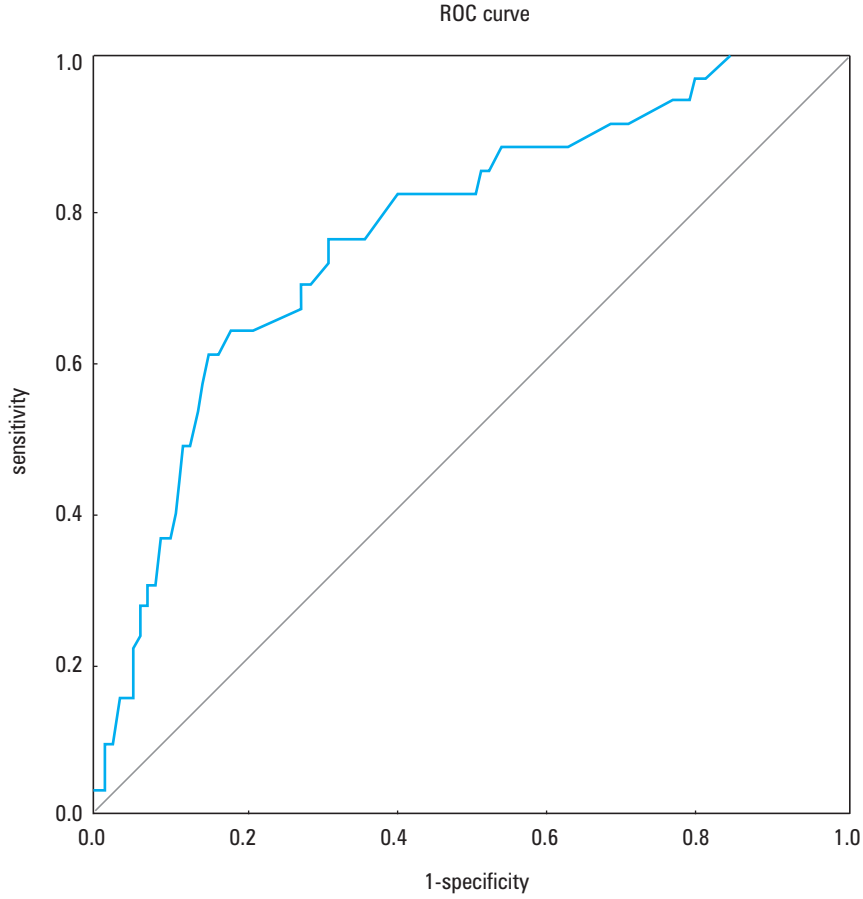

FIGURE 1 Receiver operating characteristic (ROC) curve for identification of severe obstructive sleep apnea; area under the curve for the mean pulmonary artery diameter: 0.766

TABLE 2 Demographic and clinical data in the study groups classified according to the main pulmonary artery diameter

Variables	MPAd <25 mm	MPAd ≥25 mm	*P* value
age, y	46.3 ±11.4	50.7 ±8.4	0.02
BMI, kg/m^2	29.3 ±4.9	31.3 ±4.5	0.02
AHI, events/h	13.9 ±16.2	32.9 ±28.5	<0.0001
mean saturation, %	94.7 ±1.9	93.7 ±1.9	0.007
glucose, mmol/l	5.7 ±0.8	5.9 ±0.9	0.1
metabolic syndrome, %	60.2	75.0	0.1
abdominal obesity, %	68.4	77.3	0.3
2-hour PG levels ≥7.7 mmol/l, %	23.9	27.9	0.7
eGFR, ml/min/1.73 m^2	92.0 ±19.4	86.4 ±17.9	0.1
office SBP, mmHg	158.9 ±20.5	161.3 ±26.6	0.6
office DBP, mmHg	94.5 ±13.2	99.0 ±17.1	0.2
24-hour SBP, mmHg	137.0 ±17.3	141.2 ±19.0	0.2
24-hour DBP, mmHg	83.9 ±12.8	87.7 ±13.3	0.1
daytime SBP, mmHg	142.2 ±18.5	145.7 ±20.2	0.3
daytime DBP, mmHg	87.9 ±13.0	91.3 ±14.1	0.2
nighttime SBP, mmHg	127.0 ±17.6	132.5 ±17.6	0.1
nighttime DBP, mmHg	76.0 ±12.2	81.1 ±11.6	0.03
24-hour HR, bpm	68.6 ±10.0	67.4 ±9.41	0.5

Data are presented as mean ± SD, except for categorical variables.

Abbreviations: BMI, body mass index; BSA, body surface area; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; HR, heart rate; SBP, systolic blood pressure; 2-hour PG, plasma glucose levels 2 hours after glucose administration; others, see **TABLE 1**

TABLE 3 Multivariate logistic regression model for factors associated with severe obstructive sleep apnea in presented study

Variables	Multivariate model		
	OR	95% CI	*P* value
MPAd ≥25 mm	6.54	1.594–11.858	<0.0001
male sex	6.55	2.077–20.633	0.001
2-hour PG levels, mmol/l	5.85	0.95–36.05	0.05
abdominal obesity	–	–	–
age[a]	–	–	–
obesity (BMI ≥30 kg/m^2)	–	–	–
newly diagnosed diabetes	–	–	–
non-dipping systolic BP	–	–	–
snoring	–	–	–
Epworth Sleepiness Score	–	–	–

a 10-year increase

Abbreviations: BP, blood pressure; CI, confidence interval; OR, odds ratio; others, see **TABLES 1** and **2**

TABLE 4 Univariate and multivariate logistic regression model for factors associated with a main pulmonary artery diameter of 25 mm or higher

Variables	Univariate model			Multivariate model		
	OR	95% CI	*P* value	OR	95% CI	*P* value
age[a]	1.54	1.054–2.245	0.02	–	–	–
BMI	1.09	0.013–1.180	0.02	–	–	–
nighttime DBP	1.04	1.004–1.067	0.005	1.04	1.003–1.076	0.03
mean saturation	2.58	1.312–5.985	0.006	–	–	–
severe OSA	6.54	2.810–15.239	<0.0001	5.34	2.217–13.094	<0.0001
LVMI	1.02	1.003–1.031	0.01	–	–	–

a 10-year increase

Abbreviations: LVMI, left ventricular mass index; others, see **TABLES 1**, **2**, and **3**

at up to 85%. In the Resist-POL study, the prevalence of newly diagnosed OSA was 72.1%.[14] OSA itself is a disorder associated with increased cardiovascular mortality and morbidity.[5,23] Therefore, it must be assumed that the vast majority of patients with resistant hypertension and OSA are exposed in the long term to high cardiovascular risk. Most of the published studies analyzed the impact of OSA on LV morphology and function, and the negative effects of uncontrolled hypertension and severe OSA on the left ventricle have been broadly investigated.[24] The number of published studies evaluating the influence of hypertension and OSA on RV structure and function is limited. The available data seem to be equivocal. However, those studies were performed on a different patient population. To our knowledge, our study is the first to evaluate the impact of OSA on the MPAd and RV morphology, systolic function, and pulmonary circulations in patients with resistant hypertension.

In this study, the RVAD and RVAS were the largest, the MPAd was the widest, and the AccT was the shortest in patients with severe OSA. The presented data are consistent with the study by Yang et al,[25] who grouped patients based on the duration of OSA. They established that the diameters of the right ventricle and of the main pulmonary artery on echocardiography were larger in groups where patients suffered from OSA for between 5 and 10 years and more than 10 years, compared with a control group (without OSA) (P <0.01 for both groups). These authors proved that increased RV correlated both with the duration and severity of OSA. On the other hand, Dursunoglu et al[10] found no differences in the RV end-diastolic and end-systolic diameters, between patients without OSA and patients with mild and moderate to severe OSA. Hanly et al[26] also showed no differences in the RV dimensions between non-apneic snorers and subjects with OSA.

In this study, the systolic function of RV assessed by TAPSE, FAC, and S'RV showed no significant difference between the 4 groups. Hammerstingl et al[12] found no differences in TAPSE and S'RV between patients without OSA (AHI <5) and patients with OSA (AHI >5). In addition, Guidry et al[13] did not show a correlation of RV

dysfunction with OSA. On the contrary, Dursunoglu et al[10] found a relevant correlation between the global RV dysfunction and the severity of OSA measured using the myocardial performance index. Tavil et al[11] showed that patients with hypertension coexisting with OSA had lower TAPSE and S'RV compared with the control group, but not with hypertensive patients without OSA. The discrepancy in the results may be a consequence of using a different method for systolic function assessment, as well as of the differences in patient characteristics and in the definition of OSA.

Parameters related to pulmonary circulation were also investigated. Pulmonary hypertension prevails in 12% to 34% of patients with OSA, and is associated with poor prognosis.[27-30] Therefore, it is clinically important to raise early suspicion of pulmonary arterial hypertension in patients with OSA, using noninvasive and widely accepted methods, such as echocardiography. The routine echocardiographic assessment of systolic pulmonary artery pressure relies on the tricuspid regurgitant jet velocity.[31] However, McQuillan et al[32] showed that 30% of patients did not have tricuspid regurgitant jets allowing for RV pressure assessment, making it impossible to apply this method in the whole group of patients. On the contrary, the MPAd and AccT can be measured in a vast majority of patients. Increased MPAd and decreased AccT can be used as surrogate pulmonary hypertension markers. Additionally, AccT has been shown to correlate with the tricuspid regurgitant jet velocity and invasive pulmonary artery pressures in the experimental models and in human studies of pulmonary hypertension.[33-35] Therefore, AccT was one of the parameters evaluated in this study. The linear regression model showed that the factors independently associated with AccT were severe OSA, BMI, and age.

It has been shown that an increase in the MPAd suggests pulmonary hypertension.[36] The diameter defining a dilated main pulmonary artery according to the American Society of Echocardiography guidelines was established as more than 21 mm.[18] Recent European Society of Cardiology and European Respiratory Society guidelines for the diagnosis and treatment of pulmonary hypertension suggest grading the probability of pulmonary hypertension not only based on tricuspid regurgitation at rest but also the presence of additional prespecified echocardiographic variables suggestive of pulmonary hypertension, including an MPAd exceeding 25 mm.[37] In this study, the MPAd was the widest in patients with severe OSA. Univariate and multivariate linear regression models for the factors associated with the MPAd in this study showed that only severe OSA and nighttime DBP values are independently associated with main pulmonary artery dilation (MPAd ≥25 mm). The ROC curve showed that the value of the MPAd of more than 25 mm had optimal specificity and sensitivity in the prediction of severe OSA. This may have clinical implications in the early diagnosis of severe OSA in patients with resistant hypertension. OSA is associated with repetitive nocturnal hypoxemia and hypercapnia, acute increase in the pulmonary arterial pressure, and large intrathoracic negative pressure swings. Hypoxemia induces extensive contraction of pulmonary arterioles, leading to pulmonary hypertension.[38,39] Furthermore, it may result in dilation of the main pulmonary artery. It is suggested that OSA in patients with resistant hypertension may be characterized by sodium and fluid retention, which may be related to increased venous return and subsequently lead to RV and pulmonary artery dilation. Also, during overnight recumbence rostral fluid shift contributes to the worsening of the clinical course of OSA and thus may further lead to pulmonary artery dilation.

In our study, the MPAd was independently associated with nighttime DBP and correlated with the LVMI. The latter may lead to LV diastolic impairment[40]; therefore, we speculate that an increase in the MPAd may also result from an increase in the pulmonary artery pressure caused by the LV diastolic dysfunction.

To the best of our knowledge, this is the first study that proves the impact of newly diagnosed OSA on main pulmonary artery diameter and RV morphology in patients with resistant hypertension. This finding may have clinical implications in screening for severe OSA in patients with resistant hypertension.

Our study has some limitations. The results are confined to true resistant hypertension and cannot be extrapolated to the general hypertensive population. However, we focused on the population of patients with resistant hypertension that are characterized by higher cardiovascular risk, pronounced impact of heart structure and function, and also by volume and sodium overload.[6,41] Secondly, we did not perform RV catheterization to measure systolic pulmonary artery hypertension. In our study, the RV wall thickness was not measured, because conventional echocardiography calculations with these variables are difficult and inaccurate, owing to a thin wall and marked trabeculations. In this study, the RV systolic functions were assessed by echocardiography parameters and were not corroborated by an independent method, such as magnetic resonance imaging or radionuclide ventriculography. However, we sought to simply measure enabling OSA severity stratification among patients with resistant hypertension.

In conclusion, our study showed the relationship between pulmonary artery dilation and severe newly diagnosed OSA. In this group of patients, the right ventricle was dilated with no impact on its systolic function. An MPAd of 25 mm or higher seems to be a simple new parameter that may be useful to confirm the suspicion of severe OSA in patients with resistant hypertension.

Contribution statement PD, AK, AP, AJ, and PH conceived the idea of the study. EF, JR, and PŚ

contributed to the design of the research. All authors were involved in data collection. PD and AK performed echocardiographic studies. PD, AP, AK, and AJ analyzed the data. All authors edited and approved the final version of the manuscript. AJ and PH equally contributed to the work and may be both considered as equal "senior" authors.

Supplementary material online Supplementary material is available with the online version of the article at www.pamw.pl.

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ARTYKUŁ ORYGINALNY

Poszerzenie pnia płucnego sugerujące obecność ciężkiego obturacyjnego bezdechu sennego u chorych z opornym nadciśnieniem tętniczym – badanie Resist-POL

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SŁOWA KLUCZOWE

echokardiografia, obturacyjny bezdech senny, oporne nadciśnienie tętnicze, pień płucny, prawa komora

STRESZCZENIE

WPROWADZENIE Wpływ obturacyjnego bezdechu sennego (OBS) na funkcję prawej komory (PK) i parametry związane z ciśnieniem płucnym jest niejasny.

CELE Celem badania było ustalenie, czy nowo rozpoznany OBS oraz stopień jego zaawansowania wpływa na funkcje PK i parametry echokardiograficzne związane z ciśnieniem płucnym u chorych na prawdziwie oporne nadciśnienie tętnicze.

PACJENCI I METODY W badaniu wzięło udział 155 chorych (93 mężczyzn oraz 62 kobiety; średni wiek: 47,5 ±10 lat). Oceniono wskaźnik bezdechów i oddechów spłyconych (*apnea–hypopnea index* – AHI), rozkurczową i skurczową powierzchnię PK, szerokość pnia płucnego (*main pulmonary artery diameter at diastole* – MPAd), czas akceleracji (*acceleration time* – AccT), prędkość skurczową pierścienia trójdzielnego i skurczową prędkość pierścienia trójdzielnego.

WYNIKI Wyodrębniono 4 grupy chorych: bez OBS (AHI <5, n = 43), z łagodnym OBS (AHI 5–15, n = 45), z umiarkowanym OBS (AHI 15–30, n = 27) i z ciężkim OBS (AHI >30, n = 40). Nie stwierdzono różnic w parametrach funkcji skurczowej PK między grupami. U chorych z ciężkim OBS w porównaniu z chorymi bez OBS stwierdzono szerszy MPAd (26,0 ±2,6 *vs* 23,1 ±3,7 mm; p <0,0001) i krótszy AccT (114,2 ±15,7 *vs* 133,4 ±22,1 ms; p <0,001). Punktem odcięcia charakteryzującym się najlepszą wartością obecności ciężkiego OBS była MPAd ≥25 mm z czułością wynoszącą 63,6% oraz specyficznością – 78,9%. Pole powierzchni pod krzywą ROC wartości predykcyjnej ciężkiego OBS dla MPAd ≥25mm wynosiło 0,766 (95% CI: 0,673–0,859; p <0,0001). Czynnikami niezależnie związanymi z MPAd ≥25 mm były ciężki OBS oraz wartości rozkurczowego ciśnienia tętniczego w nocy.

WNIOSKI Nasze badanie wykazało związek między poszerzeniem pnia płucnego i obecnością świeżo rozpoznanego ciężkiego OBS. Pośród ocenianych parametrów MPAd ≥25 mm okazał się najbardziej przydatny do wyodrębnienia pacjentów z ciężkim OBS.

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