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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

The influence of obstructive sleep breathing disturbances on echocardiographic and pulmonary haemodynamic parameters in patients with dilated cardiomyopathy

Damian Kawecki[1], Celina Wojciechowska[1], Wojciech Jacheć[1], Krystyna Krzemień-Wolska[1], Janusz Dola[1], Wojciech Ścierski[2], Izabela Kawecka[2], Andrzej Tomasik[1], Tomasz Brzostowicz[3], Beata Morawiec[1], Ewa Nowalany-Kozielska[1]

[1]2nd Department of Cardiology in Zabrze, Medical University of Silesia, Katowice, Poland
[2]Department of Otorhinolaryngology in Zabrze, Medical University of Silesia, Katowice, Poland
[3]Department of Cardiology, Medical Centre, Opole, Poland

Abstract

Background: It is important to identify the clinical indicators of poor prognosis and treatable conditions that might contribute to the progression of heart failure (HF) and pulmonary hypertension (PH) in the group of patients with dilated cardiomyopathy (DCM) and concomitant obstructive sleep apnoea (OSA).

Aim: To evaluate the influence of OSA on echocardiographic and haemodynamic parameters in patients with DCM, and the outcome in long-term follow-up.

Methods: We enrolled patients with DCM and severely impaired ejection fraction (EF < 30%). Each patient underwent polygraphy, echocardiography, and right heart catheterisation. Subjects were divided into groups based on the apnoea–hypopnoea index (AHI): > 0 and < 5 (group I), ≥ 5 and ≤ 15 (group II), > 15 and ≤ 30 (group III), and > 30 (group IV). We compared the OSA-free (AHI < 5) subjects with those with OSA (AHI ≥ 5). The evaluated clinical end-points were death and orthotropic heart transplant.

Results: The study population comprised 51 patients. Mean EF was 22%; 59% of patients were suffering from OSA. The increased severity of OSA correlated with worse pulmonary haemodynamics. Patients with OSA had higher mean pulmonary arterial pressure and pulmonary vascular resistance than individuals without OSA ($p = 0.044$, $p = 0.032$, respectively). The highest chamber diameters assessed in echocardiography were found in group IV ($p < 0.05$). A total of 10 end-points occurred during follow-up (8.9 ± 5.1 months), with significant differences observed between groups I–IV and the highest rate in group IV ($p < 0.001$).

Conclusions: The increasing severity of OSA worsens the prognosis of DCM patients, independently of severe HF and coexistent PH. Systematic OSA screening in patients with HF might facilitate identification of individuals at high risk of progression of pulmonary haemodynamic impairment and end-point rate.

Key words: dilated cardiomyopathy, heart failure, pulmonary hypertension, obstructive sleep apnoea

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INTRODUCTION

Despite advances in pharmacotherapy and newly introduced treatment with medical devices, mortality from heart failure (HF) is still an important clinical problem. Therefore, identification of treatable conditions that may contribute to its progression is of vital importance. Recent studies have revealed an association

Address for correspondence:
Beata Morawiec, MD, 2nd Department of Cardiology, Medical University of Silesia, ul. M. Curie-Skłodowskiej 10, 41–800 Zabrze, Poland, tel/fax: +48 32 271 10 10, e-mail: beamorawiec@wp.pl

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between untreated obstructive sleep apnoea (OSA) and incident or recurrent cardiovascular disease (CVD) events [1, 2].

It is hard to ascertain the independent role of OSA in HF. It has also been suggested that central sleep apnoea (CSA) may be associated with HF [3]. With the evidence supporting the positive effect of OSA treatment with continuous positive airway pressure on the parameters and outcomes in HF [4], there is increasing interest in the influence of OSA and its treatment on HF.

Most previous studies on OSA in HF focused on left ventricular (LV) dysfunction. In contrast, little is known on the influence of OSA on right ventricular (RV) function and pulmonary circulation. Patients suffering from HF secondary to dilated cardiomyopathy (DCM) present with clinical symptoms of LV and RV failure. Although a correlation between RV hypertrophy observed in pulmonary hypertension (PH) and the severity of OSA has been documented, its clinical role is still unclear [5]. The relationship between the severity of PH and mortality in patients with OSA was reported [6]. It is important to identify the clinical indicators of poor prognosis and treatable conditions that might contribute to the progression of HF and PH in the group of DCM patients with concomitant OSA.

The aim of this study was to evaluate the relationship between OSA of obstructive character and baseline, echocardiographic, and right heart catheterisation parameters in stable DCM patients, and to assess the influence of OSA on combined end-point in long-term follow-up.

METHODS

Patients

We enrolled patients hospitalised at 2nd Department of Cardiology in Zabrze, Medical University of Silesia, Katowice, Poland between February 2007 and August 2011. The inclusion criteria were: the diagnosis of DCM with symptoms of chronic HF in class I–III according to the classification of the New York Heart Association (NYHA), and LV ejection fraction (LVEF) < 30%, as assessed with initial echocardiography. The exclusion criteria included: acute decompensated HF and NYHA class IV, chronic obstructive pulmonary disease (COPD; in case of no history of COPD, spirometry was used to assess obstructive breathing disorders), other known chronic and/or intrinsic pulmonary disease, recent or previous history of cerebral stroke, known mental disease and treatment with continuous positive airway pressure, and bi-level positive airway pressure or oxygen.

Study protocol

Once patients were enrolled, they underwent physical examination, determination of the NYHA class, and basic laboratory tests, including N-terminal pro B-type natriuretic peptide (NT-proBNP) measurement. At the time of recruitment, information on risk factors or symptoms of sleep apnoea was collected. Somnolence during the day was evaluated with the Epworth sleepiness scale. Echocardiographic examination was performed at baseline and was followed by right heart catheterisation. Each patient performed the six-minute walk test (6MWT) and the Borg scale score was determined to assess the functional capacity.

The protocol of the study was approved by the local Bioethical Committee; all the patients gave their informed consent to participate in the study.

Echocardiographic examination

The transthoracic echocardiographic examination was performed in every patient by the same experienced echocardiography cardiologist, blinded to other results, using Vivid 7 (GE Medical Systems). The evaluated echocardiographic parameters included LV end-diastolic diameter (LVEDD), LV end-systolic diameter (LVESD) acquired from the parasternal long axis view using two-dimensional (2D) measurements, left atrial area, right atrial area, and RV diameter acquired from apical four-chamber view using 2D measurements, according to the European Guidelines [7]. LVEF was measured with LVEDD and LVESD, as assessed with the biplane Simpson method.

Right ventricular systolic pressure was estimated from the maximal systolic pressure gradient between the right ventricle and the right atrium, measured using peak regurgitant velocity of tricuspid regurgitation in the continuous-wave Doppler flow profile, obtained from the apical four-chamber view added to the right atrial pressure (RAP), estimated from the right atrial dilatation, the size of inferior vena cava, and its respiratory collapsibility. Acceleration time was measured with the pulsed-wave Doppler flow velocity profile in the RV outflow tract and was defined as the interval from the onset to the maximal velocity of the anterograde flow.

The images were stored in at least three cardiac cycles, and the final values represented the average of at least three measurements.

Right heart catheterisation

Each patient underwent right heart catheterisation using the right venous approach via the right internal jugular vein or the right iliac vein (Integris Allura 9C, Philips). We assessed the standard interventional parameters of "right heart": RAP, RV pressure, mean pulmonary arterial pressure (mPAP), and pulmonary capillary wedge pressure. Cardiac output was measured using the thermodilution technique. Pulmonary vascular resistance and total vascular resistance were calculated using commonly available formulas.

Sleep study

All patients underwent cardiorespiratory sleep study during a single night (ApneaLink, ResMed Corporation, San Diego, USA). The analysis of oronasal airflow, snoring, oxyhaemoglobin saturation, heart rate, and respiratory movements of chest and abdomen was performed. The analysed parameters

included apnoea index, hypopnoea index, apnoea–hypopnoea index (AHI), snoring, oxygen desaturation index, lowest saturation, and average saturation, and the automatic detection of central OSA. An apnoea was defined as a complete cessation of airflow through the airways, a hypopnoea was identified by a decrease in airflow of > 40%, both lasting ≥ 10 s with associated oxyhaemoglobin desaturation of at least 4% for hypopnoea [8]. Desaturation index was analysed automatically during sleep study with pulse oximetry and was defined by the number of desaturations (a drop of minimum 4% from baseline) per hour of recording. The average number of hypopnoeas and apnoeas per hour of sleep was defined as AHI. CSA was defined as cessation of airflow and respiratory effort. The automatic analysis of sleep study was confirmed by an experienced cardiologist specialised in sleep medicine.

OSA was defined by AHI = 5 or greater. Subjects were divided into four groups based on AHI: group I (free from OSA) — AHI > 0 and < 5, group II (mild OSA) — AHI ≥ 5 and ≤ 15, group III (moderate OSA) — AHI > 15 and ≤ 30, and group IV (severe OSA) — AHI > 30.

End-points

Patients were evaluated for 12 months for the occurrence of end-points: death of cardiac origin and orthotropic heart transplant.

Statistical analysis

All statistical tests were performed with Statistica 9 software (StatSoft Inc., Tulsa, OK, USA) and MedCalc (MedCalc Software, Mariakerke, Belgium). The values were tested for normality of distribution and were presented as means ± standard deviations (SD) or medians (25^{th} and 75^{th} percentiles). Comparisons between subjects with AHI < 5 and patients with AHI ≥ 5, as well as between groups I–IV, were performed with either t-student and ANOVA tests or Mann-Whitney U-test and Kruskal-Wallis test, when appropriate. The distributions of categorical values were compared with χ^2 test. Spearman correlation of provided data between groups was performed. Univariate logistic regression was used to assess the influence of sleep breathing parameters on PH. The level of < 0.05 was considered statistically significant throughout the study.

RESULTS

Population

Out of 360 patients admitted due to HF, 51 (14%) individuals met the inclusion criteria and were enrolled into the study. The main cause for exclusion was decompensated HF (64 patients, 18%), NYHA class IV (98 patients, 27%), oxygen supplementation prior to hospitalisation (31 patients, 9%), and pulmonary disease (COPD, pneumoconiosis, 75 patients, 21%). Forty-one (11%) patients declined to give informed consent. The characteristics of the study population are shown in Table 1. The mean age of the population was 53.3 ± 16.5 years, with male preponderance (80%). We observed equal proportions of NYHA classes II and III. The majority of patients presented with HF of non-ischaemic origin. The mean values of 6MWT distance and NT-proBNP corresponded to moderate or severe HF. All of the participants were on optimal treatment, which included beta-blockers (90.2%), angiotensin converting enzyme inhibitors (92.2%), angiotensin-1 receptor blockers (35.3%), aldosterone antagonists (90.2%), loop diuretics (76.5%), thiazides (13.7%), and digitalis (60.8%).

Table 1. Characteristics of the study population (n = 51)

Parameter	Value
Male	41 (80%)
Age [years]	53.3 ± 16.5
Smokers: Current	3 (6%)
Smokers: Previous	15 (29%)
NYHA class II	27 (53%)
NYHA class III	24 (47%)
Non-ischaemic aetiology	39 (76%)
BMI [kg/m^2]	26.59 ± 4.48
BSA [m^2]	1.94 ± 0.21
6MWT [m]	477.9 ± 113.8
NT-proBNP [pg/mL]	1905.6 ± 2009.2
Ejection fraction [%]	22.2 ± 8.2
Epworth scale	2.9 ± 2.5
Mean PAP [mm Hg]	30.1 ± 10.9

Values are presented as n (%) or means ± standard deviations; 6MWT — six-minute walk test; BMI — body mass index; BSA — body surface area; PAP — pulmonary artery pressure; NT-proBNP — N-terminal pro-B-type natriuretic peptide; NYHA — New York Heart Association

Sleep study

OSA was identified in 59% of patients. None of the patients had pure CSA. Based on the severity of OSA, the subjects were stratified into the following groups: group I — 41% (n = 21), group II — 31% (n = 16), group III — 16% (n = 8), and group IV — 12% (n = 6).

An assessment of the relationship between sleep variables and echocardiographic, haemodynamic, and laboratory parameters, and 6MWT was performed. Table 2 presents statistically significant correlations documented on this analysis. None of the sleep parameters showed significant association with mPAP > 25 mm Hg on univariate logistic regression. Median Epworth sleepiness scale was equal in groups with AHI < 5 and AHI ≥ 5 (2 [1.3; 3.8] vs. 3 [1; 5], p = 0.36).

Comparison between groups

Echocardiography revealed marked impairment of LVEF (mean of 22%) in the whole cohort. Other echocardiographic parameters are presented in Table 3; no significant differences were documented between groups of AHI < 5 and ≥ 5.

Regarding right heart catheterisation outcomes, patients with OSA (AHI ≥ 5) were characterised by mild elevation in mPAP, pulmonary vascular resistance, and RAP (Table 4).

Comparison of groups I–IV revealed significant differences in haemodynamic parameters of group I and group II, with higher pulmonary vascular resistance, transpulmonary gradient, total vascular resistance, and RAP, and lower cardiac output in group II ($p < 0.05$). Right and left ventricular diameters (RV, LVEDD, LVESD) in group IV were higher as compared to groups I–III ($p < 0.05$).

Table 2. Coefficients of correlation (R) between sleep variables and clinical and laboratory parameters in patients with (OSA-positive) and without (OSA-negative) obstructive sleep apnoea syndrome

Parameters	R	P
OSA-negative		
DI and AcT	–0.478	0.001
LSAT and PASP	–0.308	0.038
LSAT and PVR	–0.297	0.050
LSAT and TPG	–0.332	0.031
OSA-positive		
AHI and mPAP	0.287	0.050
AHI and RAP	0.358	0.012
DI and TPG	0.306	0.050
DI and PCWP	0.332	0.018
DI and RAP	0.394	0.004
DI and mPAP	0.364	0.011
LSAT and AcT	0.35	0.019
LSAT and EF	0.37	0.019

AcT — acceleration time; AHI — apnoea–hypopnoea index; DI — desaturation index; EF — ejection fraction; LSAT — lowest saturation; mPAP — mean pulmonary arterial pressure; PASP — pulmonary artery systolic pressure; PCWP — pulmonary capillary wedge pressure; PVR — pulmonary vessel resistance; RAP — right atrial pressure; TPG — transpulmonary gradient

The analysed groups did not differ in terms of 6MWT distance and NT-proBNP level.

A total of 10 end-points were documented during 8.9 ± 5.1 months of follow-up (Fig. 1). Four patients underwent orthotropic heart transplant (one in group III and three in group IV), and six individuals died (two in group I, two in group III, and two in group IV; $p < 0.0001$ between groups).

DISCUSSION

Obstructive sleep apnoea syndrome (OSAS) can increase the probability of HF [9], and may be associated with worse outcome in the HF population [10]. Patients with OSAS and HF are characterised by severe systolic dysfunction of the LV in the majority of published studies [11–13]. The poor echo-

Table 3. Echocardiographic characteristics of patients with (AHI ≥ 5) and without (AHI < 5) obstructive sleep apnoea syndrome; p = NS

Parameter	AHI < 5	AHI ≥ 5
EF [%]	21.53 ± 6.93	22.57 ± 7.43
EDD [mm]	70.1 ± 8.1	71.7 ± 7.6
ESD [mm]	63.18 ± 7.96	64.88 ± 5.51
EDV [mL]	237 ± 73.6	227 ± 72.5
ESV [mL]	191 ± 64.1	187 ± 51.1
LA [mm]	45.1 ± 5.9	46.7 ± 5.8
RV [mm]	36.9 ± 9.0	30.9 ± 7.6
LAA [cm^2]	27.7 ± 8.5	31.3 ± 9.8
RAA [cm^2]	21.5 ± 9.1	23.6 ± 7.8
RVSP [mm Hg]	38.7 ± 13.5	42.7 ± 16.4
AcT [ms]	98.1 ± 28.9	88.42 ± 21.3

Values are presented as means ± standard deviations; AcT — acceleration time; EDD — end diastolic diameter; EDV — end diastolic volume; EF — ejection fraction; ESD — end systolic diameter; ESV — end systolic volume; LA — left atrial diameter; LAA — left atrial area; RAA — right atrial area; RV — right ventricular diameter; RVSP — right ventricular systolic pressure; NS — not significant

Table 4. Right heart haemodynamic characteristics of patients with (AHI ≥ 5) and without (AHI < 5) obstructive sleep apnoea syndrome

Parameter	AHI < 5	AHI ≥ 5	P
Mean PAP [mm Hg]	26.03 ± 10.21	32.49 ± 10.99	0.044
HR [bpm]	76.94 ± 12.62	92.5 ± 12.38	NS
PCWP [mm Hg]	18.5 ± 10.69	21.18 ± 8.6	NS
PVR [Wood]	1.78 ± 0.90	3.29 ± 3.12	0.032
TPG [mm Hg]	7.53 ± 3.24	11.38 ± 7.60	0.020
TPR [Wood]	6.23 ± 3.11	9.10 ± 5.17	NS
RAP [mm Hg]	6.25 ± 6.03	10.26 ± 5.51	0.008
CO [l/min]	4.61 ± 1.42	4.11 ± 1.27	NS

Values are presented as means ± standard deviations; CO — cardiac output; HR — heart rate; PAP — pulmonary arterial pressure; PCWP — pulmonary capillary wedge pressure; PVR — pulmonary vessel resistance; RAP — right atrial pressure; TPG — transpulmonary gradient; TPR — total pulmonary resistance

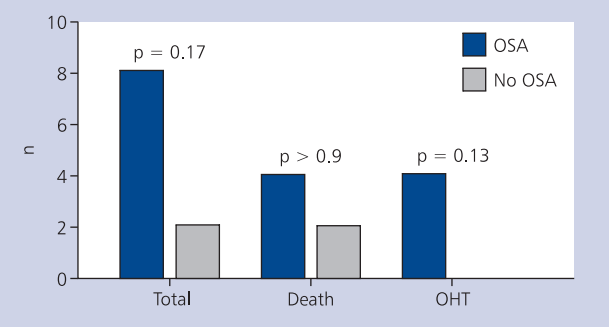

Figure 1. End-points in patients with (AHI ≥ 5) and without (AHI < 5) obstructive sleep apnoea (OSA); AHI — apnoea–hypopnoea index; OHT — orthotropic heart transplant

cardiographic measures documented in our study resulted from strict enrolment criteria (LVEF < 30%). However, none of the echocardiographic parameters were associated with the coexistence of OSA. The progress of chamber dilatation was observed with rising severity of OSA, with the highest diameters in the most severe class. This directly indicates that the severity of anatomical damage of heart chambers did not impact the development of OSA, but it could be related to (or dependent on) OSA severity despite the lack of significant differences in the levels of NT-proBNP and the outcomes of 6MWT between groups varying in terms of OSA severity. High incidence of OSAS and lack of its correlation with other risk factors for HF was also documented in other studies [14]. The majority of patients with OSAS manifest diastolic dysfunction or subclinical echocardiographic symptoms of systolic dysfunction of the LV [15]. In our study, the systolic function (LVEF) was equal in every group. The 2D diameters of the ventricles were significantly higher in group IV when compared with every other group (I–III). This suggests that OSA and its severity might accelerate the pre-existing RV and LV dilatation, thus leading to the progression of HF.

Although PH in OSAS is generally of mild to moderate degree [16], it can cause functional limitations and increase the mortality [6]. However, opposite findings, with no correlation of OSAS with haemodynamic impairment, were reported by some authors [11]. All of our patients presented with mild PH, as indicated by mPAP values documented on right heart catheterisation. The haemodynamic profile of patients was aggravated by the coexistence of OSA, as reflected by the deterioration of pulmonary haemodynamics with the worsening of sleep breathing parameters. Significantly worse parameters (pulmonary vascular resistance, transpulmonary gradient, total vascular resistance, RAP, and cardiac output) were documented in the group with mild OSA (group II), when compared with the group free from OSA (group I). A marked and sustained tendency towards worsening of the parameters with increasing severity of OSA was found in the comparison between groups with more severe OSA (group III and IV) and group I. Therefore, we hypothesised that OSA may predispose to the induction or progression of PH and right-heart failure, thus influencing the treatment strategy. In the present study, high transpulmonary gradient correlated with increasing severity of OSA. Such a condition was previously reported as a firm limitation for heart transplant [17].

It the context of evident association of sleep disturbances with pulmonary haemodynamic parameters, it should be emphasised that the more prevalent sleep disorder breathing in PH is CSA [18]. It is also known that more severely reduced LVEF and higher NYHA classes are associated with an increased risk of developing Cheyne-Stokes respiration, one of the types of CSA [19]. In this context a higher proportion of obstructive over CSA observed in our study can be surprising but is in line with several previous reports [11, 14, 18]. When compared with earlier reports, patients in our study received similar medical treatment, which included widespread use of beta-blockers [14]. The influence of beta-blockers on reduction of CSA and its severity has already been described [20] and explains no case of CSA in the study group.

To date, OSAS is considered an independent risk factor for all-cause mortality and fatal and non-fatal cardiovascular events in HF patients [10]. In spite of the high incidence of OSAS in HF, the majority of OSAS cases remain undiagnosed and, therefore, untreated [21]. Occulted and/or untreated OSAS has an adverse impact on the prognosis in HF patients [10]. Our patients did not manifest clinical symptoms of OSAS (body mass index, neck circumference, snoring, sleepiness). Mean Epworth sleepiness scale was low and did not correspond to daytime sleepiness, reported previously by other authors [14, 22]. Furthermore, it suggests that the sleep study can be beneficial, especially in HF patients with low LVEF. Moreover, the monitoring of breathing disturbances during sleep was previously shown to contribute to proper differential diagnosis of OSAS and habitual snorers [23].

Only a few studies have examined the potential influence of OSA on mortality and/or heart transplant [10]. The influence of OSAS on end-points in long-term follow-up was not documented in these studies. Similarly to previous studies [24], mortality and transplantation rates observed in our patients were high and did not differ between individuals with OSA and those without this condition. The highest rate of orthotropic heart transplant in group IV reflects the clinical status of those patients. Therefore, we conclude that the increasing severity of OSA worsens the prognosis and the clinical outcome of DCM, independently of echocardiographic evidence of severe (albeit stable) HF. Furthermore, the coexistence of mild or moderate PH should not constitute a rigid contraindication for orthotropic heart transplant.

Limitations of the study

Several limitations of the present study should be underlined. The small number of enrolled patients raises scepticism towards the statistical significance of the provided data. OSA

were evaluated with polygraphy, not polysomnography, which represents the standard of sleep disorder diagnosis. Nevertheless, both methods are complementary and are alternative diagnostic tools for OSA also in HF patients, thus reducing costs and increasing availability of OSA screening by replacing polysomnography with polygraphy. [25]. The technology involved did not allow reliable identification of CSA, identifying Cheyne-Stokes breathing and interpreting it as CSA.

CONCLUSIONS

In summary, the coexistence of OSA affects pulmonary haemodynamics in patients with DCM, and is related to impaired cardiac function and higher end-point rate in this group, with deterioration of those parameters with the increase in OSA severity. Systematic screening for OSA should be considered in hospitalised patients with HF. Early identification and repetitive evaluation of patients with DCM and OSA might reduce the progression of pulmonary haemodynamic impairment and end-point rate.

Conflict of interest: *none declared*

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Wpływ bezdechu sennego na parametry echokardiograficzne i parametry hemodynamiki płucnej u pacjentów z kardiomiopatią rozstrzeniową

Damian Kawecki[1], Celina Wojciechowska[1], Wojciech Jacheć[1], Krystyna Krzemień-Wolska[1], Janusz Dola[1], Wojciech Ścierski[2], Izabela Kawecka[2], Andrzej Tomasik[1], Tomasz Brzostowicz[3], Beata Morawiec[1], Ewa Nowalany-Kozielska[1]

[1]II Klinika Kardiologii w Zabrzu, Śląski Uniwersytet Medyczny, Katowice
[2]Klinika Otorynolaryngologii w Zabrzu, Śląski Uniwersytet Medyczny, Katowice
[3]Klinika Kardiologii, Centrum Medyczne, Opole

Streszczenie

Wstęp: W przypadku pacjentów ze współistniejącą kardiomiopatią rozstrzeniową i zespołem bezdechu sennego (ZBS), kluczowego znaczenia nabiera identyfikacja niekorzystnych czynników rokowniczych oraz dolegliwości, które mogą się przyczyniać do progresji niewydolności serca i nadciśnienia płucnego.

Cel: Celem pracy była analiza wpływu ZBS na parametry echokardiograficzne i parametry hemodynamiki płucnej u pacjentów z kardiomiopatią rozstrzeniową oraz częstość występowania punktów końcowych w okresie objętym obserwacją.

Metody: Do badania włączano pacjentów z kardiomiopatią rozstrzeniową i znacznie obniżoną frakcją wyrzutową (EF < 30%). U wszystkich chorych wykonano badanie poligraficzne i echokardiograficzne oraz przeprowadzano cewnikowanie prawego serca. Na podstawie wartości wskaźnika bezdechów i spłyconych oddechów (AHI), uczestników badania podzielono na grupy: 0–4 (grupa I), 5–15 (grupa II), 16–30 (grupa III), > 30 (grupa IV). Następnie porównywano częstość występowania klinicznych punktów końcowych (zgonu i ortotropowego przeszczepienia serca) w grupie pacjentów bez ZBS (AHI < 5) i u osób, u których rozpoznano to zaburzenie (AHI ≥ 5).

Wyniki: Ogółem do badania włączono 51 pacjentów. Średnia wartość EF wyniosła 22%; ZBS rozpoznano u 59% chorych. Większe nasilenie ZBS wiązało się z gorszymi wartościami parametrów hemodynamiki płucnej. U chorych z ZBS stwierdzono wyższe średnie wartości ciśnienia w tętnicy płucnej oraz płucnego oporu naczyniowego niż u osób bez ZBS (odpowiednio $p = 0{,}044$ i $p = 0{,}032$). Największą średnicę jam serca odnotowano w badaniu echokardiograficznym u pacjentów z grupy IV ($p < 0{,}05$). W okresie obserwacji (8,9 ± 5,1 miesięcy) odnotowano łącznie 10 punktów końcowych; częstość ich występowania różniła się istotnie między porównywanymi grupami, osiągając najwyższy poziom u chorych z grupy IV ($p < 0{,}001$).

Wnioski: Większe nasilenie ZBS jest niekorzystnym czynnikiem rokowniczym u pacjentów z kardiomiopatią rozstrzeniową, niezależnym od nasilenia niewydolności serca i współwystępującego nadciśnienia płucnego. Systematyczne badanie pacjentów z niewydolnością serca w kierunku występowania ZBS może pozwolić na identyfikację osób zagrożonych pogorszeniem parametrów hemodynamiki płucnej i wystąpieniem punktów końcowych.

Słowa kluczowe: kardiomiopatia rozstrzeniowa, niewydolność serca, nadciśnienie płucne, zespół bezdechu sennego

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Adres do korespondencji:
lek. Beata Morawiec, II Klinika Kardiologii w Zabrzu, Śląski Uniwersytet Medyczny, ul. M. Curie-Skłodowskiej 10, 41–800 Zabrze, tel/faks: +48 32 271 10 10, e-mail: beamorawiec@wp.pl
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