

Assessment of systemic right ventricular function in adult overweight and obese patients with congenitally corrected transposition of the great arteries

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Abstract

Background: In congenitally corrected transposition of the great arteries the right ventricle (RV) supports systemic circulation, and patients are prone to develop heart failure over time. Chronic volume overload secondary to obesity may contribute to premature dysfunction of the systemic RV.

Aim: The aim of our study was to assess the systemic RV function in overweight/obese adult patients with congenitally corrected transposition of the great arteries.

Methods: Transthoracic echocardiographic studies and laboratory testing (N-terminal pro-B-type natriuretic peptide [NT-proBNP] assessment) were performed in patients with congenitally corrected transposition, who were scheduled for a routine examination, and the body mass index was calculated for each patient.

Results: We studied 56 adults (31 men; mean age 33.9 years); 22 of whom were overweight (body mass index [BMI] of 25–29.9 kg/m²) or obese (BMI of 30 kg/m² or more), and 34 of whom were normal weight (BMI below 25 kg/m²). Age, gender, heart rate, and blood pressure were similar in both groups. The mean NT-proBNP levels were not significantly different. On echocardiography, the overweight/obese patients had a decreased systemic RV fractional area change (0.38) compared to normal weight patients (0.43); $p = 0.02$. Moreover, a significant reduction in the global longitudinal strain in the overweight/obese group was observed (–15.3% vs. –18.3%; $p = 0.01$).

Conclusions: Overweight/obesity in adult patients with congenitally corrected transposition of the great arteries is associated with impaired systemic RV function.

Key words: congenitally corrected transposition of the great arteries, systemic right ventricle, obesity, echocardiography

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INTRODUCTION

Congenitally corrected transposition of the great arteries is characterised by atrioventricular and ventriculo-arterial discordant connections, resulting in right ventricle (RV) functioning in the subaortic position [1]. Because the haemodynamic pathways are unchanged, patients with congenitally corrected transposition of the great arteries without concomitant lesions do not require surgery, and the diagnosis may be made early in adulthood. Long-term survival is possible [2, 3]; however, the permanent exposure to the systemic pressure, the tricuspid (systemic) atrioventricular valve regurgitation and ischaemia due to inadequate coronary supply or dyssynchrony are factors, that can lead to systemic RV dysfunction and heart

failure in long-term follow-up [4]. The increasing worldwide prevalence of obesity also affects the adult congenital heart disease population [5]. Obesity results in haemodynamic changes that lead to increases in the cardiac output and systemic ventricle enlargement. Studies in patients with structurally normal hearts have shown that obesity is associated with left ventricular (LV) hypertrophy, a potential cause of both systolic and diastolic dysfunction. In extreme cases obesity cardiomyopathy is diagnosed [6]. Apart from the direct negative impact of obesity on cardiac function, the most common comorbidities (i.e. coronary heart disease, diabetes, hypertension, and sleep apnoea syndrome) also play a role. Little is known about the influence of obesity on

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cardiac performance in adult congenital heart disease [7, 8], and data on patients with systemic RV are lacking. Chronic volume overload secondary to overweight/obesity, together with the chronic pressure overload related to systemic circulation, may lead to premature dysfunction of the systemic RV. The aim of our study was to assess the systemic RV function in overweight and obese adult patients with congenitally corrected transposition of the great arteries.

METHODS

Study population

This was a cross-sectional study. We screened consecutive adult patients with congenitally corrected transposition of the great arteries, who were referred to our outpatient clinic for a routine check-up between January 2012 and December 2014. We included patients with double (atrioventricular and ventriculo-arterial) discordance and biventricular circulation. Individuals who underwent prior cardiac surgery due to atrial/ventricular septal defect or subpulmonary outflow tract repair were included unless the operation was performed within the last 12 months prior to the study. The exclusion criteria from the study were as follows: (1) systemic hypertension, (2) severe (NYHA III/IV class) heart failure, (3) systemic atrioventricular valve replacement, (4) arrhythmia at the echocardiographic study, and (5) single ventricle palliation.

The study was approved by the institutional medical Ethics Committee, and each patient signed informed, written consent.

Clinical examination and laboratory testing

Each patient underwent routine physical examination, blood sampling, and transthoracic echocardiography on the same day. We calculated the body mass index (BMI, kg/m²) for each patient. Overweight was defined as a BMI of 25–29.9 kg/m² and obesity as a BMI of 30 kg/m² or more. Obesity was further broken down into: class I obesity (BMI 30–34.9 kg/m²), class II obesity (BMI 35–39.9 kg/m²), and class III obesity (BMI ≥ 40 kg/m²) [9]. Blood pressure measurements were obtained with a patient in the sitting position after a 5-min rest using a cuff sphygmomanometer (Omron M1, Omron Healthcare Co., Kyoto, Japan). Blood was collected into tubes containing EDTA, and the plasma concentrations of N-terminal pro-B-type natriuretic peptide (NT-proBNP) were measured using an immunoradiometric assay (Cobas e 601 analyser, Roche Diagnostics, Germany).

Echocardiographic study

Transthoracic echocardiographic examinations were performed using commercially available equipment (Vivid 9, GE Vingmed Ultrasound, Horten, Norway) with a matrix probe M5S. The systemic RV and subpulmonary LV end-diastolic diameters were measured in the apical four-chamber view at the level of basal segments. The systemic RV end-diastolic and end-systolic areas were measured to calculate the systemic

RV fractional area change from the same apical four-chamber view [10]. The tricuspid annular plane systolic excursion was acquired by the conventional M-mode method from the lateral point of the tricuspid valve. Pulsed tissue Doppler with a sample volume placed at the lateral corner of the tricuspid annulus was used to assess the systolic (s') and diastolic (e' and a') velocities. The regional myocardial function (longitudinal strain) was determined using the speckle-tracking method. Grey-scale images (mean frame rate 52/s) were analysed offline using dedicated software (Echopac version 113, GE). The programme allowed for automatic tracking of the region of interest throughout the systemic RV free wall and the intraventricular septum segments. The longitudinal systemic RV strain was defined as the mean of the strains of six wall segments in the apical four-chamber view (Fig. 1). The severity of tricuspid regurgitation was assessed according to the American Society of Echocardiography recommendations and quantified as none, mild, moderate, and severe [11]. The proximal isovelocity surface area method was used to calculate the effective regurgitant orifice and regurgitant volume [11]. Measurements of the left atrial (LA) area were performed at the end-systole of the systemic ventricle in the apical four-chamber view. From the same view, the transtricuspid (systemic atrioventricular valve) flow Doppler was recorded using the pulsed wave Doppler technique with the sample located at the tips of the valve leaflets. The early (E wave) and late (A wave) tricuspid inflow velocity, E/A ratio, and deceleration time were calculated.

Statistical analysis

Data are presented as the mean values ± standard deviation for continuous variables and as the number (percentage) for categorical variables. The normal distribution of variables was checked using the Kolmogorow-Smirnov test. The comparisons of the mean differences between the groups were made using unpaired Student's t test or nonparametric methods according to the data distribution. The linear Pearson correlation was used to indicate the strength of a relationship between the BMI and the other parameters that were considered. The intra-observer reproducibility for the systemic RV longitudinal strain in 20 randomly chosen patients was evaluated by means of the intra-class correlation coefficient and absolute difference. The strength of agreement between two measurements was assessed using linear regression analysis. A p < 0.05 confidence level was considered statistically significant. The statistical software package Statistica 10 (StatSoft Inc., USA) and software R-3.2.2 for Windows were used for data analyses.

RESULTS

Clinical and biomarker data

The study group consisted of 56 adults with congenitally corrected transposition of the great arteries (31 men/25 women; mean age 33.9 ± 12.9 years), 34 of whom were normal weight, 20 of whom were overweight, and two of whom

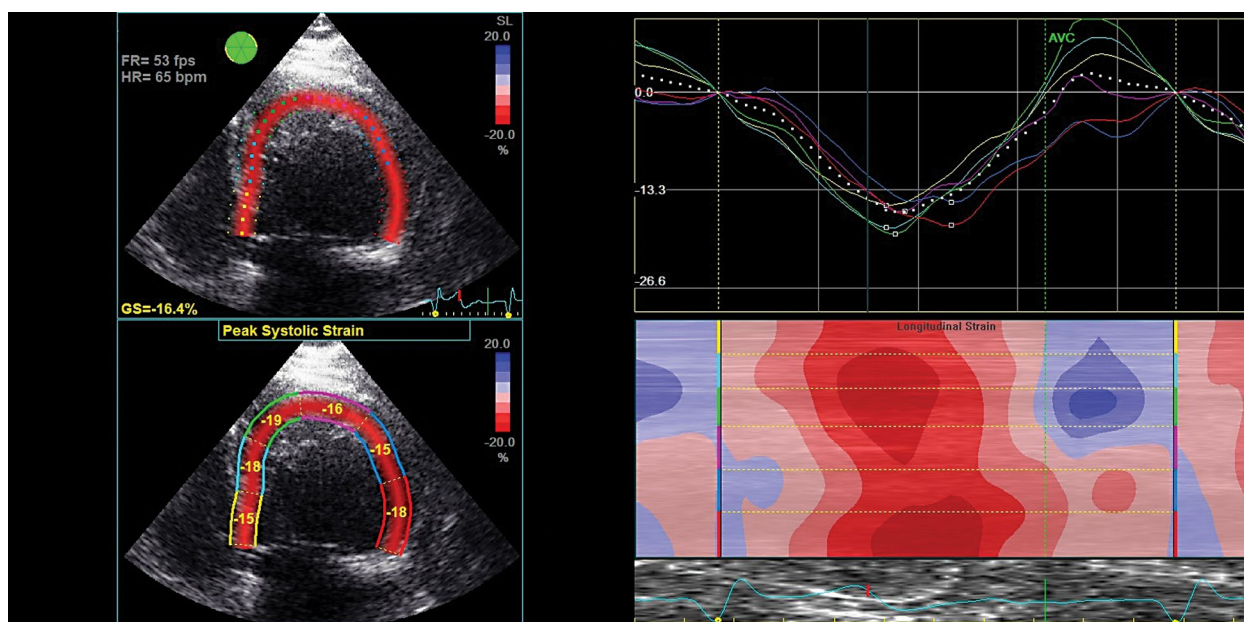


Figure 1. An example of two-dimensional longitudinal strain analysis in an overweight patient with congenitally corrected transposition of the great arteries

were obese. As there were only two individuals with a BMI > 30 kg/m² (30.6 kg/m² and 30.1 kg/m²), we analysed the overweight and obese subjects together. No significant differences were observed between the normal weight and overweight/obese patients with respect to age, gender, heart rate, concomitant heart lesions (including tricuspid regurgitation), pacemaker implantation or pharmacological treatment. The systolic and diastolic blood pressures tended to be higher in the overweight/obese group than in the normal weight group; however, these differences did not reach statistical significance. The NT-proBNP levels were considerably elevated (normal range 0–125 pg/mL) in overweight/obese patients as well as in normal weight individuals, and the mean values were comparable across both groups. No significant relationship between the NT-proBNP level and BMI was observed. The clinical characteristics of the study group are given in Table 1.

Echocardiographic data

Systolic RV function. The overweight/obese patients showed decreased fractional area change compared to that of the normal weight patients. The average systemic RV diameter was larger in the overweight/obese group than in the normal weight group, although the difference was not significant. The tricuspid annular plane systolic excursion and RV *s'* values were low and similar in both groups. Regarding the regional myocardial function, a significant reduction in the systemic RV longitudinal strain in the overweight/obese group was observed. The severity of tricuspid regurgitation was comparable in the normal weight and overweight/obese patients (Table 2).

Among the echocardiographic parameters characterising systolic function of the systemic RV, BMI was significantly correlated with systemic RV longitudinal strain ($r = 0.35$, $p = 0.009$) (Fig. 2).

Diastolic RV function. The indices of the systemic RV diastolic dysfunction are presented in Table 3. We observed lower E velocity values and a lower *e'/a'* velocity ratio in the overweight/obese patients than in the normal weight patients. The LA area was higher in the overweight/obese group than in the normal weight group. The remaining parameters were similar in both groups.

Among the parameters describing the systemic RV diastolic function, we found an inverse correlation between BMI and E velocity ($r = -0.35$, $p = 0.01$) and between BMI and *e'/a'* ratio ($r = -0.42$, $p = 0.003$) (Fig. 3). We also identified a positive correlation between BMI and LA area ($r = 0.41$, $p = 0.003$) (Fig. 4).

Feasibility and reproducibility. Echocardiograms obtained in all patients were of sufficient quality, and no systemic RV segment was excluded from the analysis of myocardial deformation. The intra-observer intra-class correlation coefficient was 0.95 (95% CI 0.89–0.98) for systemic RV longitudinal strain, and the absolute difference between the first and the second measurement was $0.97 \pm 0.79\%$. There was a very good linear agreement within intra-observer systemic RV longitudinal strain measurements ($r = 0.95$, $p < 0.001$).

DISCUSSION

Our study is the first one to suggest that overweight/obesity negatively affects the regional and global systemic RV function

Table 1. Clinical characteristics of the study population

	Normal weight patients (n = 34)	Overweight/obese patients (n = 22)	p
Mean age [years]	32.1 ± 12.8	36.7 ± 12.7	0.1
Men	16 (47%)	15 (68%)	0.12
Body mass index	22.1 ± 2.3	27.5 ± 1.6	< 0.00001
Heart rate [bpm]	70.8 ± 11.1	70.1 ± 10.7	0.94
Systolic BP [mm Hg]	117.3 ± 11.2	121.3 ± 10.3	0.26
Diastolic BP [mm Hg]	75.5 ± 6.0	79.2 ± 6.4	0.08
Moderate/severe TR	16 (47%)	6 (27%)	0.14
Significant PS	8 (23.5%)	2 (9%)	0.17
Pacemaker implanted	12 (35%)	7 (32%)	0.79
Prior surgery	11 (32%)	6 (27%)	0.69
NYHA class I/II	22/12 (65/35)	15/7 (68/32)	0.79
NT-proBNP [pg/mL]	441.2 ± 516.3	589 ± 614.4	0.28
Medications:			
ACEI/ARB	13 (38%)	8 (36%)	0.92
Beta-blocker	13 (38%)	6 (27%)	0.37
Diuretics	5 (15%)	7 (32%)	0.12

ACEI — angiotensin converting enzyme inhibitor; ARB — angiotensin receptor blocker; BP — blood pressure; PS — pulmonary stenosis; TR — tricuspid regurgitation; NT-proBNP — N-terminal pro-B-type natriuretic peptide; NYHA — New York Heart Association

Table 2. The echocardiographic assessment of the systolic systemic right ventricular function and tricuspid regurgitation severity

	Normal weight patients (n = 34)	Overweight/obese patients (n = 22)	p
sRV diameter [cm]	5.23 ± 0.7	5.61 ± 0.82	0.27
LV diameter [cm]	3.55 ± 1.31	3.96 ± 0.92	0.37
sRV diastolic area [cm ²]	25.4 ± 6.5	29.6 ± 8.2	0.07
sRV systolic area [cm ²]	14.4 ± 3.8	18.2 ± 5.6	0.01
Fractional area change	0.43 ± 0.06	0.38 ± 0.08	0.02
TAPSE [mm]	15.1 ± 4.1	15.5 ± 3.9	0.73
sRV s' [cm/s]	9.6 ± 3.2	8.8 ± 3.4	0.21
sRV GLS [%]	-18.3 ± 3.6	-15.3 ± 3.4	0.01
TR EROA [cm ²]	0.20 ± 0.15	0.22 ± 0.18	0.51
TR Vol [mL]	35.5 ± 25.9	39.0 ± 24.7	0.96

GLS — global longitudinal strain; LV — left (subpulmonary) ventricle; sRV — systemic right ventricle; TAPSE — tricuspid annular plane systolic excursion; TR EROA — tricuspid effective regurgitant orifice area; TR Vol — tricuspid regurgitant volume

Table 3. The echocardiographic assessment of the diastolic systemic right ventricular function

	Normal weight patients (n = 34)	Overweight/obese patients (n = 22)	P
TV E [m/s]	0.87 ± 0.21	0.74 ± 0.28	0.01
TV A [m/s]	0.54 ± 0.21	0.60 ± 0.22	0.53
E/A ratio	1.88 ± 0.99	1.5 ± 1.06	0.1
DT [ms]	205.8 ± 71.5	208.9 ± 60.4	0.87
sRV e' [cm/s]	14.5 ± 4.1	12.4 ± 4.2	0.09
sRV a' [cm/s]	6.6 ± 2.5	8.2 ± 2.5	0.049
sRV e'/a' ratio	2.54 ± 1.18	1.67 ± 0.84	0.003
LA area [cm ²]	24.5 ± 8.5	32.2 ± 11.6	0.01

DT — deceleration time; LA — left atrial; sRV — systemic right ventricle; TV — tricuspid valve

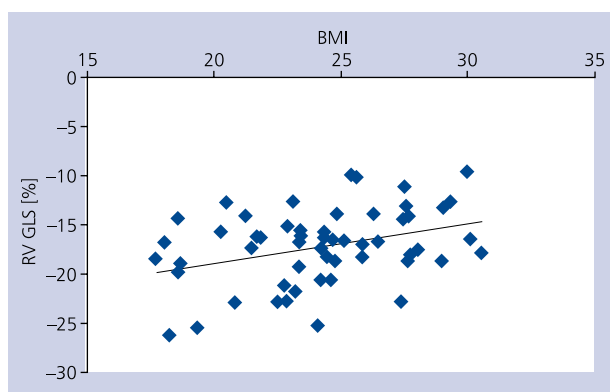


Figure 2. The correlation between body mass index (BMI) and systemic right ventricular global longitudinal strain (RV GLS) ($r = 0.35$; $p = 0.009$)

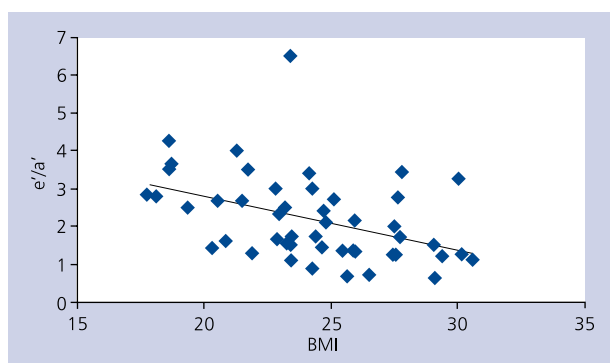


Figure 3. The correlation between body mass index (BMI) and e'/a' ratio ($r = -0.42$; $p = 0.003$)

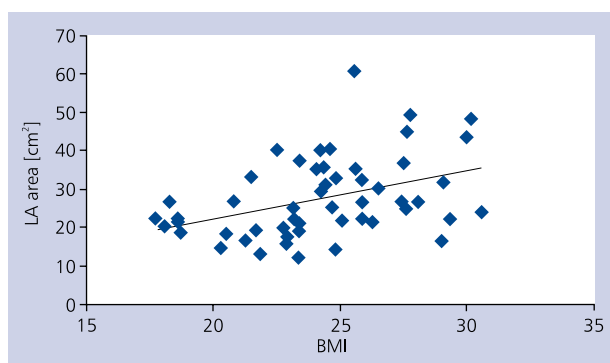


Figure 4. The correlation between body mass index (BMI) and left atrial (LA) area ($r = 0.41$; $p = 0.003$)

in the relatively “well” population of adults with congenitally corrected transposition of the great arteries. The incidence of overweight in our study group was similar to that reported

by Zomer et al. [12] in a large cohort of Dutch patients with simple congenital heart disease, i.e. isolated atrial or ventricular septal defect, patent ductus arteriosus, mild pulmonary stenosis, or isolated congenital aortic or mitral valve disease (35.7% vs. 34.3%), although the percentage of obese individuals was lower in the present study than in the one mentioned above (3.6% vs. 11.4%). We demonstrated that overweight and class I obesity are associated with impairment of the systemic RV function assessed with echocardiography. In contrast, the study by Pascual et al. [13] revealed that in patients with structurally normal hearts, the systolic function of the LV increases in the early stages of obesity. This difference might be due to the limits in the adaptation of morphological RV to the systemic load, which appears to be reached in normal weight subjects. Therefore, it seems that the increase in body weight might result in systemic ventricle dysfunction.

Among the standard echocardiographic indices of the systolic RV function, only fractional area change differed between the subgroups, and the nonparametric indices of the systolic RV function were similar in the overweight/obese and normal weight patients. However, these parameters are usually applied to evaluate the subpulmonary RV, and some investigators suggested that they are not suitable for systemic RV assessment [14, 15].

We also demonstrated that the systemic RV longitudinal deformation is significantly impaired in overweight/obese patients. This finding is of clinical importance, considering the papers by Kalogeropoulos et al. [16] and Diller et al. [17], which reported that reduced global longitudinal strain is associated with a higher risk of adverse clinical events (progression to NYHA class ≥ 3 , clinically relevant cardiac arrhythmia, or death) in patients with subaortic RV. In the present study, BMI correlated weakly with the global longitudinal strain values. The results are consistent with those of Wong et al. [18], who observed a correlation with a similar degree between BMI and LV average peak strain in more than 100 overweight or obese subjects.

Most previous studies showed that diastolic filling of the LV was impaired in obese relative to that of normal weight individuals with structurally normal hearts [13, 18–20]. Similarly, in our study, passive tricuspid filling velocity was reduced in the overweight/obese group compared to the normal weight group. In contrast to previous data on obese patients, the tissue Doppler imaging-derived RV e' velocity was similar in both analysed groups. However, we observed that the e'/a' ratio was significantly decreased in the overweight/obese patients, and it correlated moderately with higher body weight. Therefore, overweight/obesity seems to impair the systemic RV function, but little is known about the diastolic function of the systemic RV thus far [21]. These findings should be interpreted with caution and they require further investigation.

Our findings on LA enlargement in overweight/obese patients are consistent with those reported previously, showing that BMI is an independent predictor of LA size [22, 23]. The

link between obesity and LA size is thought to be from the volume overload secondary to excess adipose tissue.

The clinical utility of B-type natriuretic peptide and NT-proBNP in patients with systemic RV has been investigated in several studies; however, the results for patients with congenitally corrected transposition of the great arteries remain inconsistent [24, 25]. In our study, the NT-proBNP levels were considerably elevated, but they did not differ significantly between subgroups; however, overweight/obese patients were more likely to present with systemic ventricular dysfunction. The possible explanation might be a phenomenon described in the Framingham Study population, which reported that increased BMI is associated with lower plasma natriuretic peptide levels [26]. We speculated that the increase in NT-proBNP in overweight/obese patients might be lower than that in normal weight subjects.

In summary, our study is the first, to the best of our knowledge, to show that overweight/obesity might be a modifiable risk factor associated with the subclinical dysfunction of systemic RV. These findings emphasise the importance of aggressive prevention of obesity in adult patients with congenital heart disease. Our results are consistent with the recently published American Heart Association scientific statement on Congenital Heart Disease in the Older Adult, which stressed the role of ideal body weight attainment in this population [27].

Limitations of the study

This study focused on adult patients with congenitally corrected transposition of the great arteries and might not be applicable to other patients with systemic RV (e.g. patients with d-transposition of the great arteries after atrial switch). Another limitation of the study is the use of the BMI as the only measure of obesity because we did not measure other anthropometric parameters (i.e. waist circumference). The duration of obesity may also affect the impact of BMI on cardiac performance. However, the duration of obesity is difficult to retrospectively establish. Echocardiographic evaluation of the RV remains difficult because of complex geometry, and it does not allow us to reliably calculate its volumes, mass, and ejection fraction. Although magnetic resonance imaging overcomes the shortcomings of echocardiography in the assessment of the RV, this method was not used due to contraindications for magnetic resonance imaging, which were present in the large proportion of the study population (implanted pacemaker). The main limitations of the speckle-tracking analysis are the assessment limited to the longitudinal myocardial deformation derived only from the apical four-chamber view and the unsatisfactory inter-vendor reproducibility of the results. Finally, the cross-sectional character of the study did not allow us to assess the impact of overweight/obesity on the development of heart failure or survival in patients with congenitally corrected transposition of the great arteries.

CONCLUSIONS

Overweight and obesity in patients with congenitally corrected transposition of the great arteries is associated with impaired function of systemic RV. Since bodyweight is a modifiable risk factor and patients with congenitally corrected transposition of the great arteries might be at risk of heart failure, this is an important finding that warrants further investigation.

Conflict of interest: none declared

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Nadwaga i otyłość a czynność systemowej prawej komory u osób dorosłych z wrodzonym skorygowanym przełożeniem pni tętniczych

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Streszczenie

Wstęp: U chorych z wrodzonym skorygowanym przełożeniem pni tętniczych komora morfologicznie prawa pełni rolę komory systemowej, co sprzyja rozwojowi niewydolności serca w długim okresie obserwacji. Dodatkowe przewlekłe przeciążenie objętościowe komory systemowej wtórne do nadwagi/otyłości może prowadzić do wcześniejszego upośledzenia jej czynności.

Cel: Celem pracy była ocena czynności systemowej prawej komory u dorosłych chorych z wrodzonym skorygowanym przełożeniem pni tętniczych i nadwagą/otyłością.

Metody: Do badania włączono 56 dorosłych osób z wrodzonym skorygowanym przełożeniem pni tętniczych, którzy zgłosili się na rutynową wizytę kontrolną w Instytucie Kardiologii. U każdego chorego wykonano przezklatkowe badanie echokardiograficzne, oznaczono stężenie N-końcowego propeptydu natriuretycznego typu B (NT-proBNP) i wyliczono wskaźnik masy ciała (BMI).

Wyniki: Średni wiek w badanej grupie wyniósł $33,9 \pm 12,9$ roku, 55% stanowili mężczyźni. U 22 chorych stwierdzono nadwagę (BMI 25–29,9 kg/m²) lub otyłość (BMI ≥ 30 kg/m²), a u 34 chorych — prawidłową masę ciała (BMI < 25 kg/m²). Wiek, płeć, częstość rytmu serca i wartości ciśnienia tętniczego były zbliżone w analizowanych grupach. Nie stwierdzono także istotnych różnic w zakresie stężeń NT-proBNP. W badaniu echokardiograficznym u chorych z nadwagą/otyłością zarejestrowano natomiast niższe wartości skurczowej zmiany pola powierzchni systemowej prawej komory w porównaniu z pacjentami z prawidłową masą ciała ($0,38 \pm 0,08$ vs. $0,43 \pm 0,06$; $p = 0,02$). Wartości globalnego odkształcenia podłużnego mięśnia systemowej prawej komory były również istotnie niższe w grupie chorych z nadwagą/otyłością ($-15,3\% \pm 3,4\%$ vs. $-18,3\% \pm 3,6\%$; $p = 0,01$).

Wnioski: Nadwaga/otyłość u dorosłych pacjentów z wrodzonym skorygowanym przełożeniem pni tętniczych dodatkowo upośledza czynność systemowej prawej komory.

Słowa kluczowe: wrodzone skorygowane przełożenie pni tętniczych, systemowa prawa komora, otyłość, echokardiografia

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