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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Exercise-induced changes in left ventricular global longitudinal strain in asymptomatic severe aortic stenosis

Agnieszka K. Lech, Piotr P. Dobrowolski, Anna Klisiewicz, Piotr Hoffman

Department of Congenital Heart Disease, Institute of Cardiology, Warsaw-Anin, Poland

Abstract

Background: The management of patients with asymptomatic severe aortic stenosis (ASAS) is still under discussion. Therefore, it is advisable to search for the parameters of early damage to left ventricular (LV) function.

Aim: The aim of the study was to assess exercise-induced changes in LV global longitudinal strain (GLS) in ASAS.

Methods: The ASAS group consisted of 50 patients (26 women and 24 men, aged 38.4 ± 18.1 years) meeting the echocardiographic criteria of severe aortic stenosis (AVA < 1 cm^2, AVAI < 0.6 cm^2/m^2, Vmax > 4 m/s, mean aortic gradient > 40 mm Hg), with normal LV ejection fraction (LVEF ≥ 55%) and sinus rhythm on electrocardiogram, and without significant concomitant valvular heart diseases. The control group consisted of 21 people matched for age and sex. Echocardiographic examinations and echocardiographic stress tests with the assessment of GLS using the speckle tracking imaging were performed.

Results: The ASAS group was characterised by statistically significantly higher LV mass index (LVMI) and higher LVEF. GLS values at rest in both groups were within normal limits but were significantly higher in the control group (–18.9 ± 2.4% vs. –20.7 ± 1.7%, p = 0.006). An increase in GLS at peak exercise in both groups was observed, lower in the ASAS group (the difference was not statistically significant: –0.8 ± 3.0% vs. –2.2 ± 3.1%, p = 0.086). Changes in GLS during exercise (ΔGLS) did not correlate with the parameters of the severity of aortic stenosis. In the multivariate model, LVMI proved to be a factor associated with GLS at rest and during exercise.

Conclusions: In patients with ASAS, GLS is a non-invasive marker of an early stage of LV myocardial damage associated with myocardial hypertrophy. An increase in GLS during exercise in the ASAS group, smaller than in the control group, indicates a preserved functional reserve of the LV myocardium but smaller than in healthy individuals. The assessment of the clinical usefulness of exercise-induced changes in GLS requires further research.

Key words: global longitudinal strain, asymptomatic severe aortic stenosis, echocardiography, echocardiographic stress test

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INTRODUCTION

Aortic stenosis (AS) is currently the most common valvular disease in adults in developed countries [1, 2]. Surgical treatment is clearly advisable in symptomatic patients. The management of asymptomatic patients is still under discussion [3–5]. Left ventricular ejection fraction (LVEF) below 50% is an indication for surgical treatment [2, 4, 5]. However, LVEF is reduced at a late stage, and it is caused by various mechanisms related to left ventricular (LV) structural changes (hypertrophy, proliferation of connective tissue elements, afterload mismatch). Therefore, it is advisable to search for earlier indicators of LV dysfunction in AS, in the phase when LVEF is still normal. Changes in LV global longitudinal strain (GLS) assessed using speckle tracking echocardiography may be used as such an indicator. The usefulness of this parameter in detecting subtle changes in LV function secondary to various pathologies and their prognostic significance was shown also in AS [6–8]. Still little is known about the importance of exercise-induced changes in GLS in patients with high-gradient asymptomatic severe AS [4, 5, 9, 10].

The aim of the study is to determine exercise-induced changes in left ventricular GLS in this group of patients.

Address for correspondence:
Agnieszka K. Lech, MD, Department of Congenital Heart Disease, Institute of Cardiology, ul. Alpejska 42, 04–628 Warszawa, Poland, e-mail: alech@ikard.pl
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METHODS

Study population

From January 2011 to October 2013 consecutive patients with high-gradient asymptomatic severe aortic stenosis (ASAS group) determined in transthoracic echocardiography were included in the study. The control group consisted of healthy individuals matched for sex and age. Inclusion criteria also included: normal LVEF (≥ 55%), sinus rhythm, the ability to perform physical activity, and age 18–70 years. Exclusion criteria included: the lack of the patient's consent to participate in the study, greater than mild aortic insufficiency, concomitant significant other valvular heart diseases, cardiac arrhythmia, coexistence of myocardial diseases, symptomatic coronary artery disease or myocardial infarction, the occurrence of impaired segmental contractility during the exercise stress test, untreated or poorly controlled hypertension, and lung diseases.

Echocardiography

Transthoracic echocardiography was performed using a Vivid 9 device (GE Healthcare). Severe AS was diagnosed when the aortic valve area (AVA) calculated from the Bernoulli equation was < 1 cm^2 or the aortic valve area index (AVAI) was < 0.6 cm^2/m^2, maximum aortic valve flow velocity (Vmax) was > 4 m/s, and mean aortic gradient (Pmean) was > 40 mm Hg [2]. Rest echocardiography was supplemented with an assessment of LV GLS using speckle tracking imaging. For the subsequent assessment of GLS, apical views: four chamber, two chamber, and three chamber, were recorded accurately. Subsequently, an echocardiographic stress test on a cycloergometer was performed in the supine position, according to the World Health Organisation protocol, beginning at a load of 25 W with increases of 25 W every 2 min in each stage. Electrocardiographic (ECG) monitoring and blood pressure control was performed continuously throughout the exercise stress test. The stress test interruption criteria included: reaching the heart rate limit appropriate for the age, the occurrence of symptoms (shortness of breath, chest pain), a decrease in systolic blood pressure (SBP) by > 10 mm Hg despite an increase in the load, an excessive increase in blood pressure (SBP ≥ 220 mm Hg), ST segment depression on ECG ≥ 2 mm compared to the baseline, complex ventricular arrhythmia, fatigue, and the patient's request. The echocardiography and echocardiographic stress test protocol was identical for the ASAS group and the control group. All examinations were performed by one person with experience in echocardiography (including in GLS measurements and echocardiographic stress tests [A.L.]). The data was analysed off-line (EchoPAC GE Healthcare). The values of GLS at rest and during exercise and its increase (the difference between the value during exercise and at rest GLS — ΔGLS) were subjected to further statistical analysis. Higher GLS values are considered as more negative values according to the parameter's physiological interpretation. The study protocol was approved by the Bioethics Committee at the Institute of Cardiology.

Statistical analysis

Continuous variables were presented as mean ± standard deviation. Student's t-test was used to compare the mean values of the studied parameters. The values of GLS at rest and during exercise were compared using the Student's t-matched pairs test. In order to evaluate the correlation of the studied echocardiographic parameters, Pearson's method was used. In order to identify independent predictors related to the GLS value, linear regression models were developed. Statistically significant parameters in univariate analysis were included in the multivariate linear regression model to determine the independent effect of several variables on the GLS value. A p value < 0.05 was accepted as statistically significant. The SPSS 18.0 statistical package (SPSS Inc., Chicago, IL, USA) was used for calculations.

RESULTS

The ASAS group consisted of 50 patients, 26 women and 24 men, mean age 38.4 ± 18.1 years. Bicuspid aortic valve was found in 31 (62%) patients in the ASAS group. The control group consisted of 21 volunteers matched for sex and age (10 women and 11 men, mean age 43.4 ± 10.6 years). The groups did not differ with regard to body surface area and blood pressure at rest. Both groups reached a similar load during exercise stress tests (132.7 ± 36.9 W in the ASAS group vs. 150 ± 41.1 W in the control group; p = 0.09).

The clinical and echocardiographic characteristic of both groups are presented in Table 1. The patients with ASAS were characterised by statistically significant greater LV mass index (LVMI) and EF value, compared to the control group. During exercise a greater increase in blood pressure was recorded in the control group. LV stroke volume index at rest (SVI bas) was statistically insignificantly higher in the ASAS group compared to the control group, likewise during exercise.

Global longitudinal strain at rest was significantly greater in the control group, but in both groups the values were within the normal range (Table 2) [11–13]. An increase in the GLS value at peak exercise in both groups was observed, lower in the ASAS group but without any statistically significant difference between the groups (ΔGLS –0.8 ± 3.0% vs. –2.2 ± 3.1%; p = 0.08, respectively).

In the paired t test in the ASAS group an increase in GLS was at the limit of statistical significance, and in the control group it was statistically significant (Table 3, Fig. 1A, B).

In the ASAS group, both GLS at rest and during exercise significantly correlated with the echocardiographic parameters of the aortic valve stenosis: AVA (at rest r = 0.237, p = 0.04; during exercise r = 0.252; p = 0.04), peak transvalvular pressure gradient (Pmax) (r = –0.354, p = 0.003; r = –0.414,

Table 1. Clinical and echocardiographic variables in the presented groups

Variables	ASAS (n = 50)	Control group (n = 21)	p
Age [years]	38.4 ± 18.1	43.4 ± 10.6	0.24
Sex (female/male)	26/24	10/11	0.74
BSA [m^2]	1.84 ± 0.24	1.84 ± 0.23	0.97
SBP bas. [mm Hg]	116.5 ± 22.1	123.4 ± 18.4	0.22
DBP bas. [mm Hg]	79.0 ± 10.7	79.7 ± 9.6	0.80
SBP ex. [mm Hg]	162.0 ± 27.2	194.9 ± 29.3	< 0.0001
DBP ex. [mm Hg]	90.8 ± 12.8	95.0 ± 14.9	0.28
PW [Watt]	132.7 ± 36.9	150.0 ± 41.1	0.09
LVMI [g/m^2]	117.0 ± 27.7	88.2 ± 20.6	< 0.0001
SVI bas. [mL/m^2]	46.4 ± 7.1	42.9 ± 13.4	0.09
SVI ex. [mL/m^2]	47.3 ± 9.1	45.4 ± 10.1	0.32
Vmax bas. [m/s]	4.6 ± 0.4	1.3 ± 0.2	< 0.0001
Vmax ex. [m/s]	5.6 ± 0.6	1.9 ± 0.3	< 0.0001
Pmax bas. [mm Hg]	86.2 ± 15.6	7.3 ± 2.7	< 0.0001
Pmean bas. [mm Hg]	52.3 ± 10.6	4.1 ± 1.4	< 0.0001
Pmax ex. [mm Hg]	128.0 ± 28.0	15.9 ± 4.7	< 0.0001
Pmean ex. [mm Hg]	82.3 ± 18.2	9.0 ± 2.9	< 0.0001
AVA bas. [cm^2]	0.8 ± 0.1	2.8 ± 0.7	< 0.0001
AVA ex. [cm^2]	0.8 ± 0.2	2.7 ± 0.6	< 0.0001

ASAS — asymptomatic severe aortic stenosis; BSA — body surface area; SBP — systolic blood pressure; DBP — diastolic blood pressure; PW — maximal load exercise test; LVMI — left ventricular mass index; SVI — stroke volume index; LVEF — left ventricular ejection fraction; Vmax — maximal velocity; Pmax— maximal aortic gradient; Pmean — mean aortic gradient; AVA — aortic valve area; bas. — rest; ex. — exercise

Table 2. Rest end exercise GLS values in the presented groups

Variables	ASAS (n = 50)	Control group (n = 21)	p
GLS bas. [%]	–18.9 ± 2.4	–20.7 ± 1.7	0.006
GLS ex. [%]	–19.7 ± 3.6	–22.8 ± 2.9	0.001
ΔGLS [%]	–0.8 ± 3.0	–2.2 ± 3.1	0.086

ASAS — asymptomatic severe aortic stenosis; ΔGLS — differences between rest and peak exercise global longitudinal strain (GLS); bas. — rest; ex. — exercise

Table 3. Student's t-matched pairs test

Variables	GLS bas.	GLS ex.	p
ASAS (n = 50)	–18.9 ± 2.4	–19.7 ± 3.6	0.064
Control group (n = 21)	–20.7 ± 1.7	–22.8 ± 2.9	0.04

ASAS — asymptomatic severe aortic stenosis; GLS — global longitudinal strain; bas. — rest; ex. — exercise

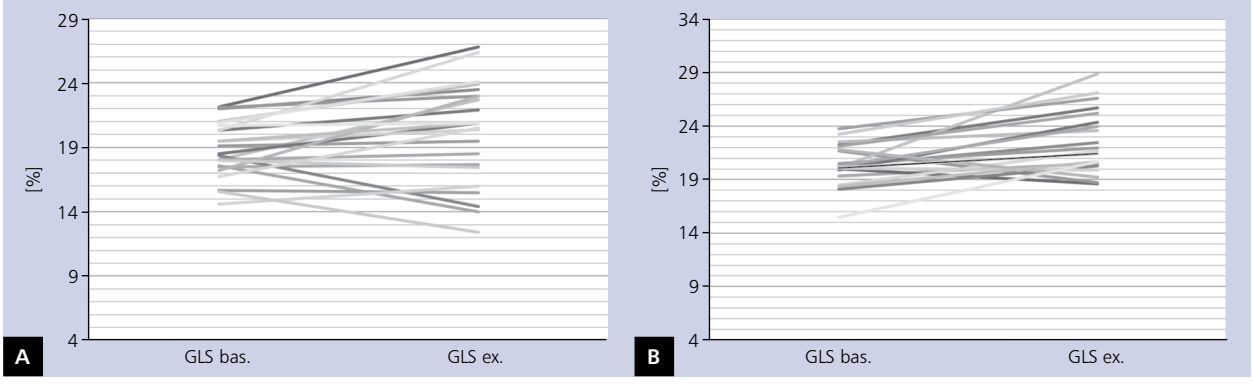

Figure 1. Global longitudinal strain (GLS) at baseline and during exercise in the asymptomatic severe aortic stenosis group (**A**) and the control group (**B**); bas. — rest; ex. — exercise

Table 4. Univariate and multivariate linear regression model for factors associated with global longitudinal strain in resting

Variables	Univariate model		Multivariate model	
	β	p	β	p
Age	0.009	0.44	–	–
Sex	0.400	0.001	–	–
LVMI	–0.546	< 0.0001	–0.539	< 0.0001
Pmax bas.	–0.354	0.003	–	–
Pmean bas.	–0.344	0.004	–	–
AVA bas.	0.237	0.05	–	–

LVMI — left ventricular mass index; Pmax bas — maximal aortic gradient in rest; Pmean bas. — mean aortic gradient in rest; AVA bas. — aortic valve area in rest

Table 5. Univariate and multivariate linear regression model for factors associated with global longitudinal strain in exercise

Variables	Univariate model		Multivariate model	
	β	p	β	p
Age	0.233	0.05	0.257	0.014
Sex	0.09	0.42	–	–
LVMI	–0.502	< 0.0001	–0.499	< 0.0001
Pmax ex.	–0.414	< 0.0001	–	–
Pmean ex.	–0.430	< 0.0001	–	–
AVA ex.	0.252	0.04	–	–

LVMI — left ventricular mass index; Pmax ex. — maximal aortic gradient in exercise; Pmean ex. — mean aortic gradient in exercise; AVA ex. — aortic valve area in exercise

$p < 0.0001$), mean transvalvular pressure gradient (Pmean) ($r = –0.344$, $p = 0.004$; $r = –0.430$, $p < 0.0001$), Vmax ($r = –0.336$, $p = 0.004$; $r = –0.404$, $p = 0.001$), and LVMI ($r = –0.546$; $p < 0.0001$; $r = –0.502$; $p < 0.0001$). Moreover, correlations with age and sex were found.

ΔGLS did not correlate with parameters of the AS at rest: AVA ($r = 0.144$; $p = 0.24$), Pmax ($r = –0.155$; $p = 0.20$), Pmean ($r = –0.199$; $p < 0.09$), age ($r = 0.207$; $p = 0.08$), and LVMI ($r = –0.182$; $p = 0.13$). In addition, no correlation of ΔGLS with the parameters of the AS during exercise was found: AVA, peak and mean aortic gradient ($r = 0.124$; $p = 0.31$; $r = –0.211$; $p = 0.08$; $r = –0.225$; $p = 0.06$, respectively).

In the multivariate model, LVMI was an independent factor related both to GLS at rest and during exercise (Tables 4, 5).

DISCUSSION

Left ventricular pressure overload in ASAS gradually leads to myocyte hypertrophy and with time to extracellular space fibrosis. The effect of this remodelling is progressive LV systolic and diastolic dysfunction, and a decrease in EF < 50% is associated with worse prognosis. Weidemann et al. [14] assessed the degree of LV fibrosis in patients with AS undergoing valve replacement surgery and its association with global and regional LV systolic function and patients' prognosis after surgery. The degree of fibrosis turned out to be an important prognostic factor for improving the longitudinal fibre function and New York Heart Association class in patients after aortic valve replacement surgery. The fibrosis process starts in the LV subendocardial layer leading to gradual damage to longitudinal fibres. Initially, despite myocardium structural changes, it does not cause a decrease in EF [14, 15]. Thus, it is believed, that the assessment of LV longitudinal fibre function can capture the earliest, subclinical stages of damage to its myocardium, as it has been shown for a number of pathologies [6–8, 16, 17].

In a group of patients with asymptomatic severe AS, significantly impaired function of longitudinal fibres at rest was found. Younan et al. [18] showed a negative correlation of GLS with LVMI. Similar results were obtained by Cramariuc et al. [19]. In the study by Dinh et al. [20] GLS correlated with a degree of LV hypertrophy assessed by magnetic resonance imaging and was significantly worse in groups of patients with a greater degree of hypertrophy. At the same time, the authors observed the improvement of GLS values three months after aortic valve replacement surgery. These observations are not confirmed by Donal et al. [4]. In the study by Delgado et al. [6], patients with severe AS had significantly lower GLS compared to patients with hypertension with a similar degree of LV hypertrophy. The authors hypothesised that in patients with severe AS the deterioration of the function of ventricular longitudinal fibres is more affected by the degree of aortic valve stenosis and associated impairment of coronary blood flow. This might result in ischaemia and subendocardial layer fibrosis and play a more important role than the severity of LV hypertrophy.

In the study by Kearney et al. [21], GLS correlated with mean aortic gradient, AVA, LVEF, and LVMI, but it did not correlate with the occurrence of diabetes and coronary heart disease. In the prospective observation a value of GLS > –15% turned out to be an independent predictor of adverse cardiovascular events (25% one-year survival without cardiovascular events in patients with more reduced GLS compared to 58% in patients with more than –15% GLS at rest) [21]. Lancellotti et al. [22] obtained similar results in a study based on a group of 163 patients. They found that during 20 ± 19 months GLS ≥ –15.9% was a predictor of symptoms, mortality, or aortic valve replacement surgery [22]. It should be noted that in this study analyses were based on both moderate and severe AS patients.

In the study by Kusunose et al. [23], GLS > –12.1% was associated with significantly reduced survival after surgery despite normal LVEF.

In our study, longitudinal strain at rest in the ASAS group was within normal range, although significantly lower

than in the control group (–18.9 ± 2.4% vs. –20.7 ± 1.7%, p = 0.006). It correlated with the echocardiographic parameters of the AS (AVA, Pmax, Pmean, Vmax) and with LVMI. In our study, the average values of GLS in the ASAS group higher than in the cited studies are probably due to the fact that patients were younger, asymptomatic, and without concomitant diseases.

There are few studies assessing changes in GLS during exercise in patients with AS. Schnell et al. [17] found that exercise-induced GLS was more severe in patients with ASAS than in a hypertrophic cardiomyopathy group despite similar resting values of GLS in both groups. In the author's opinion, higher afterload in a group of patients with AS than in hypertrophic cardiomyopathy is responsible for the observed difference. Donal et al. [4], in patients with moderate and severe AS, found impaired function of longitudinal fibres at rest compared to the healthy group. On the other hand, during exercise there was an increase in GLS values in both groups, smaller in the AS group. Significantly reduced contractile reserve was especially pronounced in patients with AS with abnormal stress test results. In a multivariate analysis, lower GLS at rest, a greater increase in the average transvalvular gradient during exercise, and smaller changes in GLS (ΔGLS) during exercise were independent predictors of abnormal response to exercise [4].

In our study the effect of physical exercise on GLS in patients with asymptomatic severe AS was analysed. In both groups an increase in strain at peak exercise was observed, in the ASAS group with borderline statistical significance, whereas in the control group it was statistically significant. This confirms preserved contractile reserve in both groups, but this is smaller in the group with asymptomatic severe AS.

Moreover, in the multivariate model LVMI was an independent factor associated with GLS at rest and during exercise, but no such relationship for echocardiographic parameters of the AS (AVA, Pmax, and Pmean) was found, both at rest and during exercise. This indicates the usefulness of the assessment of LV GLS and underlines the importance of the degree of myocardial hypertrophy in qualification for aortic valve replacement. In the current recommendations significant hypertrophy is an indication for aortic valve replacement in a group of asymptomatic patients only with recommendation class IIb (ESC). In our study, we did not show any correlation between change in longitudinal strain during exercise (ΔGLS) and parameters of severity of AS.

Based on the observed significant differences in GLS values at rest between the ASAS group and the control group, we expected to find additional data confirming LV dysfunction after exercise, depending on the severity of AS. Nevertheless, the results obtained did not confirm our assumptions. This finding may be caused by the small number of subjects in the group. Therefore, we plan the assessment of subsequent patients according to the protocol presented in this manuscript.

Limitations of the study

We understand that this study has its limitations. It was based on a relatively small sample size. Further longitudinal studies with assessment of clinical outcomes during follow-up are essential. This may prove the clinical applicability of our findings for the management of asymptomatic patients with severe aortic stenosis.

CONCLUSIONS

Many studies have shown that GLS is a non-invasive indicator reflecting an early stage of LV myocardial damage. In the present study, despite significant differences in GLS values at rest between the ASAS group and the control group, the assessment of exercise-induced changes in this parameter did not bring any additional information helpful in making decisions about surgery treatment. The observed increase in GLS values at peak exercise, insignificantly lower than in the control group, indicates a preserved functional reserve in this group of patients, although smaller than in healthy individuals. Further studies are needed with prospective observation in order to determine the role of GLS in the risk stratification of patients with AS and suitability in the qualification of asymptomatic patients for surgical treatment. It is worth noting that both our study and many cited ones indicate a significant relationship between LV mass and GLS. Maybe the degree of myocardial hypertrophy should be a stronger argument for sending asymptomatic patients for surgical treatment than it is currently. This requires further research and observation.

Conflict of interest: *none declared*

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Wysiłkowe zmiany globalnego podłużnego odkształcenia lewej komory w ciasnym bezobjawowym zwężeniu zastawki aortalnej

Agnieszka K. Lech, Piotr P. Dobrowolski, Anna Klisiewicz, Piotr Hoffman

Klinika Wad Wrodzonych Serca, Instytut Kardiologii, Warszawa-Anin

Streszczenie

Wstęp: Postępowanie z pacjentami z ciasnym bezobjawowym zwężeniem zastawki aortalnej (ASAS) pozostaje nadal przedmiotem dyskusji. Dlatego celowe jest poszukiwanie parametrów wczesnego uszkodzenia funkcji mięśnia lewej komory.

Cel: Celem pracy była ocena wysiłkowych zmian globalnego odkształcenia podłużnego (GLS) mięśnia lewej komory w przebiegu ASAS.

Metody: Grupę ASAS stanowiło 50 pacjentów (26 kobiet i 24 mężczyzn, wiek 38,4 ± 18,1 roku) spełniających kryteria echokardiograficzne istotnego zwężenia zastawki aortalnej (AVA < 1 cm^2, AVAI < 0,6 cm^2/m^2, Vmax > 4 m/s, średni gradient aortalny > 40 mm Hg), z prawidłową frakcją wyrzutową lewej komory (LVEF ≥ 55%), rytmem zatokowym w elektrokardiogramie, bez istotnych współistniejących wad zastawkowych. Grupę kontrolną stanowiło 21 osób dobranych pod względem płci i wieku. Wykonywano badanie echokardiograficzne i echokardiograficzny test wysiłkowy z oceną GLS metodą śledzenia markerów akustycznych.

Wyniki: Grupa ASAS charakteryzowała się istotnie statystycznie większą indeksowaną masą mięśnia lewej komory (LVMI) i wyższą wartością LVEF. Wartości spoczynkowego GLS w obu grupach mieściły się w normie, ale były istotnie wyższe w grupie kontrolnej (–18,9 ± 2,4% *vs*. –20,7 ± 1,7%, p = 0,006). Obserwowano wzrost GLS na szczycie wysiłku w obu grupach, niższy w grupie ASAS (różnica nieistotna statystycznie –0,8 ± 3,0% *vs*. –2,2 ± 3,1%, p = 0,086). Zmiany GLS w czasie wysiłku (ΔGLS) nie korelowały z parametrami stopnia ciężkości wady. W modelu wieloczynnikowym parametrem związanym ze spoczynkowym i wysiłkowym GLS okazała się LVMI.

Wnioski: U pacjentów z ASAS GLS jest nieinwazyjnym markerem wczesnego etapu uszkodzenia mięśnia lewej komory związanego z jego przerostem. Wzrost GLS w czasie wysiłku w grupie ASAS, mniejszy niż w grupie kontrolnej, wskazuje na zachowaną rezerwę czynnościową mięśnia lewej komory, ale mniejszą niż u osób zdrowych. Ocena przydatności klinicznej wysiłkowych zmian GLS wymaga przeprowadzenia dalszych badań.

Słowa kluczowe: globalne odkształcenie podłużne, ciasna bezobjawowa stenoza aortalna, echokardiografia, echokardiograficzny test wysiłkowy

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Adres do korespondencji:
lek. Agnieszka K. Lech, Klinika Wad Wrodzonych Serca, Instytut Kardiologii, ul. Alpejska 42, 04–628 Warszawa, e-mail: alech@ikard.pl
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