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Right ventricular and right atrial free wall deformation predicitive value in transformation of preclinical diastolic disfunction to heart failure with preserved ejection fraction

Aim	To study echocardiographic parameters of heart chamber strain in patients with left ventricular (LV) preclinical diastolic dysfunction (PDD) for determining predictors of the PDD transition to heart failure with preserved LV ejection fraction (HFpEF).
Material and methods	The study included 113 patients (including 69 women) with metabolic syndrome and LV PDD (mean age, 65±7 years). The control group consisted of 40 healthy individuals (mean age, 63.0±6.0 years, including 59% women). Metabolic syndrome was diagnosed in consistency with criteria of NCEP-ATP III 2001. PDD was diagnosed based on the absence of heart failure symptoms, normal level of brain natriuretic peptide, and the presence of at least three of the following echocardiographic criteria at rest or after diastolic stress-echocardiography (stress-echoCG): left atrial volume index (LAVI) >34 ml/m^2; the ratio of peak early transmitral filling velocity (E) to average lateral and medial mitral annular velocity (e'), E/e' >14, e' <8.5, and peak tricuspid regurgitation velocity >2.8 m/s. EchoCG that determined LV longitudinal strain (LS), right ventricular (RV) LS, right atrial (RA) LS, and left atrial (LA) LS was performed every year during the 3-year follow-up.
Results	During the follow-up period, 31 patients developed HFpEF. 19 of them reported symptoms while in the other 12 patients, HFpEF was detected by diastolic stress-echoCG. Patients with HFpEF had significantly lower absolute values of RV LS, LA LS, and RA LS (– 27.8±2.9 in the PDD group vs. –23.8±3.2 in the HFpEF group; p<0.03; 38.2±9.1 vs. 28.6±10.2; p<0.03; and 46.2±10.4 vs. 31.6±8.3; p<0.03, respectively). RV LS and RA LS were the strongest independent predictors for PDD transformation into HFpEF (odds ratio, OR, 2.7; 95% confidence interval, CI, 1.48–2.91; p<0.001 and OR 2.6; 95% CI: 1.40–2.75; p<0.001, respectively).
Conclusion	PDD is not a separate clinical nosology but rather an initial stage in the pathogenesis of HFpEF. Approximately ⅓ of PDD patients develop HFpEF. RV LS and RA LS are considered predictors of HFpEF. The duration of PDD is apparently an important factor that provides the development of HFpEF.

Keywords	Diastolic dysfunction; heart failure; preserved ejection fraction; longitudinal strain
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Introduction

Heart failure with preserved ejection fraction (HFpEF) is one of the relevant issues of cardiology with consistently elevated prevalence worldwide and with morbidity and mortality comparable with those in heart failure with reduced ejection fraction [1–3]. The diagnosis of HFpEF is complicated by the presence of often several comorbidities, each of which may have the same symptom complex as in HFpEF. Therefore, an algorithm for step-by-step diagnosis of HFpEF is adopted in order to exclude comorbidities as the cause of dyspnea and make the diagnosis based on integrated data [4–6].

HFpEF develops in diastolic dysfunction (DD) enough to cause pulmonary vascular congestion. Thus, left ventricular (LV) DD is a necessary link in the pathogenesis of HFpEF [7]. However, many patients do not develop HFpEF even in the presence of severe DD, and they have preclinical DD and, vice versa, symptoms may occur in the presence of silent DD [8, 9]. Despite its high prevalence, preclinical DD has not been properly studied. Particularly, the mechanism of its transformation to HFpEF and the predictors of such transformation are unknown. Moreover, patients with DD of the same degree may have symptoms

of varying severity, and other patients do not develop HFpEF.

This study investigates echocardiographic parameters of heart chamber strain in patients with preclinical LV DD to determine predictors of the transformation of preclinical DD into HFpEF. Since high LV intracavitary pressure in DD eventually passes to the right heart, we hypothesized that the initial changes in the functional parameters of the right heart and, in particular, the parameters of myocardial strain may be indicative of the possibility of the transformation of preclinical DD into HFpEF and serve as predictors of such transformation.

Material and methods

The study included 113 (69 female) patients with metabolic syndrome and preclinical DD (mean age 65±7 years). The control group included 40 healthy individuals (mean age 63.0±6.0 years; 59% of female patients). According to the 2001 NCEP-ATP III criteria, metabolic syndrome was diagnosed subject to the presence of at least three indicators: low high-density lipoprotein cholesterol (<40 mg/dl in male patients and <50 mg/dl in female patients), high normal blood pressure (>130/85 mm Hg) or arterial hypertension (AH), enlarged waist circumference (>102 cm in male patients and >88 cm in women), high levels of triglycerides (≥ 150 mg/dl) and fasting glucose (>100 mg/dl) [10]. Prior to inclusion in the study, patients received antihypertensive and/or antidiabetic therapy as required.

Hemodynamically significant valvular damages, liver, and kidney pathology, cor pulmonale, unstable angina, constrictive pericarditis, restrictive or hypertrophic cardiomyopathy were the exclusion criteria.

Preclinical DD was diagnosed based on normal levels of N-terminal pro-brain natriuretic peptide (NT-proBNP) and the presence of at least three of the following echocardiographic criteria at rest or after diastolic stress echocardiography: left atrial volume index (LAVI) >34 ml/m^2, the ratio of peak mitral inflow velocity (E) to peak early diastolic mitral annular velocity (e') E/e' >14, e' <8.5, peak tricuspid regurgitation velocity >2.8 m/s [11]

Echocardiography was performed following the to the American Society of Echocardiography guidelines using a Vivid 7 Dimension device and M4S and 3V probes at the rate of at least 50 fps [12]. All images were acquired and saved with in breath-holding spells. Videos and Doppler data of 3 cycles were saved to be reviewed later using EchoPack. LVEF was measured automatically using the Simpson's biplane method. Left atrial volume was measured at the end of ventricular systole, then the LAVImax index was calculated by dividing by body surface area (BSA). LV mass was calculated using the area-length method, then left ventricular mass index (LVMI) was calculated by dividing by BSA [12]. Early (E) and late (A) transmitral and transtricuspid flow velocities and E decay time, were measured from the 4 chamber apical view in pulse Doppler mode, a mark was placed between the tips of the mitral and tricuspid valves in diastole at 100 mm/s. The mean values of 3 consecutive cycles were recorded. Mitral and tricuspid annular velocities were measured in diastole by averaging the lateral and medial velocities in early diastole (e'). The E/e'ratio was measured to assess the LV and right ventricular (RV) filling pressure [11].

Treadmill diastolic stress echocardiography was performed to detect preclinical DD or HFpEF. Patients performed treadmill exercise until the heart rate increased to 120 bpm or dyspnea appeared. The test was considered positive if all of the following criteria were met: E/e' >14, lateral mirtal annular e' <10 cm/s or medial mitral annular e' <7 cm/s, and peak tricuspid regurgitation velocity (TRV) >2.8 m/s (Table 1) [11].

LV longitudinal strain (LVLS) was measured in the automated image function (AIF) mode when standard apical slices were acquired and saved, then LVLS was calculated in EchoPac. Both ends of the mitral annulus base and the LV apex were marked, after which the LV walls were automatically traced. Manual correction was performed, if necessary, after which the segment-wise and total 17 segment LVLS was calculated automatically (Figure 1, A).

RV free wall longitudinal strain (RVLS) was measured from the apical 4 chamber view adapted for RV imaging; 6 segments including the interventricular septum (IVS) were analyzed using EchoPac, after which the data of 3 RV free wall segments were averaged (Figure 1, B).

Left atrial LS (LALS) and right atrial LS (RALS) were also measured in the speckle tracking AFI mode from the apical 4 chamber view in EchoPac. LALS was calculated as the mean of two mitral annuls segments after manual exclusion of the remaining segments from the analysis, given that these LA segments are either the oval fossa in the interatrial septum (IAS), where there is almost no myocardium, or LA appendage, or segment covered by the aortic root, or the entry points of the pulmonary veins. RALS was determined from the 4 chamber apical view as the mean of 3 free wall segments and 1 IAS segment next to the tricuspid annulus excluding the oval fossa area (Figure 2). Only the maximum values of atrial strain in the reservoir phase were taken into account.

All measurements were made by a single expert. The intrastudy variability of LVLS, RVLS, LALS, and RALS was estimated in 10 subjects (5 patients and 5 healthy individuals) – 8% and 7% respectively. Patients were followed up for 3 years. Scheduled echocardiography was performed annually. If dyspnoe, palpitations, or weakness occurred during exercise or at rest during the follow-up period, all examinations were repeated.

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The study complies with the principles of the Declaration of Helsinki.

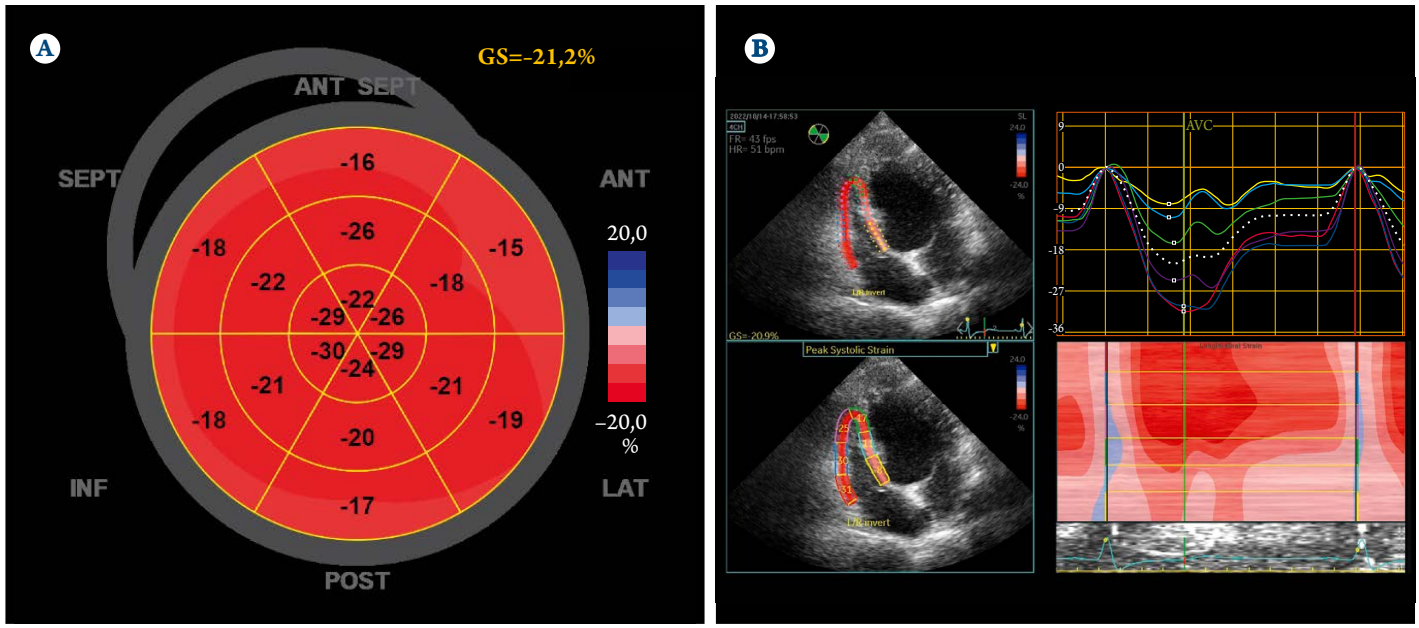
The statistical analysis of data obtained was carried out in SPSS v.16.0. The variables were tested to determine a type of distribution using the Kolmogorov-Smirnov test. Continuous parameters are expressed as the means and the standard deviations (M±SD), and categorical parameters are described as the absolute values and the percentages. The data were analyzed using the parametric Student's test and the non-parametric Mann–Whitney U-test. Linear dependencies

Table 1. Diagnosis of DD using diastolic stress echocardiography

Parameter	Absence of DD	Presence of DD
E/e'	<10	>14
TRV, m/s	<2.8	>2.8
e' lat, cm/s	—	<10
e' med, cm/s	—	<7

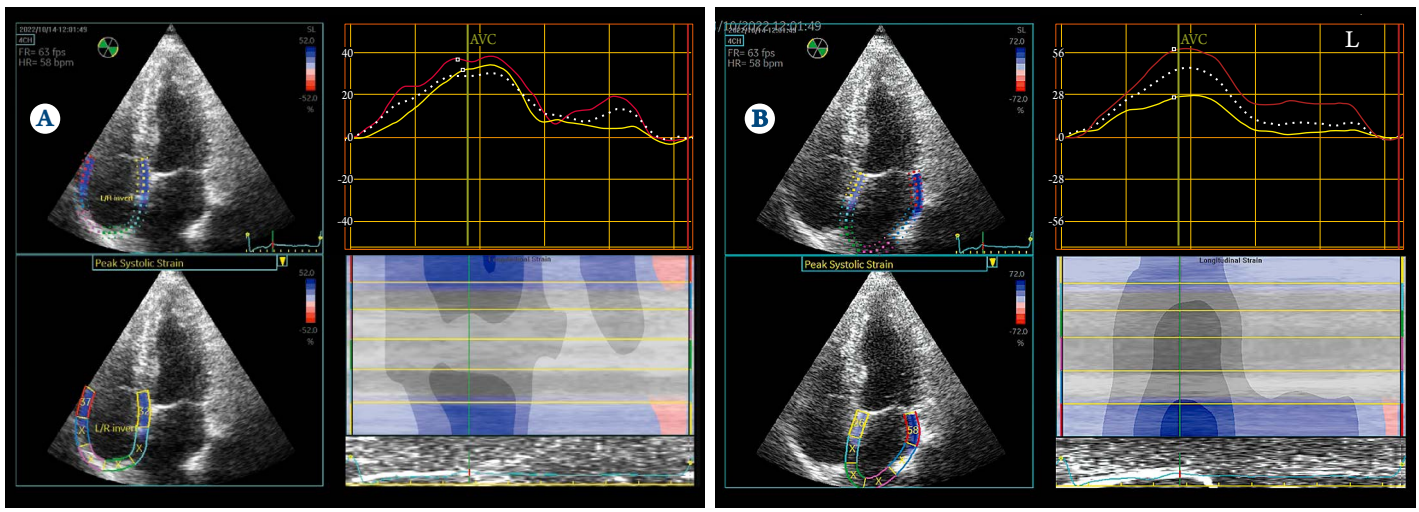
DD, diastolic dysfunction; TRV, tricuspid regurgitation velocity;
e' lat, early diastolic lateral mitral annular velocity; e' med, early diastolic medial mitral annular velocity.

Figure 1. Total and longitudinal strain of the left ventricle (A) and calculation of the longitudinal strain of the right ventricular free wall (B)



Values of interventricular septal strain were not taken into account.

Figure 2. Calculation of longitudinal strain of the left (A) and right (B) atria



were estimated in the correlation analysis. R values not less than 0.4 were considered a significant correlation between the parameters. Binary regression analysis was used to identify independent predictors of IMR.

The differences were considered statistically significant with p values being less than 0.05.

Results

Patients with LV preclinical DD had significantly lower absolute rates of strain of the heart chamber strain, LAVI, and LVMI compared to the control group, and there were no significant differences in LV and RV systolic function. LALS was significantly lower in the preclinical DD group than in the control group (Table 2).

During the 3-year follow-up period, 31 patients developed HFpEF. Of them, 19 patients reported the onset of symptoms, and in the remaining 12 patients, HFpEF was detected during exercise stress echocardiography (Table 3).

Patients with HFpEF had significantly high LVMI, LAVI, and low absolute values of RVLS, LALS, RALS, and higher values of TRV reflecting systolic pressure in the pulmonary artery (Table 4).

There was a close correlation of the results between LVLS and LALS, on the one hand, and RVLS and RALS, on the other hand, and between LALS and RALS (Table 5).

We performed an analysis to determine the independent predictors of the transformation of preclinical DD into HFpEF (Table 6).

Table 6 shows only independent predictors of the transformation of preclinical DD into HFpEF. RVLS and RALS were the strongest independent predictors of HFpEF.

Discussion

HFpEF is difficult to diagnose, subject to the presence of DD. However, the correlation between the severity of DD and its symptoms is non-linear. Severe DD is silent in many patients and is considered preclinical, which often does not transform into a symptomatic form, which is HFpEF [8, 9]. Preclinical DD is an under-studied issue. Generally, there are very few original works devoted to the natural course of preclinical DD and its clinical progression to HFpEF [13]. We showed that, even preclinical DD does not progress to HFpEF in many patients, it is not a separate clinical syndrome but a stage preceding HFpEF [8]. Preclinical DD occurs in 20–35% of the elderly population, and its frequency increases with age and in the presence of comorbidities [14, 15]. The progression of preclinical DD to HFpEF is most often due to the presence of both cardiovascular and non-cardiovascular factors. For example, patients with preclinical DD and diabetes mellitus, coronary artery disease or AH are at a higher risk of the progression of preclinical DD to HFpEF than patients with the same diseases but without preclinical DD [9]. Moreover, it was shown that effective treatment of patients with AH prevents the progression of preclinical DD to HFpEF [16].

Despite these findings, it is difficult to predict which patient with asymptomatic DD is likely to develop HFpEF. Moreover, there few studies that investigate specific parameters as predictors of the transformation of preclinical DD to HFpEF [17].

This study is devoted to identifying the factors contributing to the transformation of preclinical DD to HFpEF and identifying the predictors of this transformation.

According to our data, 27.5% of patients with preclinical DD developed HFpEF. Our data are consistent with the study by Correa de Sa et al. [17], in which a moderate progression of preclinical DD to HFpEF was observed in 31.1% of patients within the 2-year follow-up period. The authors also observed that only the presence of peripheral

Table 2. Patient demographic, clinical, and echocardiographic data

Parameter	Main group (n = 113)	Control group (n = 40)	p
Female, %	61	59	0.3
Age, years	65.0±7.0	63.0±6.0	0.5
BMI, kg/m^2	27.4±3.3	21.2±2.1	0.03
SBP, mm Hg	142.0±11.0	119.0±14.0	0.03
DBP, mm Hg	93.0±4.0	79.0±6.0	0.04
Total cholesterol, mmol/L	5.9±1.3	4.1±1.1	0.04
Diabetes mellitus, %	26.0	0	–
LVMI, kg/m^2	117.2±16.8	78.3±12.6	0.03
LAVI	32.3±8.2	23.4±9.1	0.02
LVESVI, mL/m^2	19.5±4.3	21.9±6.2	0.08
LVEDVI, mL/m^2	50.6±8.5	53.2±9.3	0.1
LVEF, %	65.8±3.9	61.4±4.6	0.07
e’	7.6±0.9	10±1.3	0.02
LVLS, %	–19.7±1.6	–20.3±2.1	0.06
RVLS, %	–27.3±3.2	–28.4±3.9	0.09
LALS, %	39.2±9.1	47.6±10.2	0.03
RALS, %	44.3±10.1	48.8±11.2	0.06

BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; LVMI, left ventricular mass index; LAVI, left atrial volume index; LVESVI, left ventricular end-systolic volume index; LVEDVI, left ventricular end-diastolic volume index; LVEF, left ventricular ejection fraction; e’, mean early diastolic lateral and medial mitral annular velocity; LVLS, left ventricular longitudinal strain; RVLS, right ventricular longitudinal strain; LALS, left atrial longitudinal strain; RALS, right atrial longitudinal strain.

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Table 3. Number of patients with HFpEF

Parameter	Year of follow-up			
	1	2	3	Total
Total, n (%)	9 (8.0)	8 (7.1)	14 (12.4)	31 (27.5)
Female, n (%)	5 (55.6)	5 (62.5)	7 (50.0)	17 (54.8)
According to DST, n (%)	2 (22.0)	4 (50.)	6 (42.9)	12 (38.7)

HFpEF, heart failure with preserved ejection fraction; DST, diastolic stress test.

Table 4. Indicators of patients with HFpEF and patients with preclinical DD not transformed to HFpEF at the end of follow-up

Parameter	Patients with preclinical DD (n = 82)	Patients with HFpEF (n = 31)	p
BMI, kg/m^2	26.7±3.1	28.1±3.4	0.08
SBP, mm Hg	135.0±11.0	137.0±12.0	0.10
DBP, mm Hg	83.0±6.0	85.0±8.0	0.09
Total cholesterol, mmol/L	4.8±1.3	4.7±1.1	0.11
Glucose, mg/dL	5.9±1.4	6.1±1.2	0.09
LVMI, kg/m^2	99.5±11.8	123.3±12.6	0.04
LAVI	33.1±8.5	41.7±8.8	0.02
LVESVI, mL/m^2	19.5±4.5	18.9±4.1	0.06
LVEDVI, mL/m^2	51.7±8.2	49.2±7.8	0.10
LVEF, %	62.7±3.5	63.2±3.1	0.08
e’	7.1±1.2	6.9±1.4	0.07
LVLS, %	–19.3±1.4	–17.2±1.7	0.04
RVLS, %	–27.8±2.9	–23.8±3.2	0.03
LALS, %	38.2±9.1	28.6±10.2	0.03
RALS, %	46.2±10.4	31.6±8.3	0.03
TRV, m/s	2.1±1.6	3.2±1.3	0.02

BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; LVMI, left ventricular mass index; LAVI, left atrial volume index; LVESVI, left ventricular end-systolic volume index; LVEDVI, left ventricular end-diastolic volume index; LVEF, left ventricular ejection fraction; e’, mean early diastolic lateral and medial mitral annular velocity; LVLS, left ventricular longitudinal strain; RVLS, right ventricular longitudinal strain; LALS, left atrial longitudinal strain; RALS, right atrial longitudinal strain; TRV, tricuspid regurgitation velocity.

Table 5. Results of the correlation analysis of the left and right heart strain values (Pearson’s r-test)

Parameter	LVLS	LALS	RVLS	RALS	LAVI
LVLS	—	0.53*	0.41	0.35	0.49*
LALS	–0.53*	—	0.36	0.58*	0.57*
RVLS	0.41	0.36	—	0.53*	0.41
RALS	0.35	0.58	0.53	—	0.57*
LAVI	0.49*	0.57*	0.1	0.57*	—

* Significant correlation (p <0.05) LVLS, left ventricular longitudinal strain; LALS, left atrial longitudinal strain; RVLS, right ventricular longitudinal strain; RALS, right atrial longitudinal strain; LAVIm left atrial volume index.

Table 6. Results of binary regression analysis

Parameter	Wald	P	OR	95% CI
LVLS	10.835	0.009	2.113	1.437–2.214
LALS	13.947	0.007	2.367	1.241–2.473
RVLS	18.234	0.001	2.689	1.478–2.831
RALS	21.138	0.001	2.598	1.398–2.752
LAVI	15.212	0.007	2.312	1.132–2.350

OR, odds ratio; CI, confidence interval; LVLS, left ventricular longitudinal strain; LALS, left atrial longitudinal strain; RVLS, right ventricular longitudinal strain; RALS, right atrial longitudinal strain; LAVI, left atrial volume index.

vascular disease and AH served in patients with preclinical DD as an independent factor of the development of HFpEF.

Our study included only patients with metabolic syndrome who were at a significant risk of the transformation of preclinical DD to HFpEF according to the data of the mentioned studies.

Studies of the transformation of preclinical DD to HFpEF are mainly epidemiological [13]. Our study of the parameters of the heart chamber LS revealed that the values of LS of the right heart were the most powerful predictors of the transformation of preclinical DD to HFpEF. Apparently, this is due to the fact that patients with low LS of the right heart and, thus, preclinical dysfunction, had longer preclinical DD that passed via the pulmonary circulation to the right heart, which reflected in the values of LS of the right heart. Thus, we assume that the duration of preclinical DD plays an important role in the development of HFpEF.

According to our findings, there was a close correlation between LALS and RALS, which can be explained by the presence of a common septum in both chambers of the heart rather than by the transfer of increased filling pressure via the pulmonary circulation. However, we took into consideration only the basal (supravalvular) segment of the septum, without middle and upper ones. Therefore, the correlation is difficult to explain only by the common IAS Moreover, there was a correlation between LVLS and RVLS, which can also be explained by the presence of the common IVS. However, we also did not include the middle and upper segments of IVS in the RVLS analysis, which is why the association of both ventricles via the pulmonary circulation is a more likely explanation for this correlation. The correlation was also observed in our study between RVLS and RALS. These data are not consistent with the data found by Padeletti et al. [18], according to which the authors explain the lack of correlation due to the inclusion of IVS in the RVLS analysis, as IVS strain depends also on the LV.

In our study, patients with HFpEF had higher values of TRV that reflect pulmonary artery systolic pressure.

Conclusion

Preclinical diastolic dysfunction is an initial stage in the pathogenesis of heart failure with preserved ejection fraction rather than a separate clinical nosology. Approximately one in three patients with preclinical diastolic dysfunction develop heart failure with preserved ejection fraction. The duration of preclinical diastolic dysfunction is an important contributing factor for heart failure with preserved ejection fraction. According to our regression analysis, right ventricular longitudinal strain and right atrial longitudinal strain are predictors of the development of heart failure with preserved ejection fraction.

Limitations

This is a single-center study and measurements were performed by a single expert. Perhaps larger population would have allowed more accurately characterizing the course of preclinical DD and its transformation. Short duration of the study may also be a limitation. According to Kane et al. [19], the prevalence of DD increased in the first 4 years, and it progressed in some patients to HFpEF within a 6 year follow-up period.

No conflict of interest is reported.

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REFERENCES

1. Greenberg B. Heart Failure Preserved Ejection Fraction With Coronary Artery Disease. Journal of the American College of Cardiology. 2014;63(25):2828–30. DOI: 10.1016/j.jacc.2014.03.033
2. Gerber Y, Weston SA, Redfield MM, Chamberlain AM, Manemann SM, Jiang R et al. A contemporary appraisal of the heart failure epidemic in Olmsted County, Minnesota, 2000 to 2010. JAMA Internal Medicine. 2015;175(6):996–1004. DOI: 10.1001/jamainternmed.2015.0924
3. Bursi F, Weston SA, Redfield MM, Jacobsen SJ, Pakhomov S, Nkomo VT et al. Systolic and Diastolic Heart Failure in the Community. JAMA. 2006;296(18):2209–16. DOI: 10.1001/jama.296.18.2209
4. Penicka M, Bartunek J, Trakalova H, Hrabakova H, Maruskova M, Karasek J et al. Heart Failure With Preserved Ejection Fraction in Outpatients With Unexplained Dyspnea. Journal of the American College of Cardiology. 2010;55(16):1701–10. DOI: 10.1016/j.jacc.2009.11.076
5. Penicka M, Vanderheyden M, Bartunek J. Diagnosis of heart failure with preserved ejection fraction: role of clinical Doppler echocardiography. Heart. 2014;100(1):68–76. DOI: 10.1136/heartjnl-2011-301321
6. Pieske B, Tschöpe C, de Boer RA, Fraser AG, Anker SD, Donal E et al. How to diagnose heart failure with preserved ejection fraction: the HFA–PEFF diagnostic algorithm: a consensus recommendation from the Heart Failure Association (HFA) of the European Society of Cardiology (ESC). European Heart Journal. 2019;40(40):3297–317. DOI: 10.1093/eurheartj/ehz641
7. Translational Approach to Heart Failure. (Smiseth OA, Remme EW, Opdahl A, Aakhus S, Skulsad H. Heart failure with normal left ventricular ejection fraction: Basic principles and clinical diagnostics. P. 25-61. doi:10.3390/jcdd3030027). Bartunek J, Vanderheyden M, editors -New York, NY: Springer New York;2013. - 516 p. ISBN 978-1-4614-7344-2
8. Louridas G, Lourida K. Heart Failure in Patients with Preserved Ejection Fraction: Questions Concerning Clinical Progression. Journal of Cardiovascular Development and Disease. 2016;3(3):27. DOI: 10.3390/jcdd3030027
9. Wan S-H, Vogel MW, Chen HH. Pre-Clinical Diastolic Dysfunction. Journal of the American College of Cardiology. 2014;63(5):407–16. DOI: 10.1016/j.jacc.2013.10.063
10. National Cholesterol Education Program (NCEP): Expert Panel on Detection and Treatment of High Blood Cholesterol in Adults. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) Final Report. Circulation. 2002;106(25):3143–421. DOI: 10.1161/circ.106.25.3143

ISSN 0022-9040. Kardiologiia. 2023;63(8). DOI: 10.18087/cardio.2023.8.n1682

11. Nagueh SF, Smiseth OA, Appleton CP, Byrd BF, Dokainish H, Edvardsen T et al. Recommendations for the evaluation of left ventricular diastolic function by echocardiography: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. Journal of the American Society of Echocardiography. 2016;29(4):277–314. DOI: 10.1016/j.echo.2016.01.011
12. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L et al. Recommendations for Cardiac Chamber Quantification by Echocardiography in Adults: An Update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. European Heart Journal – Cardiovascular Imaging. 2015;16(3):233–71. DOI: 10.1093/ehjci/jev014
13. Lam CSP, Lyass A, Kraigher-Krainer E, Massaro JM, Lee DS, Ho JE et al. Cardiac Dysfunction and Noncardiac Dysfunction as Precursors of Heart Failure With Reduced and Preserved Ejection Fraction in the Community. Circulation. 2011;124(1):24–30. DOI: 10.1161/CIRCULATIONAHA.110.979203
14. Mureddu GF, Agabiti N, Rizzello V, Forastiere F, Latini R, Cesaroni G et al. Prevalence of preclinical and clinical heart failure in the elderly. A population-based study in Central Italy. European Journal of Heart Failure. 2012;14(7):718–29. DOI: 10.1093/eurjhf/hfs052
15. Moser M, Hebert PR. Prevention of disease progression, left ventricular hypertrophy and congestive heart failure in hypertension treatment trials. Journal of the American College of Cardiology. 1996;27(5):1214–8. DOI: 10.1016/0735-1097(95)00606-0
16. Adamyan K.G., Tunyan L.G., Chilingaryan A.L., Tumasyan L.R. Predictors of heart failure with preserved ejection fraction in patients with metabolic syndrome and preclinical diastolic dysfunction. Kardiologiia. 2017;57(2):23–9. [Russian: Адамян К.Г., Тунян Л.Г., Чилингарян А.Л., Тумасян Л.Р. Предикторы сердечной недостаточности с сохраненной фракцией выброса у пациентов с метаболическим синдромом и доклинической дистолической дисфункцией. Кардиология. 2017;57(2):23-9]. DOI: 10.18565/cardio.2017.2.23-29
17. Correa de Sa DD, Hodge DO, Slusser JP, Redfield MM, Simari RD, Burnett JC et al. Progression of preclinical diastolic dysfunction to the development of symptoms. Heart. 2010;96(7):528–32. DOI: 10.1136/hrt.2009.177980
18. Padeletti M, Cameli M, Lisi M, Malandrino A, Zacà V, Mondillo S. Reference Values of Right Atrial Longitudinal Strain Imaging by Two-Dimensional Speckle Tracking. Echocardiography. 2012;29(2):147–52. DOI: 10.1111/j.1540-8175.2011.01564.x
19. Kane GC, Karon BL, Mahoney DW, Redfield MM, Roger VL, Burnett JC et al. Progression of left ventricular diastolic dysfunction and risk of heart failure. JAMA. 2011;306(8):856–63. DOI: 10.1001/jama.2011.1201