Coronary sinus dilatation is a sign of impaired right ventricular function in patients with heart failure

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Abstract

Objective: The coronary sinus (CS) has been largely ignored by physicians due to a lack of adequate data about the importance of CS enlargement in patients with heart failure (HF). We aimed to assess whether CS dilatation develops in patients with HF and to demonstrate its relation with global myocardial function of the right ventricle (RV).

Methods: In this cross-sectional study, 45 healthy subjects and 95 HF patients exhibiting left ventricular systolic dysfunction on echocardiographic examination (EF <45%) secondary to ischemic (n=56) or idiopathic dilated cardiomyopathy (DCM) (n=39) were enrolled. Patients with severe renal dysfunction and/or valve disease were excluded. CS was measured by echocardiography from the posterior atrioventricular groove in the apical four-chamber view. The RV myocardial performance index (MPI), which reflects both systolic and diastolic function of the ventricle, was detected using tissue Doppler imaging, and patients with an RV MPI >0.55 were defined as having impaired RV myocardial function. ANOVA, Kruskal-Wallis, Pearson's correlation, and multivariate logistic regression analyses were used for the statistical analysis.

Results: The CS and RV MPI values were significantly greater both in patients with ischemic and idiopathic DCM than in controls (8.79±1.7 mm and 8.33±2.1 mm vs. 5.74±0.6 mm, and 0.64±0.07 and 0.62±0.08 vs. 0.43±0.02; p<0.001 for both, respectively). For the prediction of HF patients with impaired RV function, the cut-off value for the diameter of the CS was 7.35 mm, with a sensitivity of 83% and a specificity of 79%.

Conclusion: The CS diameter can be used as a novel echocardiographic marker that provides information about impaired RV function in patients with HF. *(Anatol J Cardiol 2015; 15: 542-7)*

Keywords: coronary sinus, right ventricle, myocardial performance index, heart failure

Introduction

The venous coronary sinus (CS) is a tubular structure, 2-3 cm in length, located 1 cm from the atrial side of the atrioventricular junction, that transmits venous blood to the right atrium and that can be visualized from multiple echocardiographic views (1). A review of the literature revealed few studies examining the association between the diameter of the CS and heart disease. In an autopsy study, Potkin et al. (1) reported that the diameter of the CS was larger in patients with poor ventricular function. Additionally, other studies have found a strong association between CS diameter and pulmonary artery pressure (PAP) in patients with primary pulmonary hypertension and the severity of mitral stenosis in patients with rheumatic mitral valve disease (2, 3). Therefore, a recent cross-sectional study indicated that the increase in CS diameter in patients with both ischemic heart failure (HF) and CS dilatation might be part of the cardiac remodeling process (4). However, this study reported that the CS diameter was not correlated with PAP. According to these data, it is unclear whether the underlying cause of CS dilatation is secondary to pulmonary hypertension or a part of cardiac remodeling in patients with HF.

Chronic HF exhibiting left ventricular (LV) systolic dysfunction causes several adaptive changes in the right heart chamber due to pressure and volume overload. Right ventricle (RV) and atrium dilatation develops to allow compensatory preload and contributes to stroke volume (5). However, there are no reports describing the relationship between CS diameter, a part of the right chamber, and right ventricular (RV) myocardial function in patients exhibiting LV systolic dysfunction (LVSD) secondary to ischemic or idiopathic dilated cardiomyopathy (DCM). Thus, in the present cross-sectional study, we aimed to assess the rela-

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tionship between coronary sinus anatomic alterations and the global myocardial function of the RV in patients with ischemic and idiopathic DCM.

Methods

Study population

We enrolled 45 age- and sex matched subjects with normal echocardiographic examination as a control group (Group 1) and 95 patients with heart failure who were diagnosed for at least 2 years with LVSD secondary to ischemic or idiopathic dilated cardiomyopathy. Idiopathic DCM was defined by the presence of both a left ventricular ejection fraction (LVEF) <45% (as revealed by echocardiography) and a dilated LV cavity in the absence of coronary artery stenosis >50% (as determined by coronary angiography), valvular heart disease, secondary cardiac muscle disease attributable to any known systemic condition, and histories of acute viral myocarditis. Ischemic DCM was defined by the presence of prior myocardial infarction, history of coronary artery bypass grafting, presence of Q waves on electrocardiogram, LV regional wall motional abnormalities conforming to a typical coronary distribution on echocardiogram, and/or ≥70% luminal stenosis in any major epicardial coronary artery diagnosed by coronary angiography. A total of 56 of the HF patients consisted of ischemic cardiomyopathy (Group 2), and a total of 39 of the HF patients consisted of idiopathic DCM (Group 3). To be included in the study, patients with HF needed to fulfill the following criteria: (i) left ventricle ejection fraction (LVEF) <45% and (ii) optimized oral therapy for the treatment of HF, including the use of angiotensin-converting enzyme inhibitors or angiotensin receptor antagonists, beta-blockers, and intermittent diuretics. Exclusion criteria were as follows: patients with renal dysfunction (serum creatinine >1.2 mg/dL), cirrhotic liver disease, hypothyroidism, atrial fibrillation or flutter, severe mitral regurgitation or tricuspid regurgitation, aortic stenosis, a history of aortic or mitral valve operation, atrioventricular conduction abnormalities, and a diagnosis of persistent left superior vena cava. Additionally, patients with decompensated HF who had one of the following criteria were excluded from the study: LVSD with bilateral inspiratory rales; edema; and heart failure findings on the chest X-ray, including pulmonary venous congestion and/or pleural effusions. On the same day, all patients underwent a transthoracic echocardiography and sampling of peripheral venous blood for laboratory measurements. The study protocol was approved by our local ethical committee, and all patients gave informed consent before participation in the study.

Transthoracic echocardiography

A comprehensive echocardiographic examination, including M-mode, two-dimensional, and Doppler echocardiography, was performed in all subjects by two experienced physicians without any knowledge of the biochemical and clinical data in accordance with the combined ASE/ESC guidelines (6, 7) using a Vivid E9 (GE Vingmed Ultrasound AS, Horten, Norway). Left ventricle (LV) ejection fraction was estimated from the apical four- and two-chamber views using the Simpson's biplane method. The LV diameters were measured from the parasternal long-axis view. Left atrium diameter was measured from the apical 4-chamber view at the end of ventricular systole from the free wall of the left atrium to interatrial septum. Pulsed-wave Doppler of the mitral or tricuspid inflow and pulsed-wave tissue Doppler of the mitral or tricuspid annulus were obtained from the apical 4-chamber view with a 5-mm sample volume. Mitral or tricuspid inflow E-waves and early myocardial relaxation velocities (Ea) were then used to calculate the E/Ea ratio, which was used as an indicator of left and right ventricle filling pressure. The systolic pulmonary artery pressure (PAP) was derived from the tricuspid regurgitant jet velocity with the modified Bernoulli equation. The RV area was determined by tracing the RV endocardium at diastole from the annulus to the apex along the free wall and then back to the annulus along the interventricular septum. The right atrial (RA) area was traced at the end of ventricular systole from the lateral side of the tricuspid annulus to the septal side, discounting the area between the leaflets and annulus, following the RA endocardium, excluding the inferior and superior vena cava and RA appendage. The RV myocardial performance index (MPI) was obtained using tissue Doppler echocardiography (TDE). TDE was performed in the apical four-chamber view using a 5- to 10-mm sample volume placed on the tricuspid annulus lateral wall. The isovolumetric relaxation time (IRT) was measured from the end of the systolic velocity to the start of the early diastolic velocity (Ea). The isovolumetric contraction time (ICT) was measured from the end of the late diastolic velocity to the start of the systolic velocity. Ejection time (ET) was calculated as the duration of the systolic velocity. RV MPI was calculated using the equation (ICT+IRT)/ET. The intraobserver and interobserver variability was 3.6% and 4.0%, respectively. The reported upper reference limit of the RV MPI is 0.55 by TDE in healthy subjects (6). HF patients with an RV MPI >0.55 were defined as having impaired RV myocardial function. The CS diameter was measured from the posterior atrioventricular groove in an apical four-chamber echocardiographic view during ventricular systole, because the maximum diameter of the CS occurs during ventricular systole. CS diameter measurements were obtained from the following three areas: at the termination of the CS orifice (prox), 1 cm from the left side (mid), and between the orifice and origin on the left side (dist). The mean CS diameter was calculated as follows: mean CS=(prox CS+mid CS+dist CS)/3 (Fig. 1) (2). The intraobserver and interobserver variability was 3.2% and 4.1%, respectively.

Statistical analysis

Statistical analysis was performed with SPSS 21.0 for Windows (SPSS Inc.). Continuous data were presented as mean±SD, and categorical data were expressed as numbers and percentage. The Kolmogorov-Smirnov test was used to evaluate whether the distribution of continuous variables was normal. One-way analysis of variance (ANOVA) with posthoc Tukey test

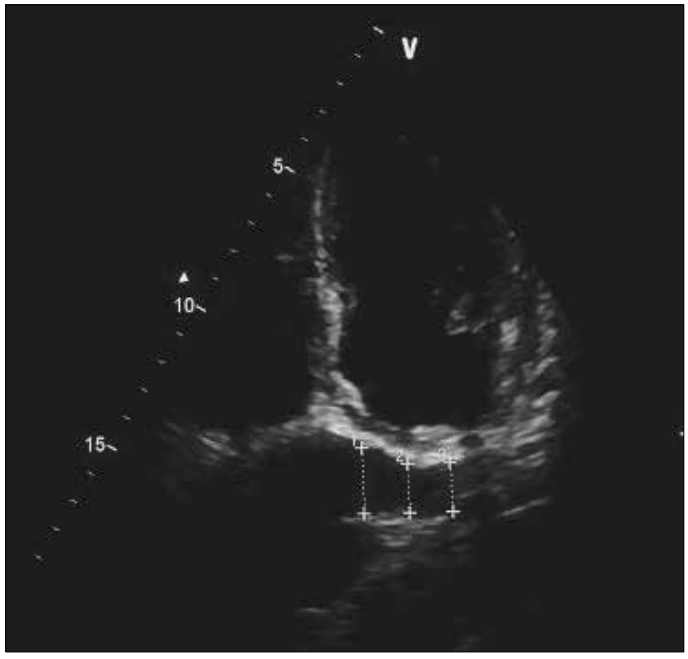

Figure 1. Echocardiographic measurements of coronary sinus are shown

or Kruskal-Wallis with posthoc Sidak test was used to compare the three groups. Categorical variables were summarized as percentages and compared with the chi-square test. Correlation analysis was performed using Spearman or Pearson test. Multivariate linear regression analysis was performed in order to determine the predictive factors for CS from variables showing significance values <0.1 in the correlation analysis. In consideration of the wide range and of the non-skewed distribution, left ventricular EF was log-transformed to reduce the effects of extreme values and to obtain a normal distribution for statistical tests. The accuracy of CS value to predict HF patients with RV MPI >0.55 mm was assessed by receiver operating characteristics curve (ROC) analysis. A p value <0.05 was considered statistically significant.

Results

The clinical and echocardiographic characteristics of the three groups are presented in Table 1. There was no significant difference between the groups (Groups 1, 2, and 3) in terms of age, gender, diabetes mellitus, hypertension, body mass index, and smoking (p>0.05). As shown in Table 1, all of the patients were stable in terms of their chronic HF status, with 13 (23.2%) ischemic HF patients in NYHA class I, 10 (17.8%) in class II, 19 (33.9%) in class III, and 4 (7.1%) in class IV and with 7 (17.9%) idiopathic DCM patients in NYHA class I, 19 (48.7%) in class II, 12 (30.7%) in class III, and 2 (5.1%) in class IV. Furthermore, the echocardiographic characteristics were similar between patients with ischemic DCM (Group 2) and with idiopathic DCM (Group 3). The diameter of CS was significantly greater in both Group 2 and Group 3 than in Group 1 (8.79±1.7 mm and 8.33±2.1 mm vs. 5.74±0.6 mm; p <0.001 for both). The MPI measured in the RV using TDE is presented in Table 1. The RV MPI was significantly higher in both Group 2 and Group 3 than in the control group (0.64±0.07 and 0.62±0.08 vs. 0.43±0.02; p<0.001 for both). Therefore, the RA area was significantly greater in both Group 2 and Group 3 than in the control group (20.4±2.6 cm^2 and 19.8±3.0 cm^2 vs. 14.9±2.1 cm^2; p<0.001 for both). The echocardiographic variables that were significantly correlated with the diameter of the CS are presented in Table 2. A strong correlation was found between the mean CS diameters and RV MPI, RA area, and systolic PAP in patients with HF (r=0.687, r=608, and r=0.481; p for each variable <0.001). Those variables showing significant correlations according to Table 2 were included in a multivariate linear regression analysis. According to our results, RV MPI (β=0.550, 95% CI: 6.623-15.506, p<0.001), RA area (β=0.232, 95% CI: 0.016-0.194, p=0.021), RV E/Ea ratio (β=0.130, 95% CI: 0.013-0.255, p=0.030), and LV E/Ea ratio (β=0.138, 95% CI: 0.018-0.174, p=0.017) were independent predictors of the CS diameter in patients with HF (Table 3). Patients with HF (n=95) were categorized into two groups with respect to the top and bottom 0.55 of their RV MPI. The group with RV MPI >0.55 mm (n=72) was defined to be HF patients with impaired RV myocardial functions. For the prediction of HF patients with RV myocardial dysfunction, the cut-off value for the diameter of the CS was 7.35 mm, with a sensitivity of 83% and a specificity of 79% (AUC=0.839, 95% CI=0.731-0.946, p<0.001) (Fig. 2).

Discussion

In the current study, we found that the CS diameter was significantly increased in patients with both ischemic and idiopathic DCM. Additionally, a dilated CS was strongly associated with RV echocardiographic characteristics, including the RV MPI, RA area, systolic PAP, and RV E/Ea ratio. There was a moderate correlation between CS diameter and echocardiographic characteristics of the left ventricle, including the end-systolic and-diastolic diameters, left atrial diameters, and EF value. Furthermore, RV MPI, RA area, RV E/Ea ratio, and LV E/Ea ratio were identified as independent predictors of CS diameter in patients with HF.

The CS is a tubular structure that transmits venous blood to the right atrium. The CS contracts simultaneously with the atria, because its wall contains atrial myocardium (8). Thus, the diameter of the CS changes at different phases of the cardiac cycle, peaking at the end of ventricular systole and becoming smallest during atrial contraction (9). For this reason, in our study, the diameter of the CS was measured at the end of ventricular systole. A dilated CS can result from increased blood flow due to abnormal venous drainage in the left superior vena cava, total anomalous intra-cardiac pulmonary venous drainage, severe tricuspid regurgitation, CS diverticulum, or a coronary artery to CS fistula. The absence of primary abnormalities is generally a manifestation of high RA pressure due to functional tricuspid regurgitation (10-12). Koberstein et al. (13) showed that CS dilatation with RA blood reflux to the CS was due to increased RA

Table 1. General characteristics and echocardiographic findings of the groups

				P values for Tukey test			*P* values for ANOVA
	Group 1 (n=45)	Group 2 (n=56)	Group 3 (n=39)	Group 1-2	Group 1-3	Group 2-3	
Age, years	54.4±6.7	56.5±7.0	53.4±7.1	-	-	-	0.089
Female gender, n (%)*	23 (51.1)	30 (53.6)	20 (51.3)	-	-	-	0.896
Hypertension, n (%)*	6 (13.3)	13 (23.2)	6 (15.4)	-	-	-	0.389
Diabetes mellitus, n (%)*	5 (11.1)	9 (16.1)	3 (7.7)	-	-	-	0.454
Smoking, n (%)*	11 (24.4)	18 (32.1)	7 (17.9)	-	-	-	0.289
Body mass index, kg/m^2	26.4±3.0	26.9±2.7	25.8±2.7	-	-	-	0.206
NYHA class (I/II/III/IV), n*	-	13/10/19/4	7/19/12/2	-	-	-	0.443
Creatinine, mg/dL	0.65±0.11	0.69±0.14	0.70±0.12	-	-	-	0.299
Hemoglobin, g/dL	13.5±1.47	13.5±1.44	13.3±1.48	-	-	-	0.271
Left ventricular parameters							
LV ejection fraction (%)**	65 (60-70)	33 (20-43)	34 (18-43)	<0.001	<0.001	0.677	<0.001
LV end-diastolic diameter, mm	44.0±3.1	53.8±4.2	55.7±5.2	<0.001	<0.001	0.074	<0.001
LV end-systolic diameter, mm	29.0±3.0	44.4±5.2	46.1±5.6	<0.001	<0.001	0.236	<0.001
IVS thickness, mm**	10 (8-16)	11 (8-14)	10 (8-14)	-	-	-	0.152
LV E/Ea ratio	7.4±2.1	10.3±2.8	9.4±3.2	<0.001	0.004	0.285	<0.001
LA_{max} diameter, mm	35.2±2.4	43.7±3.7	45.0±4.0	<0.001	<0.001	0.209	<0.001
Right ventricular parameters							
Systolic PAP, mm Hg	27.3±3.1	41.8±6.7	40.4±7.8	<0.001	<0.001	0.523	<0.001
RV myocardial performance index	0.43±0.02 0.42 (0.37-0.48)	0.64±0.07 0.64 (0.50-0.83)	0.62±0.08 0.62 (0.50-0.80)	<0.001	<0.001	0.239	<0.001
ICT (msec)	55.4±6.4	77.1±8.7	75.5±9.7	<0.001	<0.001	0.627	<0.001
IRT (msec)	57.6±3.1	72.4±7.7	71.1±8.8	<0.001	<0.001	0.659	<0.001
ET (msec)	260±15	234±19	237±18	<0.001	<0.001	0.570	<0.001
RV Sa wave, cm/s	13.6±1.3	8.5±2.7	9.7±2.8	<0.001	<0.001	0.099	<0.001
RV E/Ea ratio	1.72±0.46	3.70±2.58	3.32±1.64	<0.001	<0.001	0.597	<0.001
RV end-diastolic area, cm^2	20.8±3.2	28.2±4.0	27.3±4.1	<0.001	<0.001	0.485	<0.001
RA_{max} area, cm^2	14.9±2.1	20.4±2.6	19.8±3.0	<0.001	<0.001	0.550	<0.001
Mean CS diameter, mm	5.74±0.6	8.79±1.7	8.33±2.1	<0.001	<0.001	0.371	<0.001

Continuous variables were expressed as mean±standard deviation or median (min-max), and categorical variables were expressed as number of cases and percentage. One-way analysis of variance (ANOVA) with posthoc Tukey test was used for comparison
*The chi-square test was used to analyze categorical variables. **The Kruskal-Wallis test with posthoc Sidak test was used for the analysis of nonparametric variables
Group 1- Control, Group 2-Patients with ischemic cardiomyopathy, Group 3- Patients with idiopathic dilated cardiomyopathy.
CS - coronary sinus; ET - ejection time; ICT - isovolumetric contraction time; IRT - isovolumetric relaxation time; IVS - interventricular septum; LA - left atrium; LV - left ventricle; LV E/Ea ratio - mitral Doppler inflow E wave velocity-to-annular tissue Doppler Ea wave velocity ratio; NYHA - New York Heart Association; PAP - pulmonary artery pressure; RA - right atrium; RV - right ventricle; RV E/Ea ratio - tricuspid Doppler inflow velocity-to-annular tissue Doppler Ea wave velocity ratio; Sa - annular tissue Doppler systolic velocity

pressure. They also found that 15% of the blood sampled from the CS originated from the RA in dogs with elevated RA pressure caused by partial obstruction of the pulmonary artery. In comparison, Mahmud et al. (14) reported that the size of the CS was significantly correlated with the size of the right atrium (r=0.60, p<0.001) and RA pressure (r=0.59, p<0.001) but not with the size of the right ventricle in patients who underwent right heart catheterization for the evaluation of pulmonary hypertension. In addition, a dilated right atrium is a sign of increased RA pressure. Machraouni et al. (15) reported a strong correlation between RA pressure and the RA area index (r=0.64). However, this is a qualitative measure that does not allow the interpreter to assess RA pressure (6). Also, in our study, CS diameter was positively correlated with the RA area, and therefore, the RA area was found as an independent predictor of CS dilatation.

RV systolic and diastolic dysfunction is common in patients with HF and is associated with a poor long-term prognosis (16-19). The MPI, which is also known as the Tei index, has been described as a non-invasive measurement of ventricular function. The TDE-derived MPI reflects both the systolic and dia-

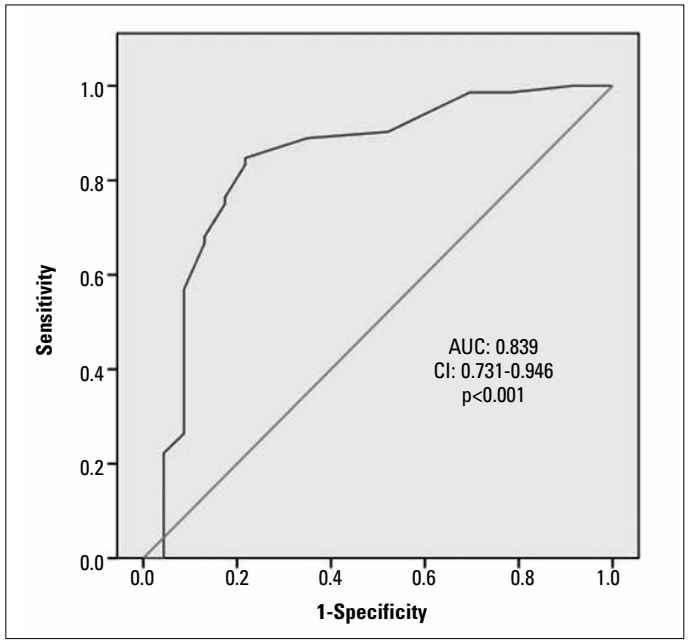

Figure 2. The cut-off value of the coronary sinus diameter for prediction of heart failure patients with impaired right ventricular myocardial function was 7.35 mm, with a sensitivity of 83% and a specificity of 79% in the ROC curve analysis AUC=0.839, 95% CI=0.731-0.946, p<0.001).
AUC - area under curve; ROC - receiver operating characteristics curve analysis

stolic function of the ventricles, defined as the ratio of the isovolumic time divided by the ET, or [(IRT + ICT)/ET] (20). The RV MPI represents subclinical RV dysfunction and volume overload in patients with congenital heart disease (21) and is increased in patients with primary pulmonary hypertension, RV infarction, and hypertrophic cardiomyopathy (22-24). In accordance with these findings, in our study, we found that the RV MPI was significantly increased in patients with idiopathic and ischemic DCM compared with normal controls. Furthermore, there was a positive and significant correlation between RV MPI and CS diameter in these patients. Similarly, Vatankulu et al. (25) reported that the size of the CS was significantly associated with increased RV MPI in patients with mitral stenosis.

Cardiac remodeling is the result of hemodynamic changes in patients with LV systolic dysfunction. The left atrial pressure and diameter increase due to pressure and volume overload in the left ventricle in these patients. An elevated left atrial pressure leads to passive elevation in the pulmonary arterial pressure, which leads to impaired functioning of the right side of the heart through overloading of the right ventricular and atrial pressures (5, 26, 27). Additionally, RV function impairs secondary to myocardial infarction or ischemia due to a (proximal) right and/or left anterior descending coronary artery lesion in HF patients (6). In our study, CS diameter was found to be negatively correlated with LV ejection fraction and positively correlated with LV E/Ea ratio, LA diameter, PAP, RV MPI, RV E/Ea ratio, and RA area. However, there were relatively more powerful correlations between the CS diameter and the parameters of RV rather than those of LV. Furthermore, the RV MPI, RA area, and RV E/Ea ratio with LV E/Ea ratio were found as independent predictors of CS dilatation. In light of these data, we conclude that the diameter of the CS may increase owing to increased RA pressure due to impaired RV function, depending on the mechanisms mentioned above in patients with ischemic and idiopathic DCM.

Table 2. The association of coronary sinus diameter with echocardiographic parameters in patients with heart failure

	r value	*P*
RV myocardial performance index*	0.687	<0.001
RA_{max} area, cm^2*	0.608	<0.001
Systolic PAP, mm Hg*	0.481	<0.001
RV end-diastolic area, cm^2*	0.276	0.007
RV E/Ea ratio*	0.408	<0.001
RV Sa wave, cm/s*	-0.383	<0.001
LV ejection fraction (%)**	-0.360	<0.001
LV end-diastolic diameter, mm*	0.251	0.014
LV end-systolic diameter, mm*	0.286	0.005
LV E/Ea ratio*	0.380	<0.001
LA_{max} diameter, mm*	0.365	<0.001

*Pearson's test, **Spearman test. LA - left atrium; LV - left ventricle; LV E/Ea ratio - mitral Doppler inflow E wave velocity-to-annular tissue Doppler Ea wave velocity ratio; PAP - pulmonary artery pressure; RA - right atrium; RV - right ventricle; RV E/Ea ratio - tricuspid Doppler inflow velocity-to-annular tissue Doppler Ea wave velocity ratio

Table 3. Independent predictors of coronary sinus diameter

	β	t	95% confidence interval	*P*
RV MPI	0.550	4.929	6.623-15.506	<0.001
RA area	0.232	2.340	0.016-0.194	0.021
RV E/Ea ratio	0.130	2.190	0.013-0.255	0.030
LV E/Ea ratio	0.138	2.423	0.018-0.174	0.017

*Multivariate linear regression analysis.
LV E/Ea ratio - mitral Doppler inflow E wave velocity-to-annular tissue Doppler Ea wave velocity ratio, MPI - right ventricular myocardial performance index; RA - right atrium; RV E/Ea ratio - tricuspid Doppler inflow velocity-to-annular tissue Doppler Ea wave velocity ratio

Study limitations

The first limitation is the relatively small number of patients; subjects were selected from patients with chronic compensated LVSD. Second, invasive measurements of RV parameters were not performed together with RV MPI measurements; however, most of our patients had higher systolic PAP. Third, contrast injection was not used for measurement of the coronary sinus. Fourth, the cross-sectional design limited our ability to determine the significance of changes in the diameter of the CS for the long-term prognosis and mortality of patients with LVSD.

Conclusion

Our results confirm our hypothesis that the diameter of the CS increases, owing to increased RA pressure due to impaired RV functions, and it can be used as a novel echocardiographic

marker to provide information about impaired RV function in patients with ischemic and idiopathic DCM. However, further study will be necessary to confirm these preliminary findings.

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