Early Diastolic Mitral Regurgitation in Left Ventricular Aneurysm

Atsuko Uema, MD[1], Yuma Tamura, PhD[2], Tokuhisa Uejima, MD, PhD[3], Megumi Hoshiai, MD[1], Asuka Ueno, MD[1], Moeko Nagao, MD[1], Takashi Tomoe, MD[1], Shoya Ono, MD[1], Eikou Maeno, MD[1], Satoshi Mizuguchi, MD[1], Atsuhiko Kawabe, MD[1], Takushi Sugiyama, MD[1], Takanori Yasu, MD, PhD[1]

[1] Department of Cardiovascular Medicine and Nephrology, Dokkyo Medical University Nikko Medical Center, Nikko, Tochigi, Japan

[2] Department of Rehabilitation, Dokkyo Medical University Nikko Medical Center, Nikko, Tochigi, Japan

[3] Department of Cardiovascular Medicine, The Cardiovascular Institute, Nishiazabu, Minato-ku, Tokyo, Japan

Corresponding author

Dr. Takanori Yasu

Department of Cardiovascular Medicine and Nephrology, Dokkyo Medical University

Nikko Medical Center

632 Takatoku, Nikko, Tochigi 321-2593, Japan

Tel.: 81-288-76-1515; Fax: 81-288-76-1030
E-mail: tyasu@dokkyomed.ac.jp

Short title

Early Diastolic MR in LV Aneurysm

Author contributions

Abstract

Diastolic mitral regurgitation is a type of functional mitral regurgitation that develops via a reversal of the left atrioventricular pressure gradient during diastole. This study aimed to explore the mechanism underlying early diastolic mitral regurgitation (EDMR) in patients with left ventricular (LV) aneurysms after anterior myocardial infarction (AMI) by assessing the intraventricular pressure difference using vector flow mapping. We enrolled 23 consecutive patients with LV aneurysms (with and without EDMR) and 15 healthy men as controls. In the control group, LV suction began from the apex during early diastole. In contrast, the blood that pooled in the apical aneurysm during systole generated a relatively higher pressure at the apex than at the basal LV during early diastole; consequently, the pressure reversal phenomenon occurred in the LV. Compared to the EDMR– group, the EDMR+ group (n=7) exhibited a significantly higher diastolic time ratio ([time from the second heart sound to the pressure inversion point]/[total diastolic time]) ($P<0.001$). The diastolic time ratio was significantly correlated with log BNP, but not with E/A, E/E', or the left atrial expansion index. In conclusion, EDMR in LV aneurysm may be due to a prolonged diastolic time ratio leading to prolonged pressure inversion in the LV during early diastole.

Keywords

Diastolic mitral regurgitation, Intraventricular pressure difference, Left ventricular aneurysm, Myocardial infarction, Vector flow mapping

Introduction

Diastolic mitral regurgitation (MR) is a type of functional MR that develops via various mechanisms and causes a reversal of the left atrioventricular pressure gradient during diastole [1]. Mid-late diastolic MR has been reported in patients with severe aortic regurgitation (AR) and an atrioventricular block [1-7]. AR-associated mid-late diastolic MR results from a considerable elevation of the left ventricular (LV) end-diastolic pressure secondary to an excessive blood flow from the aorta into the LV [1,2,7].

LV aneurysm is a serious mechanical complication of acute myocardial infarction (AMI). The low survival rate of patients with LV aneurysm is related to the infarct size, LV remodeling, and a reduced LV ejection fraction (LVEF) [8]. Thus, LV aneurysm also causes an increase in the LV end-diastolic pressure. Early diastolic MR (EDMR) has a low velocity due to a relatively low diastolic LV-left atrial (LA) pressure gradient, and consequently, is sometimes difficult to detect. Thus, reports of EDMR secondary to LV dysfunction are limited [7,9,10]; to the best of our knowledge, we are the first to report four cases of concurrent EDMR and LV aneurysm after MI[11] 「~~ (data submitted)~~. Therefore, the mechanism underlying EDMR in cases with LV aneurysms remains unclear.

Recently, Murayama et al. [10] reported a case with ischemic cardiomyopathy without aneurysm that was diagnosed using color M-mode echocardiography and vector flow mapping (VFM); in this case, vortexes on VFM revealed EDMR. Therefore, the present study aimed to explore the mechanism underlying EDMR development in patients with LV aneurysms by using VFM to assess the intraventricular pressure difference (IVPD) [12-15] during early diastole.

Methods

Study design and subjects

Twenty-three consecutive patients with LV aneurysm after anterior MI who underwent echocardiography at our institute from January 2016 to July 2021 were enrolled in this study as the LV aneurysm group (7 patients were further categorized into the LV aneurysm group with EDMR, while 16 were categorized into the LV aneurysm group without EDMR). Four patients were hospitalized due to the first anterior AMI, while the remaining nineteen patients were outpatients with anterior occlusion myocardial infarction. Fifteen healthy men aged 31±10 years were included as controls. The exclusion criteria were moderate or severe AR, atrioventricular block, ventricular pacing, and atrial fibrillation. This study had a single-center, cross-sectional, and longitudinal study design. We analyzed the IVPD occurring during early diastole in the patients and controls. For all subjects, echocardiography was performed using an ALOKA ARIETTA 850 ultrasound machine (Fuji film Ltd., Tokyo, Japan) with an S121 probe at a transducer frequency of 1–5 MHz, depth of 7–9 cm, and frame rate of 40–60 frames/s. A phonocardiogram was used to detect the second heart sound (S2). The LVEF was measured using the biplane disk summation technique and other standard systolic and diastolic parameters including trans-mitral flow (TMF) and tissue Doppler imaging (TDI). All LA volume measurements were calculated from apical 4- and 2-chamber views using the biplane area-length method. The LA expansion index was calculated using Hsiao's method [16]. Apical long-axis color Doppler images were obtained for VFM, and the IVPD was analyzed in detail during early diastole with a frame-by-frame method using a computer equipped with the DAS-RS1 software (Hitachi Ltd. Tokyo, Japan).

Image analysis

Relative pressure imaging is a novel, noninvasive method that uses VFM to assess the intracardiac pressure distribution [12-15, 17,18]. The intraventricular flow velocity vectors of VFM are calculated by solving the continuity equation using the flow velocity obtained from color Doppler and the wall velocity obtained from speckle tracking echocardiography[12-15, 17,18]. The relative pressure imaging method converts this velocity information to relative pressure distribution using the momentum equations of fluid motion (Navier–Stokes equations) [12,14,17]. Patients with LV aneurysm lost apical suction during early diastole, and the relative LV internal pressure was higher at the apical aneurysm than at the LV base and LA during very early diastole (**Figure 1a**). With basal LV relaxation, the LV basal pressure became lower than the LA pressure. Consequently, the pressure inversion point (PIP) appeared in the LV aneurysm group, leading the E wave (**Figure 1a**). The diastolic time (DT) ratio was calculated as follows (**Figure 1b**): (time from S2 until the PIP)/(total DT).

Ethics approval

This study was conducted in accordance with the principles outlined in the Declaration of Helsinki and with the ethics guidelines for clinical research from the Ministry of Health, Labour and Welfare (Tokyo, Japan). Informed consent was obtained from the patients through an opt-out system, and those who refused to provide consent were excluded. The study protocol was reviewed and approved by the institutional ethics committee of the Dokkyo Medical University Nikko Medical Center (approval number: Nikko 29006).

Statistical analysis

Continuous variables are presented as means ± standard deviations (SD), whereas categorical variables are expressed as numbers and percentages. Statistical comparisons were conducted using the Student's *t*-test, repeated analysis of variance, chi-squared test, and Fisher's exact test, as appropriate. The Pearson's correlation coefficients between the DT ratio (i.e., S2-PIP/DT) and the other parameters (E/A, E/E', log BNP, and LA expansion index) were assessed. Data were statistically analyzed using JMP-J version 14.0 (SAS Institute, Cary, NC, USA) and SPSS version 25 (IBM Corp., Armonk, NY, USA). The significance of a two-tailed P-value was set at <5%.

Results

The baseline characteristics of the 23 patients with LV aneurysms after anterior AMI are presented in **Table 1.** Color M-mode revealed EDMR in seven out of these 23 patients. The LV aneurysm group with EDMR had significantly lower LVEFs and higher serum brain natriuretic peptide (BNP) levels than the LV aneurysm group without EDMR (**Table 2)**.

Results of the VFM analysis of the IVPD during early diastole are presented in **Figures 2 and 3**. In the controls, suction began at the LV apex during early diastole; subsequently, the pressure at the LV apex decreased, thereby creating an IVPD (**Figure 2A–E**). Conversely, in the LV aneurysm group (with/without EDMR), apical suction was lost during early diastole and the relative LV internal pressure was higher at the apical aneurysm than at the basal LV. Thus, the pressure reversal phenomenon (LV pressure > LA pressure) was observed in the LV aneurysm group during early diastole (**Figure 2F–Q**). The IVPD during the isovolumetric relaxation period indicated an intra-LV pressure disparity, while the relative pressure disparity after mitral valve opening slightly indicated a pressure disparity between the LV and the LA. The DT ratios (i.e., S2-PIP/DT) were 0.38±0.18 and 0.14±0.03 in the LV aneurysm groups with and without EDMR, respectively (P<0.001, **Table 2, Figure 1C**). **Figure 3** shows the real-time serial color Doppler images, VFM images, and relative pressure difference between the LV apex and LA through the basal LV in a patient with concurrent LV aneurysm and EDMR. There was no MR at mid-systole (**Figure 3A1**), while mild MR was noted at end-systole. In early diastole, the pressure at the LV apex was relatively higher than that at the LV base and LA due to a lack of suction at the apex, and a slow blood flow from the apex to the LA through the LV base (**Figure 3B2,3–F2,3**) resulting in EDMR (**Figure 3B1–F1**) was confirmed. This phenomenon continued until the PIP appeared (**Figure 3F**) and the E-wave of the LV inflow (**Figure 3G**) appeared after the PIP. We also investigated the correlations between the DT ratio (obtained from the VFM data) and the standard parameters of systolic and diastolic function (such as E/A, E/E’, log BNP, and the LA expansion index) (**Figure 5**). Only log BNP had a significant correlation with the DT ratio (R= 0.46, P=0.034).

Discussion

The DT ratio was significantly higher in the LV aneurysm group with EDMR than in the LV aneurysm group without EDMR. Furthermore, it was significantly correlated with log BNP, but not with E/A, E/E’, or the LA expansion index.

The accuracy of real-time IVPD assessment by VFM has been validated previously [14-16). In this study, we carefully assessed the IVPD during early diastole by employing the frame-by-frame method of VFM to explore the mechanism underlying EDMR development in patients with LV aneurysms. Diastolic reversal of the left atrioventricular pressure gradient supplies a functional diastolic MR [1]. The mechanisms and clinical significance of late-diastolic MR secondary to AR or atrioventricular block are well recognized [1-7,18,19]; however, those of EDMR secondary to AR or atrioventricular block remain unclear. An apical aneurysm pools blood passively during systole and contracts passively during early diastole. Our findings indicated that a prolonged relatively higher pressure in the LV apex than in the LV base during early diastole, which leads to reversal of the left atrioventricular pressure gradient and to mitral valve opening by spreading the papillary muscle, may contribute to EDMR. Murayama et al. [10] showed that in ischemic cardiomyopathy accompanied by EDMR, a clockwise vortex existed under the anterior mitral leaflet during the cardiac cycle, whereas a counterclockwise vortex occurred just under the aortic valve during early diastole, thus generating blood flow toward the anterior mitral leaflet from the boundary of these vortices [10]. Furthermore, they reported that the early diastolic pressure was slightly higher at the apex than at the LV base, which was suggestive of a loss of LV suction [10]. Their findings are consistent with our data. Additionally, we also observed a good correlation between the DT ratio and log BNP; this indicates the potential of the DT ratio as a surrogate marker of heart failure severity in patients with LV aneurysm. However, this must be verified in a further study with a greater sample size.

In our very recent case series report (n=4), all patients with concurrent EDMR and LV aneurysm following MI presented with a low LVEF (<33%) and died of heart failure within six months following echocardiography [11]. The TMF in all the four patients revealed E-waves with lower velocities (two showed no E-waves). Despite the high pulmonary artery wedge pressure (PAWP; >25 mm Hg) and heart rate (<90 beats/mins), they also presented with end-diastolic A-waves of higher velocities than that of the E-waves and an impaired relaxation pattern. In the present study, five out of the seven patients with EDMR underwent right side cardiac catheterization, and the PAWP in all patients was ≥18 mmHg despite an impaired relaxation pattern. We believe that our study makes a significant contribution to the literature, because our findings indicate that EDMR due to prolonged pressure inversion in the LV might be associated with an impaired relaxation pattern (E/A<1) in patients with an LV aneurysm, despite a high PAWP. A possible explanation for this may be the relatively high early diastolic LV pressure, particularly in an aneurysm. Shen et al. [8] reported that out of 71 patients with anterior AMI, 10 with aneurysm and 3 without aneurysm died at a mean follow-up of 53 months. Their stepwise multivariate analysis revealed that the LVEF and obstruction status of the left anterior descending artery and collaterals were independent predictors of mortality in patients with an aneurysm. Furthermore, they concluded that the reduced survival rate in patients with an aneurysm was primarily related to severe global LV dysfunction, which may be determined by assessing the residual flow to the infarct region [8].

Our study has some limitations. First, the number of study patients was limited.

Second, due to ethical reasons, we did not measure the LV pressure and PAWP simultaneously during cardiac catheterization in Study 2 to avoid embolic complications.

In conclusion, EDMR in LV aneurysm may be attributable to a prolonged DT ratio in the LV during the early diastolic phase. Estimating the LV end-diastolic pressure by mitral inflow pattern may be difficult, particularly in cases with an LV aneurysm associated with EDMR. Further large-scale studies are required to determine whether the DT ratio or EDMR can predict adverse clinical outcomes in cases with LV aneurysms and/or ischemic cardiomyopathy.

Conflict of interest

Dr. Uejima has received grant support from Hitachi Ltd. Dr. Yasu has received grant support from Abbott Medical Japan LLC., AstraZeneca K.K., Ono Pharmaceutical Co., Ltd., Kowa Co. Ltd., and MTG Co. Ltd. The other authors declare that they have no conflict of interest.

Acknowledgments

The authors thank Prof. Satoshi Nakatani for his critical advice and Keiko Yoshizawa for her administrative assistance. We would also like to thank *Editage* (www.editage.com) for English language editing.

Declarations

Ethics approval

This study was approved by our institutional ethics committee (approval number: Nikko 29012). All procedures performed in this study complied with the national ethical guidelines for medical and health research involving human participants and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Consent to participate

Consent to publish

Funding

This work was supported by a grant from the Vehicle Racing Commemorative Foundation (to T.Y.) and by Dokkyo Medical University, Young Investigator Award (No. 2017–21) (to A.U.). Part of this paper was presented at the annual meeting of the American Heart Association in Philadelphia in November 2019.

Data availability

The deidentified participant data will not be shared.

References

1. Nagueh SF, Smiseth OA, Appleton CP, Byrd BF, Dokainish H, Edvardsen T, Flachskampf FA, Gillebert TC, Klein AL, Lancellotti P, Marino P, Oh JK, Alexandru Popescu B, Waggoner AD (2016) 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. Eur Heart J Cardiovasc Imaging 17:1321–1360
2. Agmon Y, Freeman WK, Oh JK, Seward JB (1999) Diastolic mitral regurgitation. Circulation 99:e13
3. Alessandri N, Mariani S, Messina FR, Rondoni G, Gerbasi E, Battista L, Gaudio C (2003) Diastolic mitral regurgitation: a borderline case in cardiovascular physiology. Eur Rev Med Pharmacol Sci 7:161–170
4. Konka M, Kusmierczyk-Droszcz B, Wozniak O, Hoffman P (2008) Aortic regurgitation and unusual diastolic mitral regurgitation. Eur J Echocardiogr 9:709–711
5. Schnittger I, Appleton CP, Hatle LK, Popp RL (1988) Diastolic mitral and tricuspid regurgitation by Doppler echocardiography in patients with atrioventricular block: new insight into the mechanism of atrioventricular valve closure. J Am Coll Cardiol

11:83–88
6. Berger RL, Katz E, Tunick P, Kronzon I (2008) The 'A-dip' of diastolic mitral regurgitation: an unusual Doppler flow pattern in a patient with severe aortic insufficiency and complete heart block. Eur J Echocardiogr 9:69–71
7. Sisu RC, Vinereanu D (2011) Different mechanisms for diastolic mitral regurgitation illustrated by three comparative cases. Echocardiography 28:476–479
8. Shen WF, Tribouilloy C, Mirode A, Dufossé H, Lesbre JP (1992) Left ventricular aneurysm and prognosis in patients with first acute transmural anterior myocardial infarction and isolated left anterior descending artery disease. Eur Heart J 13:39–44
9. Agrawal A, Parikh M, Verma I, Thyagarajan B (2016) Diastolic mitral regurgitation in a patient with coronary artery disease and anaemia. BMJ Case Rep 2016:bcr2016214473
10. Murayama M, Iwano H, Sarashina M, Anzai T (2020) Mechanism of early-diastolic mitral regurgitation. Circ J 84:2036
11. Uema A, Sugiyama T, Yasu T**.** A case series of patients with early diastolic mitral regurgitation and left ventricular aneurysm. Clin Cardiol J 2021;5(5):1-3.
12. Minami S, Masuda K, Stugaard M, Kamimukai T, Asanuma T, Nakatani S (2021) Noninvasive assessment of intraventricular pressure difference in left ventricular

dyssynchrony using vector flow mapping. Heart Vessels 36:92–98

13. Yotti R, Bermejo J, Benito Y, Antoranz JC, Desco MM, Rodríguez-Pérez D, Cortina C, Mombiela T, Barrio A, Elízaga J, Fernández-Avilés F (2011) Noninvasive estimation of the rate of relaxation by the analysis of intraventricular pressure gradients. Circ Cardiovasc Imaging 4:94–104
14. Tanaka T, Okada T, Nishiyama T, Seki Y (2017) Relative pressure imaging in left ventricle using ultrasonic vector flow mapping. Jpn J Appl Phys 56:07JF26
15. Itatani K, Okada T, Uejima T, Tanaka T, Ono M, Miyaji K, Takenaka K (2013) Intraventricular flow velocity vector visualization based on the continuity equation and measurements of vorticity and wall shear stress. Jpn J Appl Phys 52:7–16
16. Hsiao SH, Chiou KR (2013) Left atrial expansion index predicts atrial fibrillation in dyspnea. Circ J 77:2712–2721
17. Asami R, Tanaka T, Kawabata KI, Hashiba K, Okada T, Nishiyama T (2017) Accuracy and limitations of vector flow mapping: left ventricular phantom validation using stereo particle image velocimetory. J Echocardiogr 15:57–66
18. Nagueh SF, Bhatt R, Vivo RP, Krim SR, Sarvari SI, Russell K, Edvardsen T, Smiseth OA, Estep JD (2011) Echocardiographic evaluation of hemodynamics in patients with decompensated systolic heart failure. Circ Cardiovasc Imaging 4:220–

227

19. Dokainish H, Nguyen JS, Bobek J, Goswami R, Lakkis NM (2011) Assessment of the American Society of Echocardiography-European Association of Echocardiography guidelines for diastolic function in patients with depressed ejection fraction: an echocardiographic and invasive haemodynamic study. Eur J Echocardiogr 12:857–864

Figure Legends

Figure 1. The pressure inversion point (PIP) during the early diastolic phase is determined using the frame-by-frame method. (A) The relative pressure between the apical aneurysm and the left atrium through the basal left ventricle (LV) is inversed at PIP. (B) The diastolic time ratio is calculated as follows: (time from the second heart sound [S2] to the PIP)/(total diastolic time). (C) Histogram of the diastolic time ratio shows that the diastolic time ratios of all aneurysmal patients without early diastolic mitral regurgitation (EDMR; n=10) are lower than 0.2; however, five out of the six aneurysmal patients with EDMR show a ratio higher than 0.2. Data of one patient without EDMR and one patient with EDMR were unfortunately lost.

Figure 2. The vector flow mapping (VFM) results of three representative cases, one from each group. Real-time serial intra-c relative pressure difference during early diastole in the healthy control group (A–E), LV aneurysm group without early diastolic mitral regurgitation (EDMR; F–K), and LV aneurysm group with EDMR (L–R). (B–E) In the control group, suction begins from the apex. (H, N–P**)** Consequently, the pressure at the LV apex is relatively lower than that at the basal LV during early diastole. In contrast, the apical aneurysm passively dilates during systole and passively contracts

during very early diastole. The diastolic time (DT) ratio is calculated as follows: (time from the second heart sound [S2] to the pressure inversion point)/(total DT). The pressure reversal phenomenon is noted to occur at a DT ratio of 0.15 in the LV aneurysm group without EDMR (Q) and at a ratio of 0.45 in the LV aneurysm group with EDMR (I).

Figure 3. Real-time serial color Doppler images (A1–G1). Vector flow mapping images and relative pressure difference between the left ventricular (LV) apex and the left atrium (LA) through the basal LV in a patient with concurrent LV aneurysm and early diastolic mitral regurgitation (EDMR; A2,3–G2,3). No mitral regurgitation is noted at mid-systole (A1). The pressure at the LV apex is relatively higher than the pressure at the LV base and LA due to a lack of suction at the apex. A slow blood flow from the apex to the LA through the LV base is confirmed (B2,3–F2,3). This phenomenon is noted to continue until the pressure inversion point (PIP; F1–3), and the E-wave of the LV inflow (G1–3) is noted to appear after the PIP.

Figure 4. Histogram of the diastolic time ratio shows that the diastolic time ratios of all aneurysmal patients without early diastolic mitral regurgitation (EDMR; n=15) are

lower than 0.2; however, five out of the six patients with EDMR have diastolic time ratios higher than 0.2. Data of one patient without EDMR and one patient with EDMR were unfortunately lost.

Figure 5. Correlation between the vector mapping flow parameters and the E/A, TDI, log BNP, and LA expansion index (LAEI). The DT ratio is noted to have a good correlation with log BNP (R= 0.46, P=0.034), but not with E/A, E/E', and LAEI.

Figure 6. Pressure Inversion Point (PIP) during the early diastolic phase was determined using the frame-by-frame method. The apical aneurysm passively dilates during systole and passively contracts during the very early diastole. Thus, the pressure at the left ventricular (LV) apex is relatively higher than that at the basal LV during the early diastolic period. In patients with LV aneurysms and early diastolic mitral regurgitation (EDMR), the relative pressure at the LV apex is higher than at the left atrium, and blood flow from the apex to the base is confirmed. This phenomenon continues until the E-wave of the LV inflow appears at PIP.

Table 2a. Baseline clinical characteristics of the patients with left ventricular aneurysm

Variables	All patients (n=23)	EDMR (–) (n=16)	EDMR (+) (n=7)
Mean age (years)	72±10	68±9	79±8
Male, n (%)	11 (55%)	8 (62%)	3 (43%)
SBP (mm Hg)	119±14	121±12	114±16
DBP (mm Hg)	68±13	71±9	63±15
Heart rate (beats/min)	76±16	71±10	86±20
Hypertension	13 (65%)	10 (77%)	3 (43%)
Dyslipidemia	15 (75%)	11 (85%)	4 (57%)
Diabetes mellitus	10 (50%)	6 (46%)	4 (57%)
Chronic kidney disease	11 (55%)	6 (46%)	5 (71%)
Number of diseased coronary vessels	1.6 Vessels	1.5 Vessels	1.7 Vessels
Antiplatelet drugs	16 (80%)	12 (92%)	4 (57%)
HMG-CoA reductase inhibitor usage	18 (90%)	12 (92%)	6 (86%)

Beta-blocker usage	13 (65%)	10 (77%)	3 (43%)
Calcium channel blocker usage	2 (10%)	2 (15%)	0
Anticoagulant drug usage	12 (60%)	7 (54%)	5 (71%)
ACE inhibitor or ARB usage	13 (65%)	9 (69%)	4 (57%)
Diuretic usage	13 (65%)	7 (54%)	6 (86%)

ACE: angiotensin-converting enzyme; ARB: angiotensin II receptor blocker; DBP: diastolic blood pressure; EDMR: early diastolic mitral regurgitation; SBP: systolic blood pressure.

Table 2b. Echocardiographic data and serum brain natriuretic peptide levels of the patients

Variables	EDMR (–) (n=16)	EDMR (+) (n=7)	P value
Mean age (years)	68±9	79±9	0.017
Male, n (%)	8 (57)	3 (43)	0.31
LVDd (mm)	51±6	53±10	0.48
LVDs (mm)	39±6	42±10	0.36
LVEDVI (mL)	92±26	109±34	0.22
LVESVI (mL)	53±25	74±30	0.11
LVEF (%)	46±13	34±10	0.048
LAVI (mL)	32±11	38.8±12	0.24
E/A	0.76±0.35	0.80±0.21	0.73
BNP	109±108	1182±1335	
Log BNP	1.87±0.39	2.43±1.12	0.0005
(S2-PIP)/DT	0.12±0.0.2	0.31±0.11	0.0008

BNP: brain natriuretic peptide; EDMR: early diastolic mitral regurgitation; LAVI: left atrial volume index; LVDd: left ventricular end-diastolic diameter; LVDS: left ventricular end-systolic diameter; LVEDVI: left ventricular end-diastolic volume index; LVEF: left ventricular ejection fraction; LVESVI: left ventricular end-systolic volume index; S2-PIP/DT: (second sound to pressure inversion point)/diastolic time.