Evaluation of diastolic wall strain in patients with mitral valve regurgitation

Mükremin Coşkun[1], Mert Evlice[2], İbrahim Halil Kurt[2], Mehmet Akif Düzenli[1]

1 Department of Cardiology, Meram School of Medicine, Necmettin Erbakan University, Konya, Türkiye
2 Department of Cardiology, Adana City Training and Research Hospital, Adana, Türkiye

Abstract

Aim: Diastolic wall strain (DWS) is a noninvasive, easy, echocardiographic parameter that has been used to determine left ventricular diastolic stiffness. The relationship between left ventricle functions and diastolic wall strain is previously revealed but we don't know if this method correlates with mitral regurgitation (MR) severity or not. In this study, we sought to determine the relationship between DWS and MR.

Methods: This study included 107 subjects with primary mitral regurgitation, divided in two groups as mild-to-moderate and severe mitral regurgitation, and 54 control subjects. We calculated the DWS from the M-mode echocardiographic measurements of the LV posterior wall thickness at end-systole and end-diastole during sinus rhythm.

Results: MR group had lower values of DWS than control subjects and severe MR group had the lowest ones (0.38 ± 0.06; 0.27 ± 0.07; 0.15 ± 0.035 respectively and p <0.001). DWS was negatively correlated with the disease severity and had the largest size effect (r: -0.851 p<0.001, $\eta_p^2 = 0.701$).

Conclusions: DWS is a qualitative and easy method. It could be used for determining the severity of MR

Keywords: *Diastolic wall strain; left ventricular diastolic stiffness, mitral regurgitation, primary mitral regurgitation*

1. Introduction

The incidence of valve diseases increases with age. The prevalence is under 2% in the ages before 65 and it is 13.2% after the age of 75.[1] Increased prevalence of valve diseases with age is together with the changes in the etiology. Especially in developed countries degenerative causes are the first reason while rheumatic valve diseases are still common in developing countries.

Mitral regurgitation (MR) and multiple valve involvement are the most common valve diseases in our country.[2] MR can be primary (organic) or secondary (functional). Primary MR develops as a result of anatomical disorder of mitral apparatus and numerous anatomical lesion may cause this. Secondary or functional regurgitation is almost due to myocardial diseases and mitral valves are structural as normal.[3-4]

MR can be asymptomatic for a long time. When LV function is impaired symptoms may occur, most commonly seen as exertional dyspnea. Also orthopnea, paroxysmal nocturnal dyspnea, fatigue as a result of reduced cardiac output and decreased exercise capacity can be seen.[5]

Corresponding Author: Mert Evlice, mertevlice@hotmail.com, Received: 21.08.2024, Accepted: 21.09.2024, Available Online Date: 28.09.2024 Cite this article as: Coskun M, Evlice M, Kurt IH, Duzenli MA. Evaluation of diastolic wall strain in patients with mitral valve regurgitation. J Cukurova Anesth Surg. 2024; 7(3): 186-90. https://doi.org/10.36516/jocass.1535317

Echocardiography is the gold standard in diagnosis. Grading of MR is complex and difficult. Doppler echocardiography with clinical examination is the most important method in diagnosis. Rating of MR jet with eye- only results in error and is not recommended. Quantitative parameters such as vena contract width, regurgitant orifice area, insufficiency volume and fraction have prognostic significance and are recommended for patients with MR.[6] However, it is not always possible to use these quantitative parameters in daily practice in clinics where there are few patients. Also, differences in measurement between clinicians limit their usability.

LV diastolic wall strain (DWS) is an echocardiographic index which gives information about LV stiffness in preserved LV systolic functions. It is independent of loading conditions and noninvasive.[7] There are studies showing that impaired DWS and increased LV stiffness play an important role in the pathophysiology of both preserved and low ejection fraction heart failure and are associated with the prevalence of atrial fibrillation in normal heart. [8] The possible usefulness of DWS in patients with MR has not been reported previously. The aim of our study was to investigate whether DWS could be used for classification of MR.

2. Materials and methods

One hundred sixty-one consecutive patients; 54 controls and 107 with MR; were included in the study. We excluded patients who had other severe valvular disease, end stage renal disease, stroke,

insufficient quality of echocardiography, reduced LV ejection fraction or presence of wall motion abnormalities. These patients were admitted to Meram Medical Faculty Hospital Cardiology outpatient clinic for any cardiovascular symptoms. The Meram Medical Faculty Hospital approved the study protocol and this study was performed in accordance with the Declaration of Helsinki.

Doppler, 2-dimensional, M-mode echocardiograms were acquired using standardized acquisition protocol. LV interventricular septum thickness, LV posterior wall (PW) thickness, LV end-diastolic and end-systolic dimensions were determined from M-mode and ejection fraction was calculated by the Teichholz method.

Left atrial (LA) diameter was determined from the M-mode echocardiography as the largest distance between the posterior aortic wall and posterior wall of LA at end-systole. [9] Mitral inflow velocities, peak early E and peak late A, were acquired in the apical four-chamber view. DWS was calculated using the formula: LV DWS = (LVPWs - LVPWd) / LVPWs,, where LVPWs is the LVPW thickness at end-systole and LVPWd is the LVPW thickness at end-diastole using M-mode (figure 1).[10] In patients with MR detected by color doppler echocardiography, two different cardiologists performed visual grading and patients were grouped as mild or moderate and severe MR.

2.1. Statistical Analysis

Continuous variables were expressed as mean ± standard deviation and categorical variables as count and percentage. Oneway Anova test was used for analysis of continuous variables, and the difference between nominal variables were compared with chi-square testing. Multiple comparisons of the means of variables were performed using the LSD post hoc test. A multivariate analysis was used to adjust for the clinical and echocardiographic variables potentially affecting the MR classification and partial eta squared (η_p^2) was used to n-measure the effect size of these variables. Statistical significance was accepted at <0,05. Statistical analysis were performed with the SPSS version 16 (SPSS, Chicago, IL, USA).

3. Results

Table 1 shows clinical and demographic characteristics of the study cohort. There were no differences in age, gender, prevalence of diabetes mellitus, hypertension, hyperlipidemia, family history of heart disease and smoking between the 3 groups. However, the prevalence of atrial fibrillation was higher in MR group as expected and the difference was statistically significant (p<0.001).

Table 2 shows echocardiographic data. Patients in 3 groups had a similar LV systolic function assessed by LV ejection fraction. LV end-diastolic, end-systolic diameters and left atrial diameter were larger in MR group and correlated with the severity. There was an increase in posterior wall end diastolic thickness with severity of MR while LV posterior wall end systolic thickness decreased. When the doppler echocardiographic indices representing LV diastolic function were compared, both E/E' septal and E/E' lateral were higher in MR group and E/A was lower in MR, the difference was statistically significant in all parameters. Although, echocardiographic measurements showed significant differences among three groups, the effect size of DWS was higher than the other parameters (p<0.001, η_p^2= 0.701). DWS correlated well with E' septal , E' lateral , E/E' septal and E/E' lateral values and LA diameter (p=0.004 R= 0.228, p<0.001 R=0.275, p<0.001 R= -0.369, p<0.001 R= -0.380, p<0.001 R=-0.655 respectively). We could not find any correlation between E/A and DWS (p=0.097).

MR severity did not correlate with comorbidities like systolic and diastolic blood pressure values, hypertension, diabetes mellitus, dislipidemia, smoking or family history. There was a weak significant correlation between most echocardiographic parameters except LV ejection fraction and mitral inflow E/A ratio and MR. The strongest correlation was between MR and LV DWS (r = -0.851, p <0.001) (Table 3). DWS decreased as MR severity increased.

Table 1

Basic demographic and clinical data according to study groups

Variables	Control group (n=54)	Mild-moderate MR group (n=53)	Severe MR group (n=54)	P
Age, years	60 ± 9	57 ± 10	56 ± 10	0.448
Female, n (%)	35 (65)	37 (70)	30 (56)	0.299
Hypertension, n (%)	30 (56)	31 (58)	29 (54)	0.881
Systolic Blood Pressure, mm Hg	123 ± 15	126 ± 14	120 ± 12	0.153
Diastolic Blood Pressure, mm Hg	74 ± 9	75 ± 10	74 ± 9	0.892
Diyabetes Mellitus, n (%)	17 (31)	15 (28)	29 (54)	0.901
Hyperlipidemia, n (%)	15 (54)	11 (21)	14 (26)	0.685
Family history, n (%)	20 (37)	18 (34)	17 (31)	0.830
Smoking, n (%)	14 (26)	7 (13)	8 (15)	0.174
Atrial Fibrilation, n (%)	0 (0)	7 (13)	18 (33)	<0.001*

*Notes: LV, left ventricular; MR, mitral regurgitation; * The difference was statistically significant (p < 0.001).*

Table 2

LV diastolic wall strain and baseline echocardiographic measurements according to study groups

Variables	Control group (n=54)	Mild-moderate MR group (n=53)	Severe MR group (n=54)	P	η_p^2
LV diastolic diameter, mm	45.2 ± 4.5	48.2 ± 4.6	53.3 ± 5.7	<0.001*	0.307
LV systolic diameter mm	26.2 ± 3.6	28.5 ± 5.4	34.9 ± 6.7	<0.001*	0.313
LV diastolic posterior wall thickness, mm	9.4 ± 1.0	9.9 ± 1.2	10.7 ± 1.4	<0.001*	0.151
LV diastolic posterior wall thickness, mm	15.3 ± 1.7	13.7 ± 1.7	12.5 ± 1.5	<0.001*	0.323
LV ejection fraction, %	59.1 ± 7.7	60.0 ± 4.0	58.5 ± 2.7	0.328	0.009
Left atrium diameter, mm	31.59 ± 4.00	40.01 ± 6.13	58.70 ± 6.88	0.002**	0.514
E velocity, m/s	75.85±16.11	75.00 ± 17.21	95.70 ± 23.06	<0.001*	0.246
A velocity, m/s	69.14 ± 16.26	75.76± 20.27	78.62 ± 18.66	0.043**	0.065
E / A ratio	1.29 ± 0.40	1.14 ± 0.32	0.98 ± 0.26	<0.001*	0.118
E /E' (septal)	7.56 ± 2.55	10.56 ± 4.61	12.76 ± 4.32	<0.001*	0.273
E /E' (lateral)	6.53 ± 2.15	8.52 ± 3.87	11.22 ± 4.06	<0.001*	0.241
LV diastolic wall strain	0.376 ± 0.063	0.272 ± 0.069	0.148 ± 0.035	<0.001*	0.701

*Notes: LV, left ventricular; MR, mitral regurgitation. * The difference was statistically significant (p < 0.001). ** The difference was statistically significant (p < 0.05).*

Table 3

Correlation of LV diastolic wall stress with baseline demographic, clinical, and echocardiographic data in the study population

	r value	p value
Basic demographic and clinical data		
• Hypertension	-0.015	0.848
• Diabetes mellitus	-0.033	0.674
• Hyperlipidemia	-0.018	0.825
• Systolic blood pressure	-0.087	0.273
• Diastolic blood pressure	-0.015	0.848
• Smoking	-0.118	0.135
• Family history	-0.048	0.546
Echocardiographic parameters		
• Early mitral inflow velocity, E (m/s)	0.384	<0.001*
• Late mitral inflow velocity, A (m/s)	0.246	0.004**
• E/A ratio	0.134	0.121
• E/e' lateral	0.487	<0.001*
• E/e' septal	0.480	<0.001*
• LV ejection fraction (%)	0.070	0.381
• Left atrium diameter, mm	0.269	0.001*
• LV diastolic diameter, mm	0,269	0,001**
• LV systolic diameter, mm	0.544	<0.001*
• LV systolic posterior wall, mm	-0.566	<0.001*
• LV diastolic posterior wall, mm	0.382	<0.001*
• LV diastolic wall strain	-0.851	<0.001*

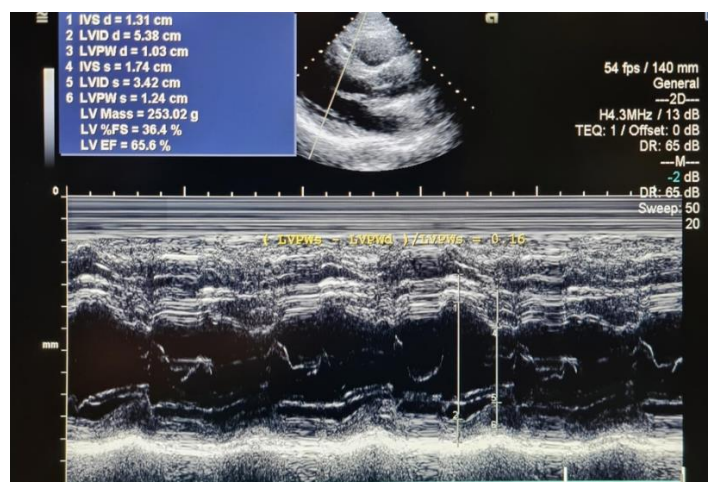
*Notes: LV, left ventricular; MR, mitral regurgitation. * The difference was statistically significant (p < 0.001). ** The difference was statistically significant (p < 0.05).*

Box plot graph of LV DWS (0.376 ± 0.063 vs. 0.272 ± 0.069 vs. 0.148 ± 0.035; p <0.001* ηp^2=0.701) values in healthy control group and patients with mild-mode MR and severe MR groups, respectively, is shown in Figure 2. LV DWS values were shown to be significantly decreased in patients with severe MR (Figure 2). DWS decreases as the severity of MR increases.

A significant correlation was found between DWS and severity of MR (r=-0.851; p<0.001) (Table 2). ROC curve analysis found that a DWS lower than 0.28 was associated with the presence of MR , with a sensitivity of 86 % and a specificity of 90 % (Area under the ROC curve=0.952; 95% CI 0.924–0.981; p<0.001) (Figure 3). ROC curve analysis found that a DWS lower than 0.2 was associated with severe MR , with a sensitivity of 95 % and a specificity of 94 % (Area under the ROC curve=0.966; 95% CI 0.939–0.993; p<0.001) (Figure 3).

Figure 1

Calculation of left ventricular diastolic (LVPWd) and systolic (LVPWs) posterior wall thickness and diastolic wall strain (DWS) by M-mode echocardiography. LV DWS = (LVPWs - LVPWd) / LVPWs



4. Discussion

To the best of our knowledge, this is the first report to investigate the usability of DWS in classification of MR. The main findings can be summarized as follows. First, we demonstrated that patients with MR had lower DWS levels than controls. Second, in severe MR the lowest DWS values were found. Third, DWS had the highest

Figure 2

Box plot graphic showing the relationship with LV diastolic wall strain in patients with mild-moderate MR, severe MR, and control subjects. Notes: MR, mitral regurgitation; LV, left ventricular.

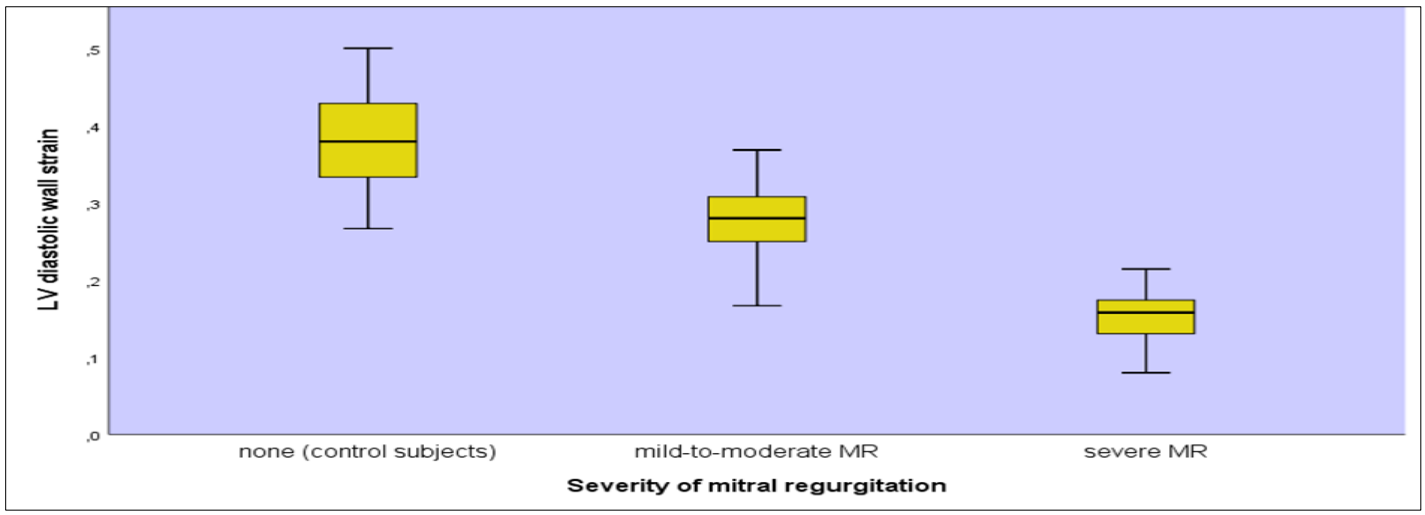
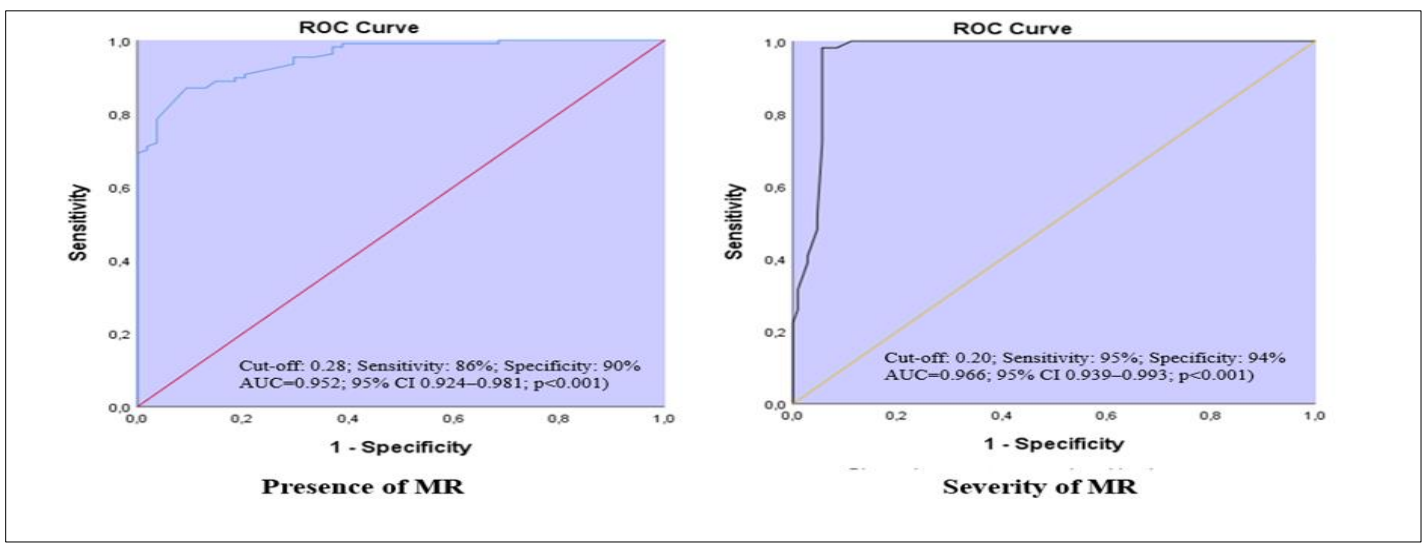


Figure 3

ROC curve analysis showing the relationship of left ventricular DWS to the presence and severity of MR. Notes: MR, mitral regurgitation; LV, left ventricular.



effect size on classification of MR and the best correlation coefficient with MR. These findings suggest that DWS, an easy measurement from 2-D transthoracic echocardiography, may be used for determining MR severity.

Takeda et al. reported that DWS is an easy and noninvasive echocardiographic measurement that indicates increased LV stiffness and correlates well with invasive methods in animal models.[11] Lower DWS values predict subtle diastolic dysfunction in preserved ejection fraction heart failure and have poor prognosis. There have been no studies using DWS value for classification of patients with MR.

LV diastolic dysfunction is common and relevant with the heart failure and increased mortality. MR negatively affects the relaxation of the LV. On account of this, quantitation of MR is more important for clinicians and evaluating MR patients include multidisciplinary approach like symptoms, exercise capacity, echocardiographic measurements and arrhythmia risk. MR constitutes a volume load on LV and increases afterload directing the LV to decompensation. LV stiffness increased and diastolic dysfunction occurs firstly. Echocardiographic measurements, which are used to evaluate MR in daily practice, are performed on 2-dimensional, continue waves doppler, pulse waves doppler and color doppler echocardiography. Some of them are quantitative and some are qualitative. The measurements in these two groups should be used together when deciding on the severity of MR.

In our study, the strain average of the control group was 37 %, the strain average of patients with mild-to-moderate MR was 26 %, and the strain average of patients with severe MR was 14 %. Strain cut value for MR is 0.20; 95% sensitivity and 94% specificity. The measured strain value was found to be effective in high range without

predicting the intensity of MR. There is a negative correlation between MR grade and diastolic strain and a decrease in the amount of residual cardiac strain in MR severity. Based on our analysis, we found that DWS was correlated well with tissue E' velocity and E/e' ratio. Nagueh et. al showed that the e' velocity measured with tissue Doppler echocardiography is sensitive for abnormal LV relaxation.[12] Takagi and colleagues reported that lower DWS was associated with increased LV stiffness and correlated with E/E' [13] and Ohtani et. al have confirmed the correlation between DWS and tissue doppler E' velocity. These previous studies encourage our findings. As a result, posterior DWS is as effective and simpler as the new clinical and echocardiographic parameters. The relationship between 3 groups and DWS is shown in Figure 2. DWS decreases as the severity of mitral regurgitation increases.

Our study has few limitations. First, this is a single center and a small group of participants study. Second, strain was only measured from 2-dimensional transthoracic echocardiography. Futrhermore, the patients with MR were grouped by only using qualitative methods.

5. Conclusion

DWS, easily calculated from 2-dimensional echocardiography, correlates well with other echocardiographic parameters and could be a determinator for MR classification. Further prospective larger group studies are needed.

Statement of ethics

The present study protocol was reviewed and approved by Meram Medical Faculty Hospital Ethics Committee ((56-2017)).

Source of Finance

During this study, no financial or spiritual support was received neither from any pharmaceutical company that has a direct connection with the research subject, nor from a company that provides or produces medical instruments and materials which may negatively affect the evaluation process of this study.

Conflict of interest statement

No conflicts of interest between the authors and/or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, shareholding, and similar situations in any firm. Mert Evlice[2], MD; İbrahim Halil Kurt[2], MD;

Availability of data and materials

The data supporting this study's findings are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

Author contributions

Idea/Concept/Design: Mükremin Coşkun[1, 2], MD; Mehmet Akif Düzenli[1], MD; Control/Supervision: Mert Evlice[2], MD; Mehmet Akif Düzenli[1], MD; Data Collection and/or Processing: Mükremin Coşkun[1, 2], MD; Mehmet Akif Düzenli[1], MD; Analysis and/or Interpretation: Mükremin Coşkun[1, 2], MD; Mehmet Akif Düzenli[1], MD; Mert Evlice[2], MD; Literature Review: Mükremin Coşkun[1, 2], MD; Mert Evlice[2], MD; İbrahim Halil Kurt[2], MD; Mehmet Akif Düzenli[1], MD; Writing the Article: Mükremin Coşkun[1, 2], MD; Mert Evlice[2], MD; Critical Review: Mert Evlice[2], MD; İbrahim Halil Kurt[2], MD; References: Mert Evlice[2], MD; Mükremin Coşkun[1, 2], MD; Materials: Mükremin Coşkun[1, 2], MD;

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