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The comparison of mitral annular plane systolic excursion (MAPSE) and mitral annular systolic velocity (Sm) in determining subclinical left ventricular systolic dysfunction in patients with type 2 diabetes mellitus

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ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is an independent factor in increasing the risk of heart failure in the absence of coronary heart disease and hypertension. Global longitudinal strain (GLS) as the gold standard in determining subclinical left ventricular (LV) systolic dysfunction is not available on all echocardiographic tools and requires good-quality images. Mitral annular plane systolic excursion (MAPSE) and mitral annular peak systolic velocity (Sm) are simpler, faster, and widely available method that can determine left ventricular systolic dysfunction regardless of image quality.

Methods: This study involved 72 asymptomatic T2DM patients, divided into two groups, patients with subclinical left ventricular systolic dysfunction (GLS >-18%) and normal systolic function (GLS ≤-18%). GLS was obtained from the mean of 18 left ventricular segments on the apical 4-chamber, 3-chamber and 2-chamber images. MAPSE was obtained on the septal and lateral sides of the mitral annulus using M-mode on apical 4-chamber view, while Sm was obtained using tissue doppler imaging (TDI).

Results: The study included 72 asymptomatic T2DM patients, 34 samples (47.2%) were found with subclinical LV systolic dysfunction. According to receiver operating characteristic (ROC) curve analysis, lateral TDI Sm had the highest area under the curve (AUC), it was 0.85, followed by average TDI Sm was 0.83 and average MAPSE was 0.81. The cut-off value of average TDI Sm <7.425 cm/s had the best sensitivity and specificity, 82.4% and 81.6%, while cut-off value of average MAPSE <13.4 mm had sensitivity of 76.5% and specificity of 73.7%.

Conclusion: TDI Sm had better accuracy than MAPSE in determining subclinical LV systolic dysfunction in T2DM patients. However, both of them can be used as alternative diagnostic methods of GLS.

Keywords: mitral, systolic, doppler, imaging.

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INTRODUCTION

The incidence type 2 diabetes mellitus (T2DM) can increase the risk of cardiovascular disease and death. T2DM is an independent factor in increasing the risk of heart failure despite in the absence of coronary heart disease and hypertension.[1] The Framingham study found an epidemiological relationship between diabetes and heart failure.[2] The risk of heart failure increases 2.4 times in men and 5 times in women. Factors associated with the occurrence of heart failure in diabetic patients are age, duration of diabetes, insulin use, ischemic heart disease, peripheral arterial disease, elevated serum creatinine, uncontrolled blood sugar levels, and microalbuminuria.[3]

Early subclinical left ventricular (LV) systolic dysfunction can be measured using global longitudinal strain (GLS) with 2-dimensional speckle tracking echocardiography (2D STE), even the LV ejection fraction (EF) is still normal (preserved) in individuals with asymptomatic T2DM.[4] However, this method requires special software which is not available in all echocardiographic machines. In addition, good quality imaging is needed to accurately determine the LV systolic function.[5]

There are other simpler, faster, and wider available methods that can determine LV systolic dysfunction in early stages. Mitral annular plane systolic excursion (MAPSE) can measure the

movement of the mitral annulus towards the apex in the systolic phase, resulting in a longitudinal shortening of the left ventricle which plays an important role in the pumping function of the heart. MAPSE has been proposed as a clinically useful echocardiographic parameter for the assessment of longitudinal function and correlates with global systolic function of the LV.[5] Measurement of the peak systolic velocity of the mitral annulus (Sm) with tissue doppler imaging (TDI) is also an easy method to determine LV systolic dysfunction. This method measured longitudinal movement of the mitral annulus, which is a good non-invasive examination for global LV systolic function.[6] MAPSE and TDI Sm can be measured in most patients regardless of image quality.

This study aims to compare the accuracy MAPSE and TDI Sm in determining subclinical LV systolic dysfunction based on GLS in asymptomatic T2DM patients. Therefore, this study is expected to provide information regarding simple diagnostic methods that have better accuracy in determining subclinical LV systolic dysfunction to optimize patient management.

METHODS

Study Populations

This study included 77 asymptomatic patients with T2DM. We excluded patients with (1) LVEF <50%; (2) ischemic heart disease; (3) history of heart failure or have previous symptoms of heart failure; (4) significant valvular abnormalities or congenital heart disease; (5) significant heart rhythm disturbance; (6) history of chemotherapy; (7) chest wall abnormalities; and (8) poor echo window in apical views with 2 or more segments not well visualized. Two patients were initially excluded due to decreased LVEF and three were excluded due to poor echo window. The Ethics committee approved the study protocol of Prof I.G.N.G Ngoerah Hospital/Faculty of Medicine, Udayana University and all patient gave informed consent before participation.

Data collection

The study was conducted in Echo Lab Prof I.G.N.G Ngoerah Hospital, from February until June 2021. The research sample data is obtained from history taking, physical examination, medical record data, and laboratory results. All patients underwent full transthoracic echocardiography study using Philips Epiq 7c machine, and measurements were obtained in accordance with the current American Society of Echocardiography guidelines. Two-dimensional imaging examination was performed in the standard parasternal long axis view, parasternal short axis view, apical 4-, 3-, and 2-chamber view. The results of echocardiographic measurements were reviewed by Cardiologist Echocardiography Consultant who was blinded to the study samples.

Measurement of GLS Using 2D STE

Speckle tracking analysis was performed using 2D strain software (Philips QLAB 10.8). GLS was assessed by means of two-dimensional speckle-tracking strains from three standard apical views (4-, 3-, 2-chamber apical view). The region of interest was traced on the endocardium at end-systole with a point-and-click approach for each three apical views. Another larger region was then generated and manually adjusted near epicardium. Each apical images were divided into six segments and six time-strain curves were obtained. GLS was determined as the average peak strain of 18 segments from three standard apical views. GLS >-18% was defined as subclinical LV systolic dysfunction, while GLS ≤-18% was defined as normal.[1]

Measurement of MAPSE

MAPSE was measured from two atrioventricular sites, the septal and lateral, through the apical 4- chamber image using M-mode. Average MAPSE was calculated based on the sum of lateral dan septal MAPSE divided by two. The M-mode cursor has to be parallel to the LV wall. The mitral annulus systolic excursion was measured from the lowest point at end-diastolic to aortic valve closure (end of the T-wave on the electrocardiogram). The measurement did not include post-systolic motion toward the apex during periods of isovolumetric relaxation, which is associated with ischemia, fibrosis, or pressure overload.[7] Displacement of the mitral annulus was measured in milimeters (mm).[6]

Measurement of Mitral Annular Peak Systolic Velocity (Sm)

The TDI of the mitral annulus was obtained from an apical 4-chamber image, after excluding high frequency signals. The sample volume of 5 mm was placed at medial and lateral mitral annulus. Mitral annular peak velocity is positive systolic velocity when the mitral ring moves toward the apex. Average TDI Sm was calculated based on the sum of lateral and septal TDI Sm divided by two. This method was measured in cm/s and recorded for 3 cardiac cycles at a rate of 100 mm/s.[6]

Statistical analysis

Continuous variables were presented as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Between group with subclinical LV systolic function (GLS >-18%) and normal group (GLS ≤-18%) were compared by independent t-test for numerical variables dan by chi-square for categorical variables. The correlation between the index parameter (MAPSE dan TDI Sm) and GLS was analyzed by Pearson test and expressed as correlation coefficients. The AUC (Area Under the Curve) of each index parameter was by ROC (Receiver Operating Characteristic) curve. The cutoff value of the index parameters was determined based on the highest sensitivity and specificity. P-value of < 0.05 was considered statistically significant. Statistical analyses were performed using IBM Statistical Package for the Social Science (SPSS) Statistics version 23 (SPSS, Inc., Chicago, IL).

RESULTS

Baseline characteristics

The baseline clinical and laboratory characteristics of the 72 patients are summarized in Table 1. The patients are divided into two groups, group with subclinical LV systolic dysfunction (GLS >-18%) and normal LV systolic function (GLS ≤-18%). Patients with subclinical LV systolic dysfunction was observed 34 patients (47.2%) were observed, and

Table 1. Clinical and laboratory characteristics of patients.

Variable	Subclinical LV Dysfunction (n=34)	Normal LV Function (n= 38)	*p*
Clinical Data			
Age, years	57.1±8.6	54.1±9.5	0.16
Male, n (%)	13 (38.2)	18 (47.4)	0.44
Female, n (%)	21 (61.8)	20 (52.6)	
DM duration, months	94.1±72.5	60.8±55.5	0.03*
Smoking, n (%)	5 (14.7)	6 (15.8)	0.53
Dyslipidemia, n (%)	9 (26.5)	5 (13.2)	0.15
Hypertension, n (%)	21 (61.8)	21 (55.3)	0.58
History of stroke, n (%)	7 (20.6)	6 (15.8)	0.60
CKD, n (%)	8 (23.5)	4 (10.5)	0.14
Obesity/overweight, n (%)	12 (35.3)	11 (28.9)	0.09
BMI, kg/m2	24.8±5.0	24.6±3.1	0.83
Laboratory Examination			
Random BG, mg/dl	218.7±101.0	235.1±88.3	0.55
Fasting BG, mg/dl	155.2±61.5	142.0±50.4	0.47
2 hours PP BG mg/dl	232.9±73.1	210.3±116.2	0.55
HbA1c, %	8.6±2.3	8.2±2.3	0.58
Total cholesterol, mg/dl	171.4±69.1	184.5±45.6	0.55
LDL, mg/dl	96.0±46.7	109.7±36.7	0.38
HDL, mg/dl	31.31±14.2	40.9±8.7	0.03*
Triglyceride, mg/dl	216.6±179	153.8±88.5	0.23
Serum creatinine, mg/dl	1.5±1.4	1.2±0.7	0.21
eGFR, ml/min/1.73 m^2	61.1±29.0	71.9±25.9	0.10

*Significant (p<0.05); BG: blood glucose; BMI: body mass index; CKD: chronic kidney disease; DM: diabetes mellitus; eGFR: estimated glomerular filtration rate; HbA1c: glycosylated hemoglobin; HDL: high density lipoprotein; LDL: low-density lipoprotein; LV: left ventricle; PP: post prandial.

the remaining 38 (52.8%) had normal LV function. The mean age between two groups was not different, 57.12±8.6 years in subclinical LV dysfunction group and 54.08±9.53 years in normal group. The proportion of females was greater than male in both groups, however this proportion was not different between two groups. Patients with subclinical LV systolic dysfunction had longer mean DM duration compared to other group (94.1 ± 72.5 months vs 60.8 ± 55.5 months; p-value 0.03). The proportion of risk factors and comorbidities, including smoking, dyslipidemia, hypertension, history of stroke, chronic kidney disease (CKD), obesity/overweight were not different between both groups.

The mean HDL level of subclinical LV dysfunction group was significantly lower (31.3±14.2 mg/dl) than the other group (40.88 ± 8.68 mg/dl), with p-value 0.03. However, the two groups' mean blood glucose levels, HbA1c, total cholesterol, LDL, triglycerides, and serum creatinine were insignificantly different.

Echocardiographic characteristics

The echocardiographic characterictic of both groups are summarized in Table 2. Left ventricle mass index (LVMI) and left atrial volume index (LAVI) were significantly larger in subclinical LV dysfunction group. However, left ventricle volume index (LVVI), left ventricle ejection fraction (LVEF BP), and tricuspid annular plane systolic excursion (TAPSE) were not significantly different between two groups. In addition, diastolic parameters, including E/A ratio, E' (septal and lateral), E/E' (septal, lateral, and average) were significantly different. MAPSE (septal, lateral, and average) and TDI Sm (septal, lateral, and average) were significantly lower in subclinical LV systolic dysfunction group (p-value <0.001). The mean GLS in the subclinical LV dysfunction group was 14.15±2.11% and normal LV function group was 19.02±1.11% (p-value <0.001).

Correlation of MAPSE and TDI Sm with GLS

The correlation was obtained using Pearson test and expressed as correlation coefficient (r). It showed significant moderate negative correlations between septal MAPSE, lateral MAPSE, average MAPSE with GLS (r = -0.44, -0.43, -051, respectively; p value <0.001). A significant moderate negative correlations were also found between septal TDI Sm, lateral TDI Sm, average TDI Sm with GLS (r = -0.523, -0.569, -0.593, respectively; p value <0.001). The correlations between MAPSE and TDI Sm with GLS are summarized in Table 3.

Receiver Operating Characteristic (ROC) Curve Analysis and Cut-off Point

ROC curve analysis of MAPSE and TDI Sm in determining subclinical LV systolic dysfunction was shown in Figure 1. The area under the curve (AUC) of all index parameters was more than 0.7. Lateral TDI Sm had the highest AUC (0.85), followed by the average TDI Sm (0.830), and average MAPSE (0.809). These results were statistically significant with p < 0.001. AUC of the index parameters are listed in Table 4.

Cut-off point of each parameter was determined based on sensitivity and specificity, as shown in line diagram (Figure 2). Cut-off points of septal MAPSE, lateral MAPSE, and average MAPSE in determining subclinical LV systolic dysfunction were <12.45 mm, <14.45 mm, and <13.4 mm, respectively. Meanwhile, cut-off points for septal TDI Sm, lateral TDI Sm, and average TDI Sm were <7.125 cm/s, <8.04 cm/s, and <7.425 cm/s, respectively. TDI Sm average <7.425 cm/s had the highest sensitivity and specificity (82.4% and 81.6%). The sensitivity, specificity, PPV (positive predictive value), NPV (negative predictive value), PLR (positive likelihood ratio), and NLR (negative likelihood ratio) for each parameters are listed in Table 5.

DISCUSSION

Subclinical LV Systolic Dysfunction in T2DM

Subclinical LV systolic dysfunction in asymptomatic DM patients are caused

Table 2. Echocardiographic characteristics of patients.

Variable	Subclinical LV Dysfunction (n=34)	Normal LV Function (n= 38)	*P value*
LVMI (g/m^2)	100.5±32.1	85.6±21.0	0.02*
LVVI (ml/m^2)	44.4±12.9	42.6±8.9	0.52
LAVI (ml/m^2)	28,8±12.1	22.1±6.3	0.04*
LVEF BP (%)	62.9±5.5	65.6±5.1	0.06
TAPSE(mm)	21.2±2.7	22.7±2.8	0.12
E velocity (cm/s)	73.4±16.3	76.3±18.0	0.49
E/A ratio	0.8±0.2	0.9±0.2	0.003*
Septal E' (cm/s)	6.6±1.8	8.4±2.1	<0.001*
Lateral E' (cm/s)	8.3±1.7	10.9±2.7	<0.001*
Septal E/E'	11.9±3.9	9.7±3.8	0.02*
Lateral E/E'	9.2±2.7	7.6±3.3	0.03*
Average E/E'	10.3±3.1	8.5±3.4	0.02*
Septal MAPSE (mm)	11.5±2.2	13.9±2.7	<0.001*
Lateral MAPSE (mm)	13.1±2.4	15.9±2.8	<0.001*
Average MAPSE (mm)	12.3±1.9	14.9±2.2	<0.001*
Septal TDI Sm (cm/s)	6.3±1.2	7.7±1.5	<0.001*
Lateral TDI Sm (cm/s)	7.2±1.4	9.1±1.4	<0.001*
Average TDI Sm (cm/s)	6.8±1.2	8.4±1.4	<0.001*
GLS (%)	-14.2±2.1	-19.0±1.1	<0.001*

*Significant (p<0.05); GLS: global longitudinal strain; LVMI: left ventricular mass index; LVVI: left ventricular volume index; LAVI: left atrium volume index; LVEF BP: left ventricular ejection fraction biplane; MAPSE: mitral annular plane systolic excursion; TAPSE: tricuspid annular plane systolic excursion; TDI: tissue doppler imaging; Sm: mitral annular peak systolic velocity.

Table 3. Correlation of MAPSE and TDI Sm with GLS.

Echocardiographic Parameters	Correlation Coefficient (r)	*p*
Septal MAPSE	-0.44	<0.001*
Lateral MAPSE	-0.43	<0.001*
Average MAPSE	-0.51	<0.001*
Septal TDI Sm	-0.52	<0.001*
Lateral TDI Sm	-0.57	<0.001*
Average TDI Sm	-0.59	<0.001*

*Significant (p<0.05); MAPSE: mitral annular plane systolic excursion; TDI: tissue doppler imaging; Sm: mitral annular systolic velocity

by microvasculopathy, myocardial hypertrophy, and cardiac fibrosis. The transforming growth factors beta, abberant differentiantion of fibroblast progenitor cells due to hyperinsulinemia, and dysregulation of extracellular matrix due to hyperglycemia are recognized as pathophysiology of cardiac fibrosis.[1] In addition, disruption of calcium (Ca^{2+}) hemostasis and decreased sensitivity of intracellular proteins to calcium causes impaired excitation-contraction coupling that contributes to subclinical and clinical myocardial systolic dysfunction.[5]

Decreased GLS has been described as an early marker of myocardial impairment in diabetic patients and is considered as gold standard in detecting subclinical LV systolic dysfunction.[5] Subclinical LV dysfunction is independently associated with all-cause mortality in diabetic patients. This result has been proved by a cohort study of 397 T2DM patients that subclinical LV systolic dysfunction is independently associated with all-cause mortality during follow-up 5 years with a hazard ratio (HR) of 2.83, 95% confidence interval (CI) 1.40 to 5.71, p = 0.004. Diabetic individuals without LV systolic dysfunction had survival rate similar to the general population, with standardized mortality ratio 0.94, 95% CI 0.52 - 1.58.[8]

A study by Mochizuki et al.[1] 2015 on 144 asymptomatic DM patients without coronary artery disease found that the prevalence of patients with subclinical left ventricular systolic dysfunction (GLS >-18%) is 37% or about 53 patients. Type 2 diabetes mellitus, overweight/obesity, hypertriglyceridemia, nephropathy, and neuropathy were independently associated with GLS of >-18%. In addition, a study in 60 asymptomatic diabetic patients with normal EF showed that the duration of diabetes is an independent predictor of decreased GLS (p = 0.031).[9] This study uses a population of asymptomatic type 2 diabetes with LVEF > 50%, the prevalence of patients with subclinical left ventricular dysfunction (GLS -18%) is slightly higher than the study of Mochizuki et al.[1], which is 47.2%. There is no difference in the two groups' proportion of patients with overweight/obesity, hypertriglyceridemia, or nephropathy. The relationship between the duration of DM and GLS is also shown by the mean duration of DM being longer in the GLS group -18% compared to normal GLS (94.12±72.5 months and 60.79±55.51 months; p = 0.031).

Association of MAPSE and TDI Sm with Left Ventricular Systolic Function

Left ventricular longitudinal shortening is a sensitive marker which reflects cardiac function and can be determined by measuring MAPSE.[10] MAPSE has been used as echocardiographic parameter in determining left ventricular longitudinal function and correlated to left ventricular global systolic function. Previous clinical studies have shown that MAPSE described the movement of the mitral annulus in the systolic phase, can be used to determine global cardiac longitudinal function and was a sensitive parameter for defining abnormalities in patients with early-stage cardiovascular disease, where longitudinal function is impaired before further abnormalities occured.[10]

Measurement of MAPSE is obained using M-mode, therefore it does not require good image quality, due to good echogenicity in the atrioventricular

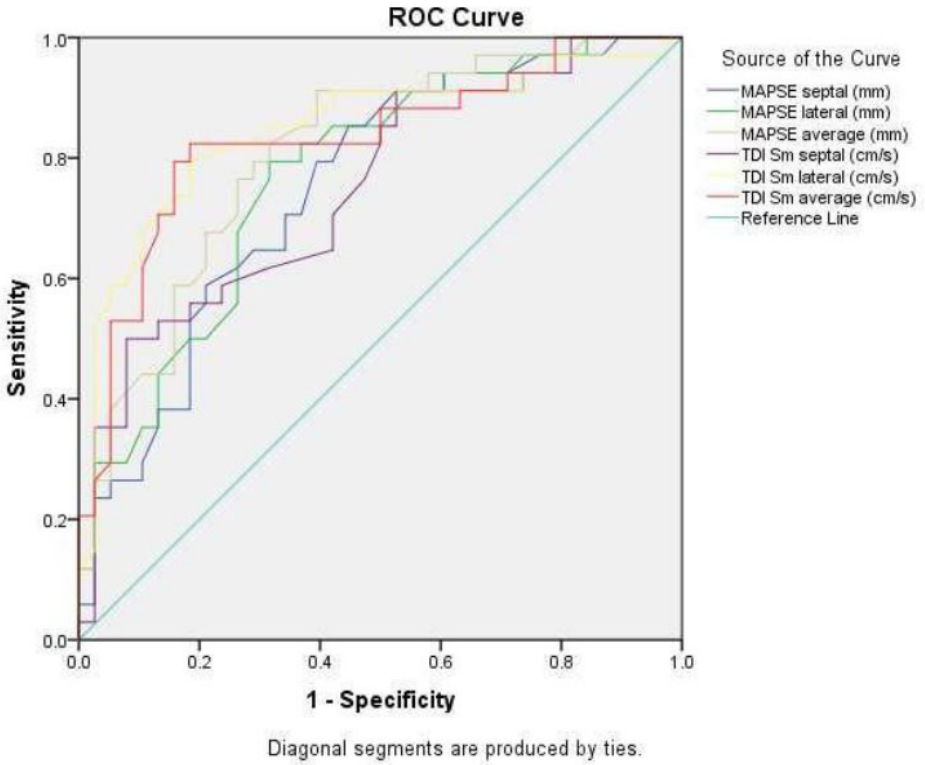

Figure 1. ROC curve analysis of MAPSE and TDI Sm in determining subclinical LV systolic dysfunction; MAPSE: mitral annular plane systolic excursion; TDI: tissue doppler imaging; Sm: mitral systolic velocity; ROC: receiver operating characteristics.

annulus. MAPSE can be measured in the septal, lateral, anterior, and posterior regions with a normal range of 12 to 15 mm. MAPSE < 8 mm is associated with a decrease in LVEF < 50% with a specificity of 82% and a sensitivity of 98%. Average MAPSE of 10 mm was associated with preserved EF (≥55%), with sensitivity of 90% to 92% and specificity of 87%. In addition, average MAPSE <7 mm is able to detect an EF <30% with sensitivity of 92% and specificity of 67% in patients with dilated cardiomyopathy and severe congestive heart failure.[10] The association between MAPSE and EF was valid in normal or left ventricular dilatation, whereas this correlation was poor in patients with left ventricular hypertrophy. Another limitation of this parameter is small localized abnormalities cannot be detected because MAPSE only evaluates the longitudinal function of the entire left ventricular wall and cannot evaluate segmental function.[10]

Mitral annular velocities can be measured using pulsed wave Doppler with TDI. Movement of the mitral annulus produce three waves, such as (1) systolic velocity (S') in the systolic phase, (2) early diastolic velocity (E'), and (3) late diastolic velocity (A') in the diastolic phase. The measurements can be performed easily without relying on image quality with low inter- and intraobserver variability. Therefore, this method can be used to determine both systolic and diastolic functions. During the ejection period, there is a longitudinal shortening of the left ventricle which causes movement of the mitral annulus and produces an S' velocity. There is a significant correlation between TDI Sm and LVEF with 2DE (r=0.738; p<0.001) and 3DE (r=0.688;p<0.001). TDI Sm < 6.8 cm/s showed sensitivity 94.1% and specificity 87, 0% in detecting left ventricular systolic dysfunction with 3DE. TDI Sm < 6.8 cm/s showed sensitivity 95.1% and specificity 91.3% in detecting left ventricular systolic dysfunction with 2DE.[7]

However, this method has limitations in determining left ventricular function globally because mitral annular velocities described regional movement and had low accuracy in patients with regional wall motion abnormalities.[11] In addition, TDI Sm only provides a quantification of myocardial movement. This method cannot distinguish whether the acquired velocities are due to active or passive movement, thus global myocardial movement and tethering effects on adjacent myocardium can result in an inappropriate increase in velocity of the dysfunctional segment.[10]

The ability of MAPSE and TDI Sm in determining left ventricular systolic function has been demonstrated through a study by Khorshid et al. The cut-off value of 7 mm septal MAPSE can indicate impaired left ventricular systolic function (EF <50%) with sensitivity 73% and specificity 100%. Lateral MAPSE 10 mm had sensitivity 82% and specificity 93%. Meanwhile, TDI Sm with a cut-off value of 7 cm/s showed impaired left ventricular systolic function with sensitivity 91% and specificity 85% for the septal annulus and sensitivity 72% and specificity 93% for the lateral annulus. When the two cut-off values of the two parameters (MAPSE and TDI Sm) are combined to detect left ventricular systolic dysfunction, the sensitivity and specificity increases to 95.9% and 100% for the mitral septal annulus and 85.5% and 97, respectively.[6]

The Role of MAPSE and TDI Sm in Determining Subclinical Left Ventricular Systolic Dysfunction

This study found that MAPSE and TDI Sm have good accuracy in determining subclinical LV systolic dysfunction in T2DM patients. Septal, lateral, and average MAPSEs have AUC of 0.752, 0.772, and 0.809, respectively. Lateral MAPSE <14.45 mm had the best sensitivity, NPV, and NLR, while average MAPSE <13.4 mm had the best specificity, PPV, and PLR of the three parameters. Meanwhile, septal, lateral, and average TDI Sm had AUC of 0.750, 0.847, and 0.830, respectively. Compared to all parameters, TDI Sm average <7.425 cm/s is the best parameter in determining subclinical LV systolic dysfunction in T2DM patients with a sensitivity 82.4%, specificity 81.6%, PPV 80.0 %, NPV 83.8%, PLR 4.47, and NLR 0.22.

The results of this study are similar

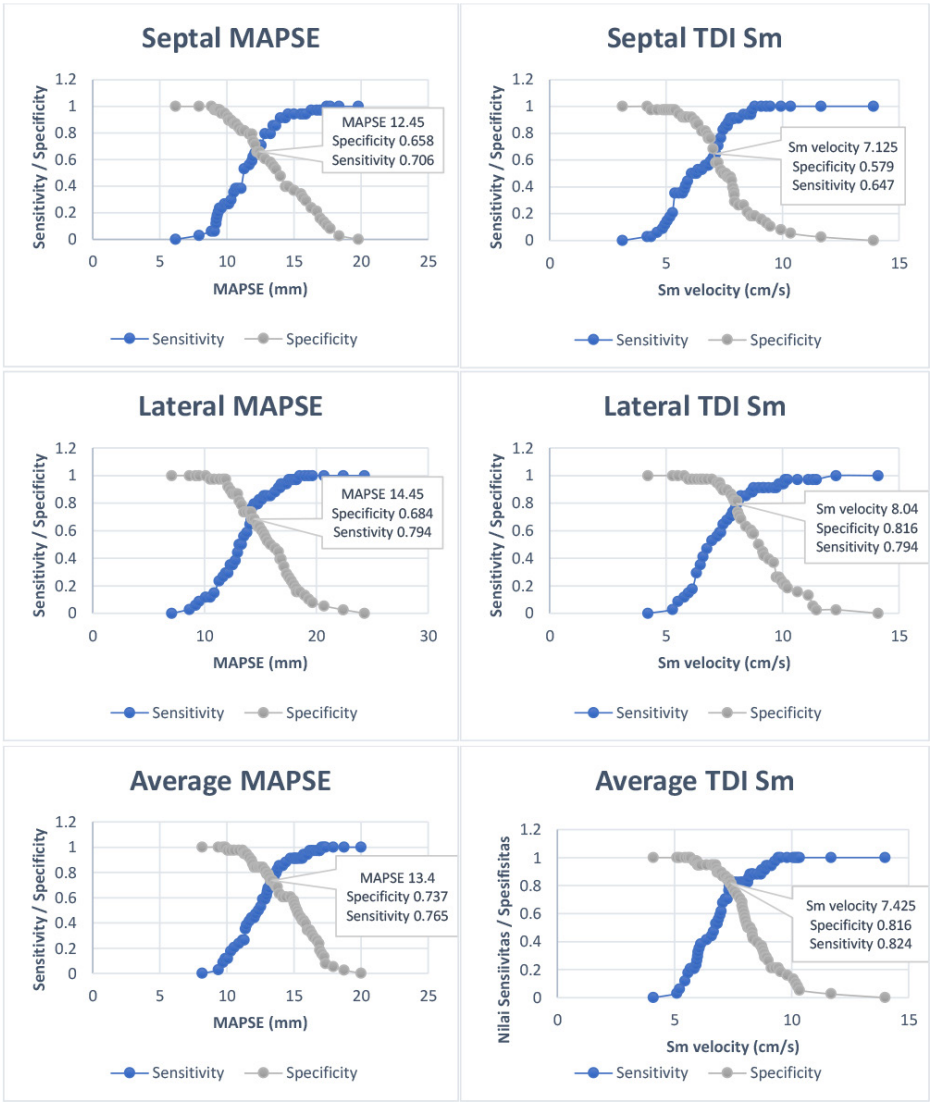

Figure 2. Line chart for cut-off point of MAPSE and TDI Sm in determining subclinical LV systolic dysfunction based on sensitivity and specificity; MAPSE: mitral annular plane systolic excursion; TDI: tissue doppler imaging; Sm: mitral systolic velocity.

Table 4. AUC of MAPSE and TDI Sm based on ROC curve analysis.

Parameter	AUC	*p*	95% CI
Septal MAPSE	0.75	<0.001*	0.64 – 0.86
Lateral MAPSE	0.77	<0.001*	0.66 – 0.88
Average MAPSE	0.81	<0.001*	0.71 – 0.90
Septal TDI Sm	0.75	<0.001*	0.64 – 0.86
Lateral TDI Sm	0.85	<0.001*	0.75 – 0.94
Average TDI Sm	0.83	<0.001*	0.73 – 0.93

*significant (p<0.05); MAPSE: mitral annular plane systolic excursion; TDI: tissue doppler imaging; Sm: mitral systolic velocity; AUC: areas under the curve; ROC: receiver operating characteristics; CI: confidence interval

to the study by Magdy et al.[5] in 80 asymptomatic T2DM patients found that MAPSE was strongly correlated with GLS (r=0.789; p<0.001). The 16.4 mm MAPSE have 100% sensitivity and 73.2% specificity (AUC 0.887; PPP 48; NPP 100; p<0.001) in predicting subclinical LV dysfunction in T2DM patients.[5] However, based on the study by Hamza et al.[12], the average MAPSE had better AUC than TDI Sm in determining subclinical LV systolic dysfunction in T2DM patients (0.820 vs 0.681, respectively). The MAPSE cut-off value of 12 mm had sensitivity 68.52% and sensitivity 82.29%. Meanwhile, the cut-off value of TDI Sm 9 cm/s had sensitivity 100% and specificity 28.12%. However this study used different cut off for subclinical LV dysfunction (GLS >-17.1%). This study also showed a significant linear correlation between MAPSE and EF, GLS, and TDI Sm (r=0.565; 0.723; 0.595, respectively, p < 0.01). MAPSE also had a significant negative correlation with DM duration and HbA1c levels (r= -0.495; -0.776, respectively, p < 0.01).

In addition, the use of MAPSE and/or TDI Sm can also be a reliable alternative parameter in the early detection of left ventricular systolic dysfunction in patients receiving anthracycline chemotherapy. This is demonstrated through a study by Sadeq et al.[13] in 78 patients who received anthracycline chemotherapy. The mean GLS before chemotherapy is -21.8±2.4% and 3 months after chemotherapy is -19±2.2%, with a relative decrease of 13% (p<0.0001). Significant longitudinal changes in peak systolic strain are mainly found in the basal and mid left ventricular segments, whereas in the apical segment there were no changes. A decrease in MAPSE is also found before and 3 months after chemotherapy, from 11.76±1.9 mm to 10.64±1.95 mm, with a relative decrease of 9% (p=0.016). The Sm septal TDI parameter also shows a significant decrease from 8.2±1.6 cm/s to 7.3±1.1 cm/s, with a relative decrease of 11% (p=0.01).[13] Another study even shows that the parameters MAPSE and TDI Sm have greater sensitivity and specificity than EF in determining subclinical LV systolic dysfunction in patients with hematological malignancies receiving chemotherapy. MAPSE has a sensitivity of 82% and a specificity of 93%, while the TDI Sm has a sensitivity of 89% and a specificity of 95%.[14]

The mitral annulus (Sm) TDI parameter can detect early myocardial compromise in asymptomatic systemic lupus erythematosus (SLE) patients without a history or evidence of cardiovascular

Table 5. Sensitivity, specificity, PPV, NPV, PLR, and NLR of the cut-off point of MAPSE and TDI Sm for subclinical LV systolic dysfunction.

Parameter	Sensitivity (95% CI)	Specificity (95% CI)	PPV (95% CI)	NPV (95% CI)	PLR (95% CI)	NLR (95% CI)
Septal MAPSE <12.45 mm	70.6% (52.5 – 84.9)	65.8% (48.7 – 80.4)	64.9% (53.1 – 75.1)	71.4% (58.6 – 81.5)	2.06 (1.26 – 3.37)	0.45 (0.25 – 0.79)
Lateral MAPSE <14.45 mm	79.4% (62.1 – 91.3)	68.4% (51.4 – 82.5)	69.2% (57.8 – 78.7)	78.8% (65.0 – 88.2)	2.51 (1.53 – 4.14)	0.3 (0.15 – 0.6)
Average MAPSE <13.4 mm	76.5% (58.8 – 89.3)	73.7% (56.9 – 86.6)	72.2% (59.7 – 82.0)	77.8 (65.0 – 86.9)	2.91 (1.65 – 5.11)	0.32 (0.17 – 0.6)
Septal TDI Sm <7.125cm/s	64.7% (46.5 – 80.3)	57.9% (40.8 – 73.3)	57.9% (46.8 – 68.3)	64.7% (51.9 – 75.7)	1.54 (0.98 – 2.41)	0.61 (0.36 – 1.04)
Lateral TDI Sm <8.04 cm/s	79.4% (62.1 – 91.3)	81.6% (65.7 – 92.3)	79.4% (65.9 – 89.7)	81.6% (69.2 – 89.7)	4.31 (2.16 – 8.6)	0.25 (0.13 – 0.5)
Average TDI Sm <7.425 cm/s	82.4% (65.5 – 93.2)	81.6% (65.7 – 92.3)	80.0% (66.8 – 88.8)	83.8 (71.1 – 91.6)	4.47 (2.25 – 8.89)	0.22 (0.1 – 0.45)

MAPSE: mitral annular plane systolic excursion; TDI: tissue doppler imaging; Sm: mitral systolic velocity; PPV: positive predictive value; NPV: negative predictive value; PLR: positive likelihood ratio; NLR: negative likelihood ratio; CI: confidence interval

disease. There is a significant difference in mean lateral and medial Sm between SLE patients and controls. In SLE patients, the mean Sm is 9.52±1.57 cm/s and the control group is 11.96±1.55 cm/s (p 0.032) for the lateral annulus. Meanwhile, for the Sm medial annulus, the LES group was 7.2±1.55 cm/s and the control group is 9.68±1.18 cm/s (p 0.029). In addition, SLE patients with a duration of > 5 years have lower lateral and medial Sm compared to those with a disease duration of < 5 years (p 0.003 and p 0.001). However, in this study there is no difference in MAPSE in the control group of patients.[15]

Study Limitations

First, this study involved a relatively small number of patients in a single center study, therefore future studies of larger populations are necessary to get better data precision which can be extrapolated to general diabetic population. Second, patients with concomitant hypertension and possibility of coronary artery disease were not excluded from this study. However, these conditions also can affects left ventricular longitudinal function.

CONCLUSION

MAPSE and TDI Sm can be used as alternative modality in determining subclinical LV systolic dysfunction in asymptomatic T2DM patients. The MAPSE average and TDI Sm average had good level of accuracy, with AUC values of 0.809 and 0.830, respectively. Compared to other parameters, the TDI Sm average with a cut-off value of <7.425 cm/s had the best sensitivity, specificity, PPV, NPV, PLR and NLR.

ETHICAL CONSIDERATION

This study has been approved by ethical committee Faculty of Medicine- Prof. I.G.N.G Ngoerah Hospital, Bali, Indonesia with ethical clearance reference number 591/UN14.2.2.VII.14/LT/2021.

CONFLICT OF INTEREST

All author declares there is no conflict of interest regarding publication of this study.

AUTHOR CONTRIBUTION

All authors had contributed to manuscript writing and agreed for final version of the manuscript for publication.

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