# MITRAL ANNULAR PLANE SYSTOLIC EXCURSION (MAPSE);

MITRAL ANNULAR PLANE SYSTOLIC EXCURSION (MAPSE) UNDERESTIMATES LEFT VENTRICLE SYSTOLIC FUNCTION IN PATIENTS WITH LEFT VENTRICLE HYPERTROPHY

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ABSTRACT... Background and Objectives: Assessment of left ventricle function is the single most common indication for transthoracic echocardiogram. Out of different indicators of LV function, ejection fraction is the most validated one. MAPSE has promised recently to be a reliable and easily obtainable indicator for LV function even in inexperienced hands but its value in case of left ventricle hypertrophy (LVH) is questionable. Study Design: Cross-sectional comparative study. Setting: Rehmatul-lil-Alameen Institute of Cardiology, Lahore. Period: January 2015 and March 2015. Methods: 100 consecutive patients presenting for echocardiography at Rehmatul-lil-Alameen Institute of Cardiology, Lahore. Patients were divided into two groups on the basis of presence of left ventricle hypertrophy (LVMI >115 g/m<sup>2</sup> in males and > 95 g/m<sup>2</sup> in females) and further subdivided according to LVEF into those with preserved (EF>55%) and depressed EF (EF<55%). EF was calculated by visual quantification (eyeballing). MAPSE was measured using M-mode at all four mitral annular sites-medial, lateral, anterior and posterior and values averaged. Results: Among 100 patients; 66(66%) were without LVH while 34(34%) were having LVH. Without LVH group had 43 (65%) patients with preserved LVEF (EF>55%) while 23 (35%) had depressed LVEF (EF>55%). In LVH group 22 (64.7%) had preserved LVEF while 12 (35.3%) had depressed LVEF. Mean EF (%), without LVH and preserved LVEF (61.6±3.6), LVH with preserved LVEF (61.9±3.6). Mean EF (%); without LVH and depressed LVEF (41.1±6.4), LVH with depressed LVEF (42.9±5.4), p=0.663. Mean MAPSE score (mm); without LVH with preserved LVEF (13.2 $\pm$ 1.7), LVH with preserved LVEF (10.6 $\pm$ 0.9), p<0.001. Mean MAPSE score (mm); without LVH and depressed LVEF (9.17±0.9), LVH with depressed LVEF (7.4±1.5), p=0.002. Mann Whitney U test was applied to compare the MAPSE score of groups and it showed statistically significant difference in MAPSE score of patients with and without left ventricle hypertrophy with values much lower in the presence of LVH. Conclusion: Statistically significant difference exists in values of MAPSE in patients with and without left ventricle hypertrophy irrespective of LV function.

Key words: Mitral Annular Plane Systolic Excursion, Left Ventricle Hypertrophy, Ejection Fraction.

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### **INTRODUCTION**

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Mitral annular plane systolic excursion (MAPSE) measurement is an uncomplicated, quick, and reproducible<sup>1-5</sup> method for left ventricular (LV) functionassessment. Alsoknownasatrioventricular plane displacement (APVD) it reflects the global LV long-axis systolic function.<sup>6,7</sup> MAPSE has also been advocated as a reliable prognostic indicator in patients with ischemic heart disease and heart failure.<sup>8-11</sup> Its correlation with LV ejection fraction (LVEF) has also been established.<sup>1-4</sup> MAPSE

is reflection of contraction of subendocardial, longitudinal myocardial fibres, whereas LVEF depicts contractility of epicardial, circumferential myocardial fibres.<sup>12,13</sup>

The association between MAPSE and LVEF is so far considered valid only in patients with normal or dilated left ventricles<sup>14,15</sup> whereas in patients with LV hypertrophy this correlation seems doubtful.<sup>16</sup> MAPSE declines with the increasing LVH severity.<sup>3,5</sup> Ischaemia resulting from LV wall stress secondary to LV hypertrophy affects subendocardial fibres more preferentially than epicardial fibres resulting in early decline in MAPSE value as compared to LVEF. This fact makes MAPSE an earlier and more reliable marker than LVEF of LV function in patients with left ventricle hypertrophy. This difference in reflection of fibres (subendocardial vs epicardial) is suggested to be the reason of lack of correlation between MAPSE and LVEF in patients with left ventricle hypertrophy.

The aim of the present study was to examine and document the underestimation of MAPSE in the presence of left ventricle hypertrophy.

#### **METHODS**

This was a cross-sectional comparative study of 100 consecutive patients (Age=20-70 yrs) reporting for transthoracic echocardiography at Rehmatul-lil-Alameen institute of Cardiology Lahore between January 2015 and March 2015. Written informed consent was taken and study was approved by ethical review committee. Demographic and clinical data were collected. LVH risk factors were recorded according to standard definitions.

Exclusion criteria included patients on ionotropic support, atrial fibrillation, hypertrophic obstructive cardiomyopathy, severe mitral annular calcification and aortic stenosis.

Transthoracic echocardiogram was done by a consultant cardiologist and reporting was done by the same cardiologist. Echocardiography was done on Toshiba aplio artida echo machine using 3 mHz transducer. Standard protocol of transthoracic echocardiography was followed Doppler including examination. Cardiac dimensions were measured in the parasternal long axis view using M-mode. LV mass was calculated by the area -length method and indexed to body surface area (LVMI). LV hypertrophy was diagnosed on the basis of LV mass index; Males with LVMI > 115g/m2 and females >95 g/m2 were labeled as having hypertrophied LV.

Patients were divided into two groups depending on presence of LVH. Patients were further subdivided into groups on the basis of LV ejection fraction (LVEF). Those with EF >55% (Preserved LV systolic function) and those with EF < 55% (Depressed LV systolic function). Ejection fraction was calculated by eyeballing (visual quantification) based on segmental and global wall motion assessment. MAPSE was measured in milimeters by M-mode at four sites of mitral annulus-septal, lateral, anterior and posterior in apical two chamber and 4 chamber views. At each site two values were taken and then all values were averaged to obtain final MAPSE value.

#### **Statistical Analysis**

The analysis was performed using SPSS V 16.0 for windows. Quantitative data was expressed as mean value  $\pm$  1 standard deviation. Qualitative variables were presented by calculating frequency and percentage. Data was stratified for LVH and further for LVEF. Baselines characteristics were compared by t-test. Comparison of MAPSE score between groups was performed by Mann-Whitney U test. A 2-tailed P value less than 0.05 was considered statistically significant.

#### RESULTS

Among 100 patients; 66(66%) were without LVH while 34(34%) were having LVH (Table-I). Without LVH group had 43 (65%) patients with preserved LVEF (EF>55%) while 23 (35%) had depressed LVEF (EF>55%). In LVH group 22(64.7%) had preserved LVEF while 12 (35.3%) had depressed LVEF (Table-I).

Mean age; Without LVH group with preserved LVEF (52.62  $\pm$  9.46 years), LVH group with preserved EF (50.52  $\pm$  9.40 years), p value 0.273. Mean age; Without LVH group with depressed EF (52.62  $\pm$  9.46 years), LVH group with depressed EF (50.52  $\pm$  9.40 years), p value 0.273.

Mean LVMI (g/m<sup>2)</sup>; without LVH and preserved LVEF (74.7 $\pm$ 22.1), LVH and preserved LVEF (124.6 $\pm$ 12.5), p <0.001. Mean LVMI (g/m<sup>2</sup>); without LVH with depressed LVEF (66.8 $\pm$ 21.6), LVH with depressed LVEF (118.7 $\pm$ 14.1), p<0.001.

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			Without LVH(n=66)	With LVH(n=34)	P value
Preserved LV Systolc Function	Number		43	22	
	Age		53.1±12.2	54.0±12.9	0.793
	Sex	Male	27	13	
		Female	16	09	0.528
	LVMI(g/m2)		74.7±22.1	124.6±12.5	< 0.001
	EF (%)		61.6±3.6	61.9±3.6	0.841
	MAPSE(mm)		13.2±1.7	10.6±0.9	< 0.001
Depressed LV Systolic Function	Number		23	12	
	Age		50.0±12.0	56.2±10.7	0.160
	Sex	Male	14	07	
		Female	09	05	0.889
	EF (%/)		41.1±6.4	42.9±5.4	0.663
	LVMI(g/m2)		66.8±21.6	118.7±14.1	< 0.001
	MAPSE(mm)		9.17±0.9	7.4±1.5	0.002

Table-I. Comparison of patients with and without left ventricle hypertrophy

Mean EF (%), without LVH and preserved LVEF (61.6±3.6), LVH with preserved LVEF (61.9±3.6). Mean EF (%); without LVH and depressed LVEF (41.1±6.4), LVH with depressed LVEF (42.9±5.4), p=0.663. Mean MAPSE score (mm); without LVH with preserved LVEF (13.2±1.7), LVH with preserved LVEF (10.6±0.9), p<0.001. Mean MAPSE score (mm); without LVH and depressed LVEF (9.17±0.9), LVH with depressed LVEF (7.4±1.5), p=0.002. Mann Whitney U test was applied to compare the MAPSE score of groups and it showed statistically significant difference in MAPSE score of patients with and without left ventricle hypertrophy with values much lower in the presence of LVH.

#### DISCUSSION

In this observational study, statistically significant effect of left ventricle hypertrophy on MAPSE is documented which exists in patients with both preserved and depressed LV systolic function.

LV longitudinal shortening is a sensitive parameter reflecting cardiac pump function<sup>17,18</sup> High echogenicity in the atrioventricular annulus with no requirement of accurate endocardial border identification makes measurement of MAPSE relatively easy and readily reproducible. The average normal value of MAPSE derived from previous studies for the four annular regions (septal, anterior, lateral, and posterior) ranges from 12 to 15 mm.<sup>19,20</sup> In normal individuals value of lateral MAPSE is the highest.

Studies have correlated MAPSE with ejection fraction. One study has linked MAPSE < 8 mm with a depressed LV EF (<50%)<sup>19</sup> while in another study MAPSE of 10 mm was associated with preserved EF (55%).<sup>1,24</sup> In another study MAPSE score < 7 mm was associated an EF < 30%.<sup>3</sup> Even in cases with regional wall motion abnormalities this correlations was validated.<sup>3,4,22</sup>

In a study by B Wandt et al<sup>16</sup> there was no significant relation between MAPSE and ejection fraction in patients with LVH, irrespective of cause of LV hypertrophy.<sup>16</sup> We excluded hypertrophic cardiomyopathy patients from our study but our results match with the results of B Wandt in terms of lack of relation between EF and MAPSE in LVH.

MAPSE measurements are highly reproducible even in presence of LVH.<sup>23,24</sup> In addition to underestimating left ventricular function in patients with hypertrophy, as concluded in the present study, studies have also correlated MAPSE with severity of aortic stenosis and MAPSE is being evaluated as a marker of severity of aortic stenosis.

Measurement of MAPSE is an easy and simple method but still some expertise is required in its measurement as during the isovolumic relaxation phase the annular ring longitudinal motion towards apex continues in some patients and ignoring it can result in underestimation of MAPSE.<sup>25</sup>

In a recent study, reference values for MAPSE for a wide range of body size and age were reported.<sup>6</sup> Even though studies have shown correlation between mitral ring motion and ejection fraction, it is still questionable to translate ring motion to ejection fraction specially in the presence of LVH, a different set of values of MAPSE may be required to predict EF.

Several researchers have investigated the reason of low MAPSE in case of LVH including theory of increased mean muscle diameter<sup>26</sup>, presence of areas with marked fibre disarray and disorganization of the intercellular junctions.<sup>27,28</sup> But more logical explanation for the result of the present study is the ischaemia theory. Ischaemia resulting from increased wall stress in LVH preferentially affects subendocardial longitudinal fibre function which is reflected as low MAPSE value. However, the reason for this depressed long axis fibre function in left ventricular hyper trophy still remains to be investigated further.

#### **CONCLUSION**

In conclusion, this study has shown that MAPSE underestimates LV Systolic function in patients with left ventricle hypertrophy as compared to those without left ventricle hypertrophy.

The limitations of the study were that study population was a limited number and exclusion of patients with HOCM & aortic stenosis. It would also be interesting to follow up patients for prognostic significance, and study the effect of low MAPSE scores in LVH patients.

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#### REFERENCES

- 1. Alam M, Höglund C, Thorstrand C. Longitudinal systolic shortening of the left ventricle: An echocardiographic study in subjects with and without preserved global function. Clin Physiol 1992; 12:443–52.
- Alam M, Höglund C. Serial echocardiographic studies following thrombolytic treatment in myocardial infarction with special reference to the atrioventricular valve plane displacement. Clin Cardiol 1992; 15:30–6.
- 3. Alam M, Höglund C, Thorstrand C, Philip A. Atrioventricular plane displacement in severe

congestive heart failure following dilated cardiomyopathy or myocardial infarction. J Int Med 1990; 228:569–75.

- Alam M, Höglund C, Thorstrand C, Hellekant C. Haemodynamic significance of the atrioventricular plane displacement in patients with coronary artery disease. Eur Heart J 1992; 13:194–200.
- Wandt B, Bojö L, Wranne B. Influence of body size and age on mitral ring motion. Clin Physiol1997; 17:635– 46.
- Willenheimer R, Cline C, Erhardt L, Israelsson B. Left ventricular atrioventricular plane displacement: an echocardiographic technique for rapid assessment of prognosis in heart failure. Heart 1997; 78:230–6.
- Willenheimer R, Erhardt L, Cline C, Rydberg ER, Israelsson BA. Prognostic significance of changes in left ventricular systolic function in elderly patients with congestive heart failure. Coron Artery Dis 1997; 8:711–7.
- Willenheimer R, Israelsson B, Cline C, Rydberg E, Broms K, Erhardt L. Left atrioventricular plane displacement is related to both systolic and diastolic left ventricular performance in patients with chronic heart failure. Eur Heart J 1999; 20:612–8.
- Rydberg E, Willenheimer R, Erhardt L. Atrioventricular plane displacement determined by echocardiography: a clinically useful, independent predictor of mortality in patients with stable coronary artery disease. J Intern Med 2003; 254:479–85.
- Brand B, Rydberg E, Ericsson G, Gudmundsson P, Willenheimer R. Prognostication and risk stratification by assessment of left atrioventricular plane displacement in patients with myocardial infarction. Int J Cardiol 2002; 83:35–41.
- Rydberg E, Arlbrandt M, Gudmundsson P, Erhardt L, Willenheimer R. Left atrioventricular plane displacement predicts cardiac mortality in patients with chronic atrial fibrillation. Int J Cardiol 2003; 91:1–7.
- Lundbäck S. Cardiac pumping and the function of the ventricular septum. Acta Physiol Scand 1986; 550 (suppl):1–101.
- Henein M, Priestley K, Davarashvili T, Buller N, Gibson DG. Early changes in left ventricular subendocardial function after successful coronary angioplasty. Br Heart J 1993; 69:501–6.
- 14. Hoffman EA, Ritman EL. Invariant total heart volume in the intact thorax. Am J Physiol 1985; 2 4 9:H883-90.
- 15. Carlh€all CJ, Lindstrom L, Wranne B, Nylander E.

Atrioventricular plane displacement correlates closely to circulatory dimensions but not to ejection fraction in normal young subjects. Clin Physiol 2001; 21:621-8.

- 16. Wandt B, Boj€o L, Tolagen K, Wranne B. Echocardiographic assessment of ejection fraction in left ventricular hypertrophy. Heart 19 99; 82:192-8.
- Carlsson M, Ugander M, Mosen H, Buhre T, Arheden H. Atrioventricular plane displacement is the major contributor to left ventricular pumping in healthy adults, athletes, and patients with dilated cardiomyopathy. Am J Physiol Heart Circ Physiol 2007; 292:H1452-9.
- 18. Jones CJ, Raposo L, Gibson DG. Functional importance of the long axis dynamics of the human left ventricle. Br Heart J 19 90; 63:215-20.
- Simonson JS, Schiller NB. Descent of the base of the left ventricle: an echocardiographic index of left ventricular function. J Am Soc Echocardiography 198 9; 2:25-35.
- Mondillo S, Galderisi M, Ballo P, Marino PN. Left ventricular systolic longitudinal function: Comparison among simple M-mode, pulsed, and M-mode color tissue Doppler of mitral annulus in healthy individuals. J Am Soc Echocardiogr 2006; 19:1085-91.
- 21. Silva JA, Khuri B, Barbee W, Fontenot D, Cheirif J. Systolic excursion of the mitral annulus to assess

septal function in paradoxic septal motion. Am Heart J 19 96; 131:13 8-45.

- 22. Alam M. The atrioventricular plane displacement as a means to evaluate left ventricular function in acute myocardial infarction. Clin Cardiol 1991; 41:588–94.
- Höglund C, Alam M, Thorstrand C. Atrioventricular plane displacement in healthy persons. An echocardiographic study. Acta Med Scand 1988; 224:557–62.
- Hammar ström E, Wranne B, Pinto FJ, et al. Tricuspid annular motion. J Am Soc Echocardiography 1991; 4:331–9.
- Wandt B, Bojö L, Hatle L, Wranne B. Left ventricular contraction pattern changes with age in normal adults. J Am Soc Echocardiogr 1998; 11:857–63.
- Tanaka M, Fujiwara H, Onodera T, Wu D-J, Hamashima Y, Kawai C. Quantitative analysis of myocardial fibrosis in normals, hypertensive hearts, and hypertrophic cardiomyopathy. Br Hear t J1986; 55:575–81.
- Sepp R, Sever s NJ, Gourdie RG. Altered patter ns of cardiac intracellular junction distributed in hypertrophic cardiomyopathy. Heart 1996; 76:412–17.
- Hoshino T, Fujiwara H, Kawai C, Hamashima Y. Myocardial fiber diameter and regional distribution in the ventricular wall of normal adult hearts, hypertensive hearts and hearts with hypertrophic cardiomyopathy. Circulation 1983; 67:1109–16.

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