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ORIGINAL ARTICLE

Validity of the distance between mitral leaflets coaptation point and annular plane in differentiation between ischemic and dilated cardiomyopathy

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KEYWORDS

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Abstract *Background:* Identification of patients with ischemic cardiomyopathy (ICM) from those with idiopathic dilated cardiomyopathy (DCM) is important therapeutically and prognostically.
Objective: To assess the validity of the distance between the mitral leaflets coaptation point and the mitral annular plane (CPMA) at low dose dobutamine stress echocardiography (DSE) for differentiation between ICM and DCM.
Patients and methods: Echocardiographic indices and CPMA were measured at baseline and during dobutamine infusion for 50 patients who were presenting with heart failure and reduced ejection fraction (EF). Patients were divided into two groups depending on coronary angiographic findings, group I (ICM with significant coronary artery disease) and group II (DCM with normal coronary arteries).
Results: Compared with baseline values, the CPMA at low dose DSE decreased significantly in ICM patients (11.8 ± 2.2 vs 8 ± 1.2 mm, $P < 0.01$) while it showed non-significant change in patients with DCM (11.66 ± 2.3 vs 11.99 ± 2.22 mm, $P > 0.05$). At low dose DSE ICM group showed a high statistically significant negative correlation between CPMA and both EF ($r = -0.749$, $P < 0.0001$) and viability index ($r = -0.782$, $P < 0.0001$) and significant positive correlation with WMSI ($r = 0.79$, $P < 0.0001$). Receiver operating characteristic (ROC) CPMA cut-off value $\leqslant 9$ mm at low dose DSE, had sensitivity of 76.92%, specificity of 85.71% in detecting patients with ICM.

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Conclusion: Measurement of CPMA at low dose DSE might help in identifying patients with ICM from those with DCM.

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1. Introduction

ICM is chronic LV dysfunction due to the sequel of diffuse coronary artery disease giving a picture which is often indistinguishable from DCM.[1]

Under normal conditions, the coaptation point of the mitral valve leaflets in systole practically reaches the level of the mitral annulus. This point is displaced apically in abnormal conditions, such as morphological abnormalities of the leaflets or dilatation of the left ventricle.[2]

As a result of the chronic hypoperfusion state, the LV becomes larger and more spherically draws the papillary muscles out from the mitral valve annulus and results in abnormal cooptation of the mitral valve.[3,4]

The aim of this study was to assess the validity of (CPMA) at low dose DSE for differentiation between ICM and DCM in patients with LV systolic dysfunction.

Patients and methods

A total number of 50 patients presenting with heart failure and reduced EF $<50\%$ were referred for coronary angiography. According to angiographic findings patients were classified into two groups, group I included 26 patients with significant coronary artery disease (ICM group) and group II included 24 patients with normal coronary arteries (DCM group). All patients gave their informed consent. The study was approved by the ethics committee of our institute.

Dobutamine protocol

Dobutamine was administered intravenously from 5 to 40 μg/kg/min in 3-min dose increments during continuous electrocardiogram and blood pressure monitoring. Atropine (to a total dose of 1 mg) was added if 85% of the maximum age-predicted heart rate was not achieved by the end of the dobutamine protocol. The test was concluded at the peak dose or earlier if the patient developed ischemia or had intolerable side effects.[5]

Stress echocardiography

Before stress testing, baseline echocardiographic study was done with an ultrasound equipment (HP Sonos 5500, USA) with the patient in the supine and left lateral position. Standard 2-dimensional views were obtained from parasternal (long and short axis) and apical (4- and 2-chambers views) windows. Digital acquisition of images was obtained at rest, at low dobutamine dose (10 mg/kg/min), at peak stress, and during recovery for side-by-side display in quad-screen format.[5]

All segments were scored at rest and stress as normal or abnormal (hypokinetic, akinetic, or dyskinetic), with the 16-segment model and the interpretation criteria of the American Society of Echocardiography.[5] The WMSI was calculated as the sum of WMS of each segment divided by total umber of segments and the viability index was defined as the number of dyssynergic segments showing improvement at LDD divided by the total number of dyssynergic segments.[6]

CPMA was measured as the distance between mitral leaflets coaptation point and the mitral annular plane at the end systole.[2] The EF was measured by the modified Simpson method.[7]

Statistical analysis

Continuous variables are expressed as mean ± standard deviation. The unpaired Student *t* test was used to compare continuous variables, and categorical data were compared using the Chi-square (χ^2) test correlation between variables which was done using correlation coefficient (*r*) to detect if changes in one variable were accompanied by changes in other variable. Agreement test (kappa coefficient) was used to relate low dose CPMA with the type of dobutamine response. Cut-off values for CPMA were estimated by receiver operating characteristic curve (R.O.C.). Statistical significance was set at less than 0.05 level.[8]

Results

Table 1 shows the demographic data of the study population. Hypertension, DM and hyperlipidemia were significantly more common in ICM group compared with the DCM group {17 (65.38%) vs 12 (50%), $P < 0.05$}, {10 (38.46%) vs 7 (29.17%), $P < 0.05$} and {22 (84.61) vs 10 (41.67%), $P < 0.05$}, respectively.

Compared with baseline values, patients with ICM showed significant decrease of both CPMA and WMSI, whereas the EF was significantly increased at low dose DSE {11.8 ± 2.2 vs 8 ± 1.2 mm, $P < 0.01$}, {1.3 ± 0.1 vs 1.06 ± 0.07 mm, $P < 0.05$} and {41.8 ± 4.2 vs 44.3 ± 5.3 mm, $P < 0.05$}, respectively (Table 2, Fig. 1).

Table 1 Demographic data of the study population.

Variables	Group I ($n = 26$)	Group II ($n = 24$)	*P*
Age (years)	56 ± 8	54 ± 7	>0.05
Sex (No. & %)			
M	19 (73.07%)	16 (66.67%)	>0.05
F	7 (26.92%)	8 (33.33%)	>0.05
Smoking (No. & %)	17 (65.38%)	14 (58.33%)	>0.05
Hypertension (No. & %)	17 (65.38%)	12 (50%)	<0.05
D.M. (No. & %)	10 (38.46%)	7 (29.17%)	<0.05
Dyslipidemia (No. & %)	22 (84.61)	10 (41.67%)	<0.05

M = Male, F = Female, D.M. = Diabetes mellitus.
$P < 0.05$ = Significant, $P > 0.05$ = Non-significant.

Table 2 CPMA, EF and WMSI at baseline and low dose DSE in the study groups.

	Group I (n = 26)			Group II (n = 24)		
	Baseline	Low dose DSE	P	Baseline	Low dose DSE	P
CPMA (mm)	11.8 ± 2.2	8 ± 1.2	< 0.01	11.66 ± 2.3	11.99 ± 2.22	> 0.05
EF (%)	41.8 ± 4.2	44.3 ± 5.3	< 0.05	35 ± 4.8	38.3 ± 2.8	> 0.05
WMSI	1.3 ± 0.1	1.06 ± 0.07	< 0.05	1.6 ± 0.1	1.79 ± 0.04	> 0.05

EF = Ejection fraction, WMSI = Wall motion score index.

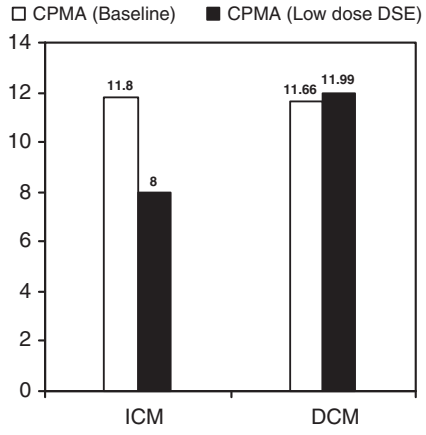

Figure 1 CPMA at baseline and at low dose DSE in the ICM and DCM groups.

At low dose DSE, compared with the DCM group, the ICM patients showed a significantly higher EF {44.3 ± 5.3% vs 38.3 ± 2.8%, $P < 0.01$}, smaller CPMA {8 ± 1.2 vs 11.99 ± 2.22 mm, $P < 0.01$}, lower WMSI {1.06 ± 0.07 vs 1.79 ± 0.04, $P < 0.01$} and a higher viability index {0.8 ± 0.17 vs 0.1 ± 0.16, $P < 0.01$} Table 3.

At low dose DSE ICM group showed a high statistically significant negative correlation between CPMA and both EF ($r = -0.749$, $P < 0.0001$) and viability index ($r = -0.782$, $P < 0.0001$) and significant positive correlation with WMSI ($r = 0.79$, $P < 0.0001$) Figs. 2 and 3.

ROC analysis showed a cut-off value of CPMA ⩽ 9 mm at low dose DSE could identify patients with ICM and differentiate them from those with DCM with a sensitivity of 76.92% and a specificity of 85.71%. The biphasic response was able to detect ICM patients with a sensitivity of 69.23% and a specificity of 100%.

This cut-off value (CPMA ⩽ 9 mm) at low dose showed agreement with biphasic response (kappa coefficient = 1, $P < 0.001$) while it showed disagreement with other types of dobutamine response ($P > 0.05$) Table 4.

Discussion

Identification of patients with ICM is of utmost importance so as to improve their outcome by directing patients for attempts at coronary revascularization to salvage the chronically hypoperfused myocardium.

This study aimed to assess the validity of CPMA at low dose DSE in identification of patients with ICM from those presenting with heart failure and reduced LV systolic function.

The occurrence of incomplete mitral leaflet closure was initially thought to be caused by dyskinesia of the left ventricular myocardium beneath one of the papillary muscles. Godley and colleagues[9] specifically detected incomplete mitral leaflet closure in the vast majority of patients with mitral valve regurgitation after myocardial infarction.[3]

In subsequent studies, it was shown that incomplete mitral leaflet closure is associated with raised left ventricular filling pressure and is not specific for the subset of patients with papillary muscle dysfunction or acute myocardial infarction.[10]

In this study at low dose DSE, patients with ICM showed a reduction in CPMA and WMSI with increase in EF, while in DCM patients all these indices showed non-significant changes. These findings may be explained by the recruitment of contractile reserve during low dose DSE which lead to the improvement of systolic function.

Kaul and colleagues[11] conclusively showed that incomplete mitral leaflet closure was related to reduced left ventricular function and CPMA correlated with left ventricular and mitral annulus size, but fractional shortening of the left ventricle was the only predictor of the presence of incomplete mitral leaflet closure on multivariate analysis.

It was suggested that a restriction in the motion of the leaflets due to CAD leading to leaflets tethering which displaces the coaptation zone from the mitral annulus toward the apex

Table 3 Echocardiographic findings at low dose dobutamine in the study groups.

Variables	Group I (n = 26)	Group II (n = 24)	P
EF (%)	44.3 ± 5.3	38.3 ± 2.8	< 0.01
CPMA (mm)	8 ± 1.2	11.99 ± 2.22	< 0.01
WMSI	1.06 ± 0.07	1.79 ± 0.04	< 0.01
Viability index (VI)	0.8 ± 0.17	0.1 ± 0.16	< 0.01

CPMA = The distance between the mitral leaflets coaptation point and the mitral annular plane, EF = Ejection fraction, WMSI = Wall motion score index.

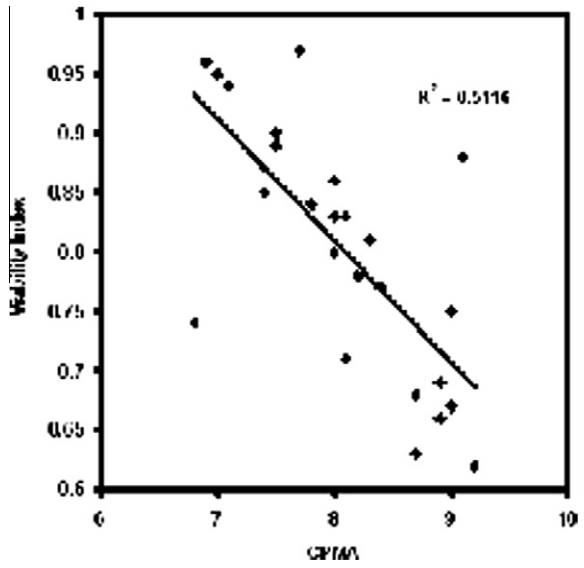

Figure 2 Correlation between low dose CPMA and viability index in ICM group.

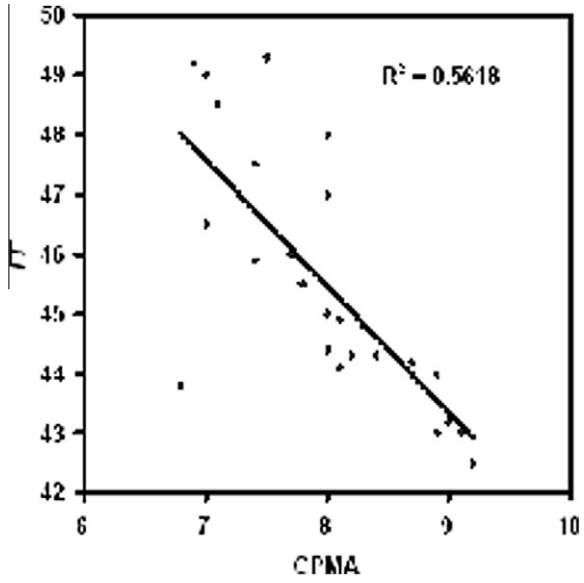

Figure 3 Correlation between low dose CPMA and EF in ICM group.

Table 4 Association between CPMA $\leqslant$ 9 mm at low dose and dobutamine response (agreement test).

Response	Low dose CPMA $\leqslant$ 9 mm	Low dose CPMA $>$ 9 mm	Kappa coefficient	*P*
Biphasic (*n*)	17	1	1	<0.01
Sustained improvement (*n*)	5	0	0.39	>0.05
No change (*n*)	0	16	0.13	>0.05
Worsening (*n*)	0	11	0.09	>0.05

of the left ventricle causes an incomplete closure of the mitral valve in systole.[12,13]

Kinney and Frangi[10] studied 73 patients with incomplete mitral leaflet closure and found that only 10% of them had acute myocardial infarction. The most important determining factor for the occurrence of incomplete mitral leaflet closure was the presence of mitral valve "B bumps" on M mode, which is typically associated with raised left ventricular end diastolic pressure and an increase in left ventricular end diastolic dimension in both dilated and ischemic cardiomyopathy.

Other investigators also suggested that local remodeling of the left ventricle displaces papillary muscles and leads to a traction on the mitral leaflets. Incomplete leaflet closure may also be the consequence of regional wall motion abnormalities observed after a myocardial infarction or in severe chronic myocardial ischemia.[14]

CPMA is also favored by the imbalance between tethering forces and decreased ventricular forces acting to close the leaflets. These decreased ventricular forces are the consequence of the left ventricular contractile dysfunction.[15]

In this study, patients with ICM showed a statistically significant negative correlation between CPMA and EF, viability index and significant positive correlation with WMSI, moreover, there was a highly significant agreement between CPMA $<$ 9 mm at low dose DSE and biphasic response at DSE.

After administration of an inotrop (dobutamine), contractile function improved and the distance between the mitral annulus plane and the coaptation point of the mitral leaflets (that is, the CPMA) decreased, further supporting our hypothesis that this is an index of systolic function.

Conclusion

From this study, it could be concluded that the use of CPMA during low DSE as a marker of myocardial viability can be of help in conjunction with other parameters used in this regard such as the biphasic or sustained improvement types of dobutamine responses, in the identification of patients with ICM.

Clinical implication

CPMA at low dose DSE may be of help in diagnosing patients with ICM who might have viable myocardium and who can benefit from revascularization and could be used in patients with suboptimal echo window in contrast to other indices used during DSE that depend on the analysis of wall motion of the LV and hence an optimal echo window is a must to get good results.

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