Journal of Medicine in Scientific Research

Volume 5 | Issue 4 Article 7

Subject Area:

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Recommended Citation

Badran, Mohamed R. (2023) "The role of myocardial mechanical and QTc dispersion in detecting significant coronary artery disease," *Journal of Medicine in Scientific Research*: Vol. 5: Iss. 4, Article 7. DOI: https://doi.org/10.4103/jmisr.jmisr_132_20

Original Article

The role of myocardial mechanical and QTc dispersion in detecting significant coronary artery disease

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Abstract

Background
Prolonged myocardial contraction and QT interval duration may be found in ischemic viable myocardium, yet it is unclear if noninvasive examination of regional nonhomogeneous myocardial mechanics and QT duration may detect patients with significant coronary artery disease (CAD).

Aim
To assess the ability of cardiac mechanical and QTc dispersions to identify significant CAD in patients having chest pain with no prior myocardial infarction.

Patients and methods
We examined 125 patients who had chest pain and evaluated by coronary angiography. They are divided into two groups. CAD group included 75 patients with significant CAD, and control group included 50 patients without significant CAD. We used echocardiographic speckle tracking to calculate longitudinal strain. We measured cardiac contraction duration as the time between the ECG first deflection in QRS complex and longitudinal strain peak within every segment of the 17-segments left ventricular model. We measured mechanical dispersion by calculating the SD of 17-time intervals (dispersionSD17) or by subtracting the shortest contraction duration from the longest one (dispersion-delta). QTc dispersion was defined as the difference between the shortest and the longest QTc intervals as measured in the 12-lead ECG. We compared CAD group and control group regarding mechanical and QTc dispersions.

Results
The CAD group had longer average contraction duration than the control group (436.13 ± 51.97 vs. 417.79 ± 40.76 ms, $P = 0.036$), and the contraction duration was significantly associated with QTc interval ($r = 0.501$, $P < 0.001$). Mechanical dispersion had an independent correlation with CAD ($P < 0.001$), but QTc dispersion and duration did not exhibit a significant association with CAD. Mechanical dispersion exhibited an incremental value over global longitudinal strain, wall motion score index, and conventional risk factors in identifying significant CAD.

Conclusions
In individuals with no prior myocardial infarction, the myocardial mechanical and QTc dispersions can predict the existence of significant CAD. Cardiac mechanical dispersion has an incremental value on global longitudinal strain for identification of patients with significant CAD.

Keywords: Coronary artery disease, mechanical dispersion, QT dispersion, QT interval

Introduction

It is a difficult scenario in clinical practice to diagnose coronary artery disease (CAD) in patients who experience chest pain without myocardial infarction (MI). Two-D echocardiographic

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Access this article online

Quick Response Code:

Website: www.jmsr.eg.net

DOI: 10.4103/jmisr.jmisr_132_20

Submitted: 24-Dec-2020 **Revised:** 17-Feb-2021 **Accepted:** 21-Feb-2021 **Published:** 11-Mar-2023

How to cite this article: Badran MR. The role of myocardial mechanical and QTc dispersion in detecting significant coronary artery disease. J Med Sci Res 2022;5:461-7.

speckle tracking could distinguish patients with significant CAD by perceiving subclinical impairment in performance of left ventricle (LV) [1–6].

Although the longitudinal peak systolic strain amplitude is the base of many strain parameters, it was found that myocardial deformation pattern can discriminate normal from ischemic myocardium [7].

Maximum of longitudinal shortening happens close to the aortic valve closure (AVC) in the normal myocardial segments, but ischemic viable myocardium exhibits prolonged longitudinal shortening away from the AVC leading to myocardial contraction duration dispersion [8]. However, normal LV may exhibit nonhomogeneous contraction resulting from the difference in LV structural design and local tension. So, it is unclear if strain imaging measurement of dispersion of myocardium's mechanics can distinguish ischemic from normal myocardium. It is also unknown if the QTc dispersion, correlated with increased probability of CAD [9–11], is associated with the mechanical dispersions in patients with significant CAD. Long duration of cardiac action potential is correlated with long contraction duration in patients with hereditary long QT syndrome, but it is unclear if this relation is present in patients with CAD [12].

So, our study aimed to investigate if significant CAD can be diagnosed by myocardial mechanical and QTc dispersions, and the relationship linking them in individuals experiencing chest pain without previous MI history.

Patients and methods

This is a case–control study. It was conducted at Cardiology Department, National Heart Institute, Egypt. It was done during the period between June 2017 and January 2020. It included retrospectively 125 consecutive patients with expected CAD evaluated by coronary angiography. They were divided into two groups. CAD group included 75 patients with significant CAD and control group included 50 patients without significant CAD.

Inclusion criteria of the study were as follows:

(1) Adequate 2D-STE image quality.
(2) Significant left anterior descending (LAD) artery stenosis.

Exclusion criteria of the study

The following were the exclusion criteria:

(1) Arrhythmias.
(2) Intraventricular conduction defect.
(3) Prior MI.
(4) Significant cardiac valve disease.
(5) Taking medications that lengthen the QT duration.

Ethical consideration

Consent was obtained from every patient after explanation of the procedures. Medical research and ethics committee approved the study.

All patients were subjected to complete history taking, as well as full general and local cardiac examination.

ECG

Just before or after echocardiographic examination, participants underwent resting ECG recording with a 12-lead ECG instrument. The QT duration was determined manually from the QRS-wave beginning until the T-wave end. We used Bazett formula to correct QT to heart rate [13,14]: QTc = QT (heart rate/60) $^{1/2}$. QTc dispersion was defined as the difference between the shortest and the longest QTc intervals as measured in the 12-lead ECG [15,16].

Echocardiography and two-dimensional speckle tracking

All patients were investigated by echocardiography before coronary angiography by using EPIC 7 Philips ultrasound machine. Images of three successive cardiac cycles were stored for offline analysis. Calculation of left ventricular ejection fraction (LVEF) was performed by modified Simpson's biplane rule. The LV division into 17 segments was performed to cover the whole LV myocardium. The four-point grading scale was applied to grade every segment (1 – normokinesia; 2 – hypokinesia; 3 – akinesia; and 4 – dyskinesia). The wall motion score index (WMSI) is then calculated by dividing the sum of the aforementioned segmental values by the number of myocardial segments [17]. Two-D speckle tracking was done on three apical views (apical long-axis, apical four-chamber, and apical two-chamber) at a frame rate of 60–80 frames/s. AVC was identified on two-dimensional image. The region of interest was adjusted to cover the myocardial thickness. Global longitudinal strain (GLS) was automatically measured from each of the three views as the mean of the global peak systolic strain from each of the three views [18]. We measured contraction duration as the time between the ECG first deflection in QRS complex and longitudinal strain peak involving postsystolic shortening [12]. It was evaluated in every LV segment. We measured mechanical dispersion by the SD of 17-time intervals (dispersionSD17) or by subtracting the shortest duration from the longest one (dispersion-delta) [19,20]. We measured and made a comparison between mechanical dispersions of apical, mid, and basal LV segments (dispersionSD6), and also compared between six anteroseptal and 12 posteroinferolateral LV segments (dispersionSD regional).

Coronary angiography

Angiography of the coronary arteries was performed according to standard clinical practice and current guidelines. We identified significant CAD as diameter stenosis more than or equal to 70% in any epicardial coronary artery. Each coronary artery examination was carried out in two perpendicular planes [21].

Statistics

Recorded data were analyzed by the Statistical Package for the Social Sciences, version 20.0 (SPSS Inc., Chicago, Illinois, USA). Quantitative data were stated as mean ± SD or median ± interquartile range. Qualitative data were

expressed as percentage. Analytical statistics were done using the following tests: Student *t* test, χ^2, Pearson correlation, multivariate and univariate logistic regression tests, and relative operating characteristic analysis. *P* value less than 0.05 was considered significant.

Results

Table 1 shows the clinical demographics of the population study. Patients with significant CAD included more males, were older, and were more hypertensive and diabetic compared with controls. Table 2 shows the angiographic characteristics of the CAD group. One-vessel disease was seen in 29 (38.7%) patients, two-vessel disease was seen in 27 (36%) patients, and three-vessel disease was seen in 19 (25.3%) patients.

Table 3 shows the comparison between the CAD group and the control group according to findings in echocardiography and ECG. In comparison with the controls, patients with significant CAD had more prolonged mean contraction durations and QTc intervals, more mechanical dispersion, higher WMSI, less LVEF, and lower GLS. Yet, QTc dispersion values were similar in significant CAD and controls. QTc intervals were significantly correlated with mechanical contraction durations ($r = 0.501$, $P < 0.001$) (Fig. 1).

Table 4 shows the univariate and multivariate regression analyses for determining the parameters related to significant CAD. Indices of mechanical dispersion and GLS were independently correlated with significant CAD. They showed the same pattern after correction for factors known to affect the longitudinal strain (age, sex, diabetes, and hypertension), whereas QTc duration and dispersion did not.

Fig. 2 shows the relative abilities of conventional risk factors (male sex, age, systemic hypertension, diabetes, hyperlipidemia, ischemic heart disease positive family history, and smoking), QTc intervals, GLS, WMSI, and mechanical dispersions in detecting significant CAD. QTc intervals exhibited an incremental value to conventional risk factors in significant CAD identification. The diagnostic value is augmented by addition of GLS and WMSI on top of QTc intervals and conventional risk factors. Lastly, diagnostic accuracy of the model is more raised by addition of mechanical dispersion to the combined GLS, WMSI, QTc intervals, and conventional risk factors.

Relative operating characteristic analysis

DispersionSD17 had a cutoff value = 42 ms that could significantly predict significant CAD [specificity = 81%, sensitivity = 82%, area under the curve (AUC) = 0.887, confidence interval (CI) 0.836–0.948, $P = 0.001$]. A cutoff value of dispersion delta of 134 ms had the same specificity and sensitivity in identification of significant CAD (AUC = 0.907, CI = 0.856–0.968, $P = 0.001$). We did not find a significant difference in accuracy between mechanical dispersion-delta and dispersionSD17 ($P = 0.123$ for the difference between the AUCs). Notably, both mechanical dispersionSD17 and dispersion-delta kept a good discriminative power when constricting analysis to patients with no wall motion abnormalities (WMA), single-vessel CAD, and multivessel CAD (Table 5). By using a cutoff value = 407 ms, QTc interval identified significant CAD (specificity = 63%, sensitivity = 62%, AUC = 0.642, CI 0.540–0.744, $P = 0.026$). QTc dispersion exhibited a discriminative performance only in the diagnosis of significant CAD in multivessel CAD patients (AUC = 0.703, CI = 0.601–0.805, $P = 0.005$). We used the method of DeLong *et al.*[22] to compare between the values of the AUC.

QTc and mechanical dispersions and the extent of coronary artery disease

Table 6 shows the echocardiographic findings of myocardial contraction durations, mechanical dispersions, GLS, and WMSI in relation to the extent of CAD. It also shows ECG findings of QTc interval, QTc dispersions, and their

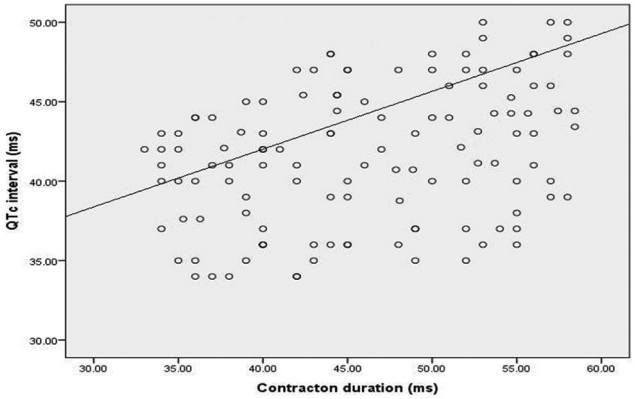

Figure 1: Contraction duration is associated with QTc interval.

Table 1: Clinical demographic data

Characteristics	CAD group (*n*=75)	Controls group (*n*=50)	*P*
Age (years)	57.66±8.37	53.01±10.23	0.006
Male/female [*n* (%)]	56 (74.6)/19 (25.4)	20 (40.0)/30 (60.0)	<0.001
Smoker [*n* (%)]	32 (42.7)	15 (30.0)	0.137
Diabetics [*n* (%)]	27 (36.0)	9 (18.0)	0.033
Dyslipidemic [*n* (%)]	59 (78.7)	39 (78.0)	1.000
Hypertensive [*n* (%)]	70 (93.3)	36 (72.0)	<0.001
Family history of IHD [*n* (%)]	32 (42.7)	25 (50.0)	0.563

CAD, coronary artery disease; IHD, ischemic heart disease.

relation to the magnitude of CAD. QTc dispersion showed no significant difference among the groups. However, QTc interval showed significant difference between patients with three-vessel CAD and control individuals. We found that the more extensive the CAD, the more markedly diminishing the global and segmental LV systolic performance. This is denoted by impairment of GLS and rising of WMSI parallel to the CAD extent (Fig. 3). However, the group of patients with two-vessel CAD exhibited more mechanical dispersions than the groups of patients with one-vessel CAD, three-vessels CAD, and control.

Table 2: Angiographic characteristics of the coronary artery disease group

Coronary angiography findings [*n* (%)]	CAD group (*n*=75)
One-vessel disease	29 (38.7)
Two-vessels disease	27 (36.0)
Three-vessels disease	19 (25.3)
LAD stenosis	75 (100.0)
LCX stenosis	24 (32.0)
RCA stenosis	41 (54.7)

CAD, coronary artery disease; LAD, left anterior descending coronary artery; LCX, left circumflex coronary artery; RCA, right coronary artery.

Reproducibility

Intraobserver variability ICCs were as follows: dispersionSD17 (0.941), dispersion-delta (0.922), QTc dispersion (0.805), GLS [0.902 (CI 0.825–0.941)], and WMSI [0.951 (CI 0.941–0.960)]. Interobserver variability ICCs were as follows: dispersionSD17 (0.776), dispersion-delta (0.844), QTc dispersion (0.398), GLS (0.863), and WMSI (0.834).

Discussion

Our study showed that patients with significant CAD had prolonged myocardial contraction durations and QTc interval compared with controls. We also found an independent association linking mechanical dispersions to significant CAD. However, QTc dispersion did not express this independent association with significant CAD. On adding mechanical dispersions to GLS, WMSI, QTc intervals, and conventional risk factors resulted in increase in the discriminative capability of diagnosing significant CAD.

Physiological versus pathological mechanical dispersions

Normal and ischemic LV may exhibit regional nonhomogeneous relaxation and contraction [8,23]. Physiological mechanical

Table 3: Comparison between coronary artery disease group and control group according to findings of echocardiography and ECG

Echocardiography and ECG	CAD group (*n*=75)	Controls group (*n*=50)	*P*
LVEF (%)	56.05±12.23	67.25±6.11	<0.001
WMSI	1.40±0.45	1.06±0.11	<0.001
GLS (%)	−17.63±4.79	−23.13±2.96	<0.001
Contraction duration (ms)	436.13±51.97	417.79±40.76	0.036
Mechanical dispersion			
DispersionSD17 (ms)	60.12±23.44	32.61±13.25	<0.001
Dispersion delta (ms)	193.61±79.48	101.90±39.74	<0.001
QTc interval (ms)	424.92±29.55	413.71±25.48	0.047
QTc dispersion (ms)	50.95±20.38	47.89±19.36	0.516

CAD, coronary artery disease; GLS, global longitudinal strain; LVEF, left ventricular ejection fraction; WMSI, wall motion score index.

Table 4: Univariate and multivariate regression tests to detect variables related to significant coronary artery disease

Parameters	Univariate		Multivariate	
	OR (95% CI)	*P*	OR (95% CI)	*P*
Age	1.071 (1.034-1.109)	0.006	1.056 (0.973-1.144)	0.447
Diabetes mellitus	2.608 (1.137-5.981)	0.029	2.763 (0.419-18.207)	0.339
Dyslipidemic	1.079 (0.486-2.398)	0.921	-	-
Male	4.908 (2.439-9.870)	<0.001	6.901 (1.443-33.003)	0.019
Smoker	1.854 (0.905-3.797)	0.115	-	-
Hypertensive	5.608 (2.305-13.642)	<0.001	52.097 (4.636-592.240)	<0.001
QTc duration	1.034 (1.019-1.049)	0.048	1.011 (0.981-1.041)	0.679
QTc dispersions	1.027 (1.006-1.049)	0.503	-	-
WMSI	1.677 (1.147-2.199)	<0.001	1.828 (1.067-3.133)	0.038
GLS	1.557 (1.340-1.809)	<0.001	1.293 (1.033-1.618)	0.043
DispersionSD17	1.137 (1.098-1.178)	<0.001	1.113 (1.037-1.192)	0.016
Dispersion delta	1.061 (1.048-1.075)	<0.001	1.054 (1.028-1.080)	0.008

CI, confidence interval; GLS, global longitudinal strain; OR, odds ratio; WMSI, wall motion score index.

Table 5: Receiver operating characteristic analysis

	All patients		Patients without WMA	
	AUC (95% CI)	*P*	AUC (95% CI)	*P*
QTc interval	0.642 (0.540-0.744)	0.026	0.550 (0.418-0.683)	0.634
QTc dispersion	0.581 (0.469-0.693)	0.233	0.540 (0.408-0.673)	0.782
WMSI	0.754 (0.683-0.836)	<0.001		
GLS	0.846 (0.785-0.917)	<0.001	0.754 (0.652-0.866)	<0.001
DispersionSD17	0.887 (0.836-0.948)	<0.001	0.836 (0.744-0.927)	<0.001
Dispersion delta	0.907 (0.856-0.968)	<0.001	0.846 (0.754-0.937)	<0.001
	Single-vessel disease		Multivessels disease	
QTc interval	0.581 (0.448-0.703)	0.348	0.683 (0.571-0.805)	0.009
QTc dispersion	0.520 (0.397-0.652)	0.955	0.703 (0.601-0.805)	0.005
WMSI	0.673 (0.571-0.795)	0.008	0.774 (0.683-0.866)	<0.001
GLS	0.815 (0.723-0.907)	<0.001	0.856 (0.897-0.937)	<0.001
DispersionSD17	0.887 (0.805-0.968)	<0.001	0.856 (0.774-0.937)	<0.001
Dispersion delta	0.897 (0.825-0.978)	<0.001	0.876 (0.805-0.958)	<0.001

AUC, area under the curve; CI, confidence interval; GLS, global longitudinal strain; WMSI, wall motion score index.

Table 6: ECG and echocardiographic findings regarding coronary artery disease extent

	Controls	One-vessel CAD	Two-vessel CAD	Three-vessel CAD	*P*
Contraction duration (ms)	417.79±40.76	423.90±50.95	442.25±54.01	446.32±48.91*	0.016
DispersionSD18 (ms)	32.61±13.25	60.12±22.42*	66.24±26.49*¥	52.99±16.30*	<0.001
Dispersion delta (ms)	101.90±39.74	186.48±67.25*	217.05±95.79*¥	172.21±65.22*	<0.001
QTc interval (ms)	413.71±25.48	418.81±26.49	425.94±30.57	432.06±29.55*	0.034
QTc dispersion (ms)	47.89±19.36	48.91±20.38	47.89±19.36	56.05±20.38	0.219
GLS (%)	−23.13±2.96	−18.95±4.08*#	−16.92±5.20*	−16.51±5.10*	<0.001
LVEF (%)	67.25±6.11	60.12±10.19*#	57.06±12.23*¥	49.93±13.25*	<0.001
WMSI	1.06±0.11	1.26±0.34#	1.42±0.44*	1.57±0.55*	<0.001

CAD, coronary artery disease; GLS, global longitudinal strain; LVEF, left ventricular ejection fraction; WMSI, wall motion score index. **P* less than 0.05 versus controls. #*P* less than 0.05 versus three-vessel CAD. ¥*P* less than 0.05 versus three-vessel CAD.

dispersions result from the regional dissimilarities in ventricular structural design and wall tension, but pathological mechanical dispersions result from postsystolic contractions of ischemic viable myocardial regions, which is discovered to be an active process and consumes energy [24,25]. So, the difference in durations of maximum contraction among ischemic and normal segments caused more mechanical dispersion in patients with significant CAD in comparison with those without. Yet, the association linking the amount of CAD to the degree of mechanical dispersions was not linear, as mechanical dispersion was more in patients having two-vessel CAD than patients having three-vessel CAD and control groups (Fig. 3). This is because patients with three-vessel CAD have more ischemic segments, as denoted by GLS and WMSI, leading to longer myocardial contraction durations within most LV segments and so less variation of myocardial contraction durations than patients with less CAD extent. Notably, mechanical dispersion kept a valuable discriminative performance in significant CAD diagnosis within subgroups of patients without WMA, patients with either single-vessel, or those with multivessels CAD.

However, physiological myocardial postsystolic shortening might be found in nearly 33% of normal regions because of spatial and chronological heterogeneity of the relaxation of myocardium [7]. Therefore, besides dissimilarities resulting from the existence of CAD, a mechanical dispersion gradient from apex-to-base was also detected and significant dispersion dissimilarities among LV anteroseptal and posteroinferolateral regions in control individuals. This finding strengthens the idea that in normal patients, mid regions of the anteroseptal wall relax before the adjoining basal and apical regions resulting in an early postsystolic lengthening of mid-wall regions and an early postsystolic shortening of basal and apical segments [7,23]. Yet, the degrees of physiological dispersions were significantly less than pathological dispersions and were similar to previously reported findings [12,19].

QTc durations and QTc dispersions

Besides hereditary syndromes with mutations in genes of myocardial ion channels, other causes of prolonging QTc interval were found, like myocardial ischemia, high glucose-insulin level, obesity, LV hypertrophy, myocardial scar, and autonomic neuropathies [11,26,27]. In the current study, significant CAD patients had prolonged QTc intervals and more commonly had systemic hypertension, segmental LV dysfunction, and diabetes than patients without CAD. Therefore, QTc interval showed an additive value to

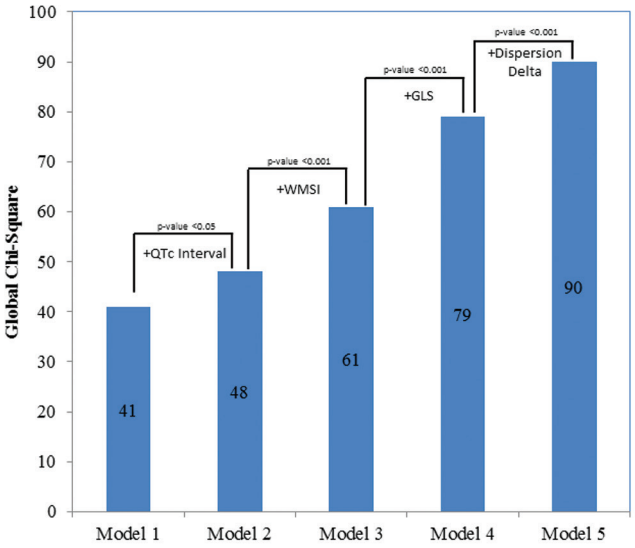

Figure 2: Mechanical dispersion had an incremental value in detecting significant CAD. Model 1 is a logistic regression model which contains CAD conventional risk factors and is nested in all other models. On adding mechanical dispersion-delta, it provided incremental value over GLS, WMSI, QTc interval, and conventional risk factors for identifying significant CAD. CAD, coronary artery disease; GLS, global longitudinal strain; WMSI, wall motion score index.

conventional risk factors in diagnosing significant CAD patients, but in multivariate regression analysis, QTc was not independently correlated with CAD, whereas QTc dispersion exhibited discriminative power in identification of significant CAD in patients with multivessel CAD only. Thus, it remains undetermined if QTc prolongation and dispersion are indicators of massive CAD or only epiphenomena due to higher level cardiac risk factors. Notably, our findings suggest that the clinical usefulness of QTc dispersion and duration in diagnosing patients with significant CAD might be hindered by its low reproducibility and less diagnostic accuracy.

Potential electromechanical associations in patients with significant coronary artery disease

Our hypothesis was that the prolonged QTc intervals were correlated to myocardial contraction prolongations, and our results support such association in the case of CAD (Fig. 1). Undoubtedly, this association should be studied by invasive electrophysiological trials, but by utilizing a similar noninvasive method, Haugaa *et al.*[12] observed that patients with hereditary long QT syndrome had mechanical dispersion significantly associated with QTc dispersion. In patients without previous MI, echocardiographic strain mechanical dispersion predicted the ventricular arrhythmias recurrence in post-MI patients independently of LVEF [19]. A valid contributing association between electrical alterations and mechanical dysfunctions and their probable arrhythmogenic ability in significant CAD patients without previous MI necessitate further investigation.

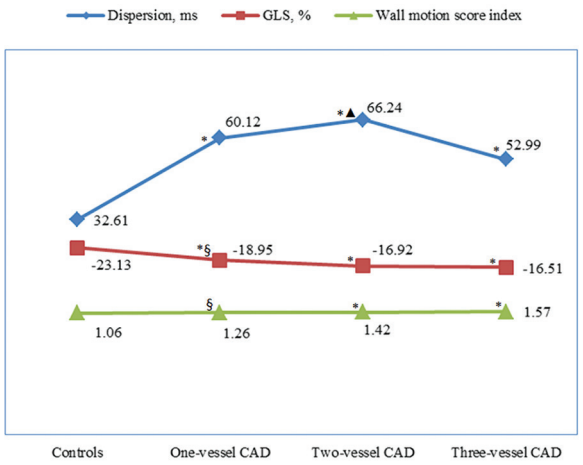

Figure 3: Mechanical dispersion (dispersionSD17) WMSI, and GLS with regard to the extent of CAD. Asterisk denotes *P* value less than 0.05 versus controls; filled triangle denotes *P* value less than 0.05 versus three-vessels CAD; section symbol denotes *P* value less than 0.05 versus three-vessels CAD. CAD, coronary artery disease; GLS, global longitudinal strain; WMSI, wall motion score index.

Mechanical dispersion's incremental value for detecting coronary artery disease

A step-by-step rise in regression models' accuracy on adding GLS and WMSI to conventional risk factors verifies the significance of LV dysfunction in identifying patients with significant CAD. Furthermore, the residual diagnostic uncertainty can be additionally decreased by evaluating mechanical dispersion of the myocardium. Mechanical dispersion and GLS do not present identical data about segmental LV function, although they are obtained from the same longitudinal strain curves. GLS is determined by the peak amplitudes of regional strains and might remain normal because of the effect of averaging but mechanical dispersion is determined by time measuring and were affected even with mild LV dysfunction. Notably, mechanical dispersion became smaller with the increase of number of ischemic segments in individuals with multivessels CAD, but still significantly abnormal.

Clinical implications

Exclusion of significant CAD in patients with chest pain and negative cardiac biomarkers is an expensive process and consumes time because it needs stress tests, multislice computed tomography angiography of the coronaries, or coronary angiography. New echocardiographic indices, depending on GLS, can at rest identify patients with significant CAD, without needing stress tests [1–4,27]. Our study showed that measuring cardiac mechanical dispersion may detect patients with significant CAD. This simple technique for detection of significant CAD by evaluating cardiac mechanical dispersion can increase the accuracy of currently used algorithms in assessments of patients having chest pain.

Conclusions

The dispersions of myocardial contraction durations and QTc intervals are correlated to the existence of significant CAD in patients without prior MI. Mechanical dispersion measured by echocardiographic strain has incremental value on GLS, WMSI, QTc interval, and conventional risk factors for significant CAD identification.

Limitations

The major limitations of this study are that it is a single center-based experience and may be limited by the small number of patients. The mechanical dispersion utilization in diagnosing significant CAD without involving LAD is still unclear, as all patients in the current study got significant LAD obstruction. There is a need for larger studies to clarify the predictive role of myocardial mechanical dispersions in patients without selection.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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