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Echocardiography Quantified Myocardial Strain – a Marker of Global and Regional Infarct Size that Stratifies Likelihood of Left Ventricular Thrombus

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Introduction

Among patients with acute myocardial infarction (MI), extent of myocyte necrosis is a key determinant of adverse remodeling – including contractile dysfunction and left ventricular thrombus (LVT).^{1–3} Echocardiography (echo) is widely used post-MI, but conventional approaches can be limited for estimation of MI burden and occasionally miss LV thrombi detected by more sensitive techniques such as cardiac magnetic resonance (CMR).^{4–6} Data have also shown MI pattern to strongly impact risk for LVT independent of LV dysfunction and chamber remodeling.^{1, 7, 8} Anterior MI is known to markedly increase risk for LVT, possibly due to alterations in LV flow pattern. Our group and others have recently used CMR to demonstrate LVT to be strongly linked to apical MI size even among anterior MI patients without advanced global LV dysfunction or aneurysm.⁸ Given that LVT provides a substrate for embolic events and a rationale for anticoagulation, improved echo-based assessment of risk factors that predispose for post-MI thrombus is of substantial importance.

Echo strain imaging provides a novel means of quantifying subtle alterations in myocardial deformation. Prior studies have shown strain to improve detection of impaired LV function in patients with coronary artery disease (CAD) as compared to visual wall motion or quantitative LV ejection fraction (LVEF).^{9–11} Strain has also been shown to yield improved agreement with volumetric chamber quantification as provided by well-validated techniques such as CMR.^{12, 13} Pilot studies have shown global strain to moderately correlate with MI size,^{14, 15} although these studies have been limited by small sample size and wide variations in interval between echo and CMR. Moreover, regional strain impairment has yet to be

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tested against CMR quantified regional MI size and functional indices. Uncertainty also exists as to whether the additive value of strain quantification requires dedicated image acquisition, or whether incremental utility can be gained via post processing of routine (non-tailored) echo datasets. More broadly, the impact of global or regional strain impairment on risk for LVT has yet to be studied.

This study tested utility of strain for assessment of MI size and LVT among anterior MI patients who prospectively underwent echo and CMR within a narrow interval. For this study, strain analysis was solely based on post-processing of routine echo datasets – acquisition was in no way tailored for the purpose of strain optimization. Aims were two-fold: (1) to assess echo-derived longitudinal strain as a marker of LV dysfunction and MI as quantified via CMR, and (2) to test the incremental utility of strain – measured by post processing of routine echo – for stratification of risk for post-MI LVT.

Methods

Study Population

The study population consisted of patients with anterior ST elevation MI (STEMI) (left anterior descending culprit vessel occlusion) enrolled in a prospective study (clinical trial # NCT00539045) focused on post-MI remodeling.⁸ In all patients, echo and CMR were performed within a narrow interval (72 hours) at a target of 1-month post-MI. Demographic, clinical and laboratory indices were collected at time of study enrollment, inclusive of CAD risk factors and MI treatment strategies. Electrocardiographic quantification of infarct size using Q-wave area was performed in accordance with previously validated methodology.¹⁶ Initial patient recruitment and image acquisition was performed between 9/2006 and 9/2014 at Weill Cornell Medical College (NY, NY). For the current study, available primary digital echo images were retrieved from data archives and re-analyzed (blinded to CMR and demographic indices) for the specific purpose of testing incremental utility of myocardial strain as a marker of infarct size and thrombus. This study was conducted with approval of the Weill Cornell Institutional Review Board.

Imaging Protocol

Echocardiography—Transthoracic two-dimensional echos as well as Doppler indices were acquired in accordance with ASE guidelines using commercially available equipment (General Electric Vivid 7) equipped with 3.5 MHz transducers. Parasternal long and short axis as well as conventional apical (2-, 3-, 4- chamber) views were acquired during breath hold and archived in cine loop format from a minimum of 3 consecutive heart beats.

CMR—CMR was performed using 1.5 Tesla scanners (General Electric [Waukesha, WI]). Cine-CMR employed a steady-state free precession pulse sequence; delayed enhancement (DE-) CMR was performed 10–30 minutes post-gadolinium (0.2 mmol/kg) via an inversion recovery sequence.¹⁷ Cine- and DE-CMR were acquired in matched contiguous LV short-axis (mitral annulus through apex) and long-axis (2-, 3-, 4-chamber) images. DE-CMR included standard (TI=250–350msec) imaging for assessment of MI, and “long TI” imaging (TI=600msec) for dedicated identification of LVT.^{1, 18, 19}

Image Analysis

Echo and CMR were each analyzed (at time of acquisition) for conventional indices of LV function, MI size, and thrombus by experienced (ACC/AHA level III) physicians using previously validated methods^{20–22}. Echo strain quantification was performed via retrieval analysis of stored images, which were queried from image archives and re-analyzed for the purpose of this study: Strain analysis was performed blinded to prior echo and CMR results, as well as clinical history.

Echocardiography—Longitudinal strain (reported as absolute values) was quantified in (2-, 3-, 4- chamber) long axis orientations, for which images were acquired with frame rates > 50 Hz. Automated speckle tracking analysis was performed on a frame-by-frame basis using vendor independent software (Cardiac Performance Analysis, TomTec [Unterschleissheim, Germany]). Regional strain was determined using a 16-segment model. Global longitudinal strain (GLS) was calculated as a weighted average of all LV segments. Segments that could not be evaluated (e.g. due to poor endocardial definition with resultant inaccurate border tracking) were excluded from analysis. Abnormal GLS was defined as <17%.

LV chamber size and function was quantified based on linear LV dimensions, for which measurements were performed in accordance with ASE guidelines using methods previously applied in large-scale population-based research.^{23, 24} Regional wall motion was scored in segments corresponding to strain indices, for which segmental contraction was graded as follows: 0=normal; 1=mild hypokinesia; 2=moderate hypokinesia; 3=severe hypokinesia; 4=akinesia; 5=dyskinesia.

CMR—Global LV geometry and function was quantified on cine-CMR based on planimetry of end-diastolic and end-systolic chamber volumes, which yielded both LVEF and stroke volume. CMR regional deformation was quantified using a previously validated automated algorithm for LV volumetric segmentation,^{25–27} which was used to measure temporal and geometric patterns of LV deformation at equidistant landmarks in the basal, mid, and apical LV. MI size was measured on DE-CMR. In accordance with established methods: Infarct transmural extent was graded on a 5-point scale for each affected LV segment (0=no hyperenhancement; 1=1–25%; 2=26–50%; 3=51–75%; 4=76–100%), and global infarct size (% LV myocardium) was calculated by summing segmental scores and dividing by the total number of regions.^{1, 28} LVT on DE-CMR was identified in accordance with previously validated methods, for which it was defined as an LV mass with avascular tissue properties on post-contrast inversion-recovery imaging via selective nulling of avascular tissue.^{1, 18, 19}

Statistical Methods

Continuous variables were compared between groups using Student's t-test (expressed as mean ± standard deviation); correlation coefficients and linear regression analyses were used to test magnitude of associations between variables. Categorical variables were compared using Chi-square or, when fewer than 5 expected outcomes per cell, Fisher's exact test. Receiver operating characteristic (ROC) analysis was used to evaluate diagnostic performance of echo parameters for detection of thrombus. Two-sided $p < 0.05$ was

considered indicative of statistical significance. Statistical calculations were performed using SPSS 24.0 (SPSS Inc. [Chicago, IL]).

Results

Population Characteristics

The population comprised 74 patients who underwent echo and CMR one month (28 ± 7 days) post STEMI as part of a prospective research protocol: All patients had echo and CMR within 72 hours (99% same day). For the current study, digitally available echoes were retrieved from imaging archives and specifically re-analyzed (blinded to initial data) to test the additive value of GLS for stratification of infarct burden and thrombus. 90% of patients had adequate echo image quality for assessment of strain in all 16 LV segments – in the remaining 10%, a mean of 8 ± 4 segments were evaluable. Figure 1 provides a representative example of strain analysis.

Table 1 details population characteristics in relation to normal or impaired GLS. As shown, GLS was impaired in over two-thirds (72%) of the population: Patients with and without impaired GLS were similar with respect to age, gender, and cardiovascular risk factors. Whereas chest pain to revascularization interval was longer among patients with impaired GLS ($p=0.03$), groups were otherwise similar with respect to revascularization strategy and medication regimen (all $p=NS$).

GLS in Relation to Global Infarct Burden and Dysfunction

GLS was examined in relation to MI size as measured by both conventional (enzymatic and ECG) indices, as well as CMR. As shown in Table 2, GLS differed in relation to global MI size as measured via multiple approaches: CMR-quantified MI size was 2.5 fold larger among patients with abnormal GLS, paralleling 2.6–3.1 fold differences in Q wave size and peak CPK respectively (all $p < 0.002$). When assessed as a continuous variable, GLS moderately correlated with extent of myocardial injury; correlation was highest with CMR quantified MI ($r=0.66$; $p<0.001$), but also significant for Q wave size ($r=0.44$; $p=0.001$) and enzymatic indices ($r=0.52$; $p<0.001$). As shown in Figure 2, GLS decreased stepwise in relation to population-based quartiles of CMR-evidenced MI size ($p<0.001$); mean GLS was almost two fold higher among those in the lowest versus highest quartile of MI.

Table 2 also demonstrates that GLS was associated with MI size, LV contractile impairment, and adverse chamber remodeling. Mean LVEF on cine-CMR was 17 points lower among patients with abnormal GLS, reflecting a disproportionate increase in LV end-systolic volume (94 ± 37 vs. 54 ± 20 ml; $p<0.001$) and a lesser increase in LV end-diastolic volume (170 ± 41 vs. 143 ± 33 ml; $p=0.01$). Table 2 also demonstrates that GLS yielded similar magnitude of difference with respect to echo-derived LVEF (mean $=14.9$ points), consistent with the notion that strain-based associations with LV remodeling indices are modality independent.

Regional Strain in Relation to Localized Infarct Transmurality and Dysfunction

Regional myocardial strain pattern was further tested in relation to corresponding indices of infarct size and contractile dysfunction. As shown in Table 3, LV strain was lower in the base, mid, and apical LV among patients with CMR-defined transmural MI in each respective territory (all $p < 0.05$). Similarly, Table 4 demonstrates that regional strain correlated with both cine-CMR quantified regional volumetric EF ($r = 0.53$ – 0.71) and echo ($r = 0.45$ – 0.69) defined visual wall motion scores.

Strain Based Risk Assessment for LVT

Among the overall study cohort, 18% (10/74) had LVT identified by the reference of delayed enhancement CMR, only 50% (5/10) of whom had LVT concordantly detected by echo. Patients with LVT had larger MI (26 ± 9 vs. $17 \pm 9\%$; $p = 0.004$) and lower LVEF (44 ± 7 vs. $52 \pm 13\%$; $p = 0.02$) on CMR than did patients without LVT.

Echo-derived global and regional strain pattern varied in relation to LVT: GLS yielded a 1.7-fold difference between patients with and without LVT (9 ± 4 vs. $14 \pm 5\%$; $p = 0.002$) whereas echo derived LVEF yielded non-significant differences between groups (45 ± 6 vs. $48 \pm 12\%$; $p = 0.3$). Regarding regional strain, Figure 3 (top) illustrates that patients with and without thrombus had similar strain in the LV base (89 ± 26 vs. $99 \pm 22\%$; $p = 0.23$) - in the mid LV, mean strain was 26% lower in patients with thrombus (70 ± 17 vs. $95 \pm 25\%$; $p = 0.002$) whereas in the apical LV, mean strain was 2-fold lower (29 ± 11 vs. $60 \pm 29\%$; $p < 0.001$). As shown in Figure 3 (bottom), visually scored apical wall motion was higher among patients with LVT (13 ± 5 vs. 8 ± 6 ; $p = 0.02$), paralleling differences in strain. However, whereas mid LV strain was significantly lower among thrombus-affected patients ($p = 0.002$), conventional assessment based on visual wall motion score demonstrated non-significant differences between groups (7 ± 4 vs. 6 ± 6 ; $p = 0.80$).

Figure 4 provides ROC curves for corresponding global (left) and regional (right) contractile indices. As shown, regional apical strain yielded best overall diagnostic performance for LVT (AUC=0.83, CI: 0.72–0.93, $p = 0.001$), which was slightly higher than that of visually assigned wall motion score in corresponding apical segments (AUC=0.73, CI: 0.58–0.88, $p = 0.02$). Similarly, GLS yielded higher overall test performance than did global wall motion score (AUC=0.78, CI: 0.65–0.90, $p = 0.005$ vs. AUC=0.66, CI: 0.52–0.79, $p = 0.12$) or LVEF (AUC=0.58, CI: 0.45–0.72, $p = 0.41$). Table 5 provides diagnostic performance of echo using strain and regional wall motion cutoffs optimized to provide maximal sensitivity for LVT. As shown, an apical strain score of 50 would have enabled all patients with thrombus to be referred for contrast echo and/or DE-CMR while allowing for unnecessary further testing to be avoided in nearly half (47%; 34/73) of cases. Additionally, at matched specificity, apical and total strain yielded greater sensitivity and accuracy for LVT than did regional wall motion scores, adding to the notion that regional and global strain provides incremental utility to conventional echo measures of LV function for the prediction of LVT.

Discussion

Primary findings of this study provide validation concerning utility of myocardial strain for assessment of post-MI remodeling. Key findings are as follows: First, among a well-characterized cohort of anterior MI patients undergoing echo and CMR within a narrow interval, GLS decreased in relation to LV injury severity as measured by chest pain duration, conventional enzymatic and ECG (Q wave) indices, and tissue-evidenced MI size measured by CMR (all $p < 0.05$). Magnitude of GLS impairment correlated with CMR LVEF ($r = 0.79$) and MI ($r = 0.66$, both $p < 0.001$), and was nearly 2-fold lower among patients in the largest (vs. smallest) quartile of CMR-evidenced MI size ($p < 0.001$). Second, regional strain impairment in the basal, mid, and apical LV corresponded to regional MI and functional indices: Patients with DE-CMR evidenced transmural MI in each territory had lower regional strain - volumetric EF measured by cine-CMR in each territory correlated with matched regional strain ($r = 0.53$ – 0.71 , $p < 0.001$). Third, GLS was nearly 2-fold lower (9 ± 4 vs. $14 \pm 5\%$; $p = 0.002$) among patients with LVT on CMR, reflecting regional decrements in mid and apical LV strain (both $p < 0.05$). Apical strain yielded higher diagnostic performance for CMR-evidenced LVT (AUC = 0.83, CI: 0.72–0.93, $p = 0.001$) than did visual wall motion score in corresponding LV segments (AUC = 0.73, CI: 0.58–0.88, $p = 0.02$). Similarly, total strain performed better as a diagnostic test for LVT (AUC = 0.78 CI: 0.65–0.90, $p = 0.005$) than did LVEF (AUC = 0.58, CI: 0.45–0.72, $p = 0.41$). Using diagnostic thresholds selected to yield optimal (100%) sensitivity, apical strain yielded higher specificity for LVT (54%) than did apical (44%) or total wall motion score (34%). Applied clinically, echo-based strain partitions selected to provide perfect sensitivity would have enabled all patients with thrombus to be appropriately referred for contrast echo and/or DE-CMR, while avoiding unnecessary additional testing in nearly half of the population.

To the best of our knowledge, this is the first study to test myocardial strain as a means of stratifying likelihood for LVT. Our observed association between global strain and thrombus is consistent with the general concept of impaired LV contractile function as a pro-thrombotic risk factor. Our finding of lower GLS in patients with thrombus, despite non-significant differences in LVEF (derived via linear indices), adds to growing literature supporting incremental utility of strain for quantitative assessment of LV performance.^{29–31} Our finding that strain decreased stepwise from LV base to apex among patients with thrombus is consistent with the general notion that apical dysfunction augments risk for thrombus. More broadly, while it is generally believed that longitudinal strain assessment is limited in the distal LV, our data showed apical strain impairment to correlate with both echo assigned visual wall motion score and CMR-quantified regional volumetric EF ($r = 0.69$ – 0.71 ; $p < 0.001$). Taken together, these data indicate that echo-evidenced strain impairment can be used to assess regional dysfunction after MI and support use of apical strain impairment as a surrogate to identify patients warranting further assessment for LVT via contrast-enhanced echo or CMR.

Our observed associations between strain and MI size build upon prior studies conducted in smaller cohorts. Among 39 patients with anterior MI, Grabka et al. reported GLS to correlate with CMR-evidenced MI size ($r = 0.68$, $p = 0.001$).¹⁵ That study, however, compared echo (performed 6 days post-MI) to CMR performed 3 months afterwards, prohibiting direct

comparisons of imaging tests performed at near identical post-MI time points. Similarly, Cimino et al. reported that GLS correlated with CMR-quantified LVEF ($r=0.88$, $p<0.001$) and that regional strain was lower in areas of DE-CMR evidenced transmural MI ($p=0.02$).¹⁴ However, that study was limited to 20 patients who underwent PCI within a narrow interval (6 hours) after MI, raising uncertainty as to whether observed associations between strain, LV function, and infarct transmural extent could be extrapolated to a general MI patients undergoing revascularization at variable time points after chest pain onset. Our results – demonstrating GLS to reflect MI size assessed clinically (chest pain to PCI time) as well as via conventional (ECG, enzymatic) and CMR indices among a broad cohort of post MI patients – provide confirmatory evidence of strain as a marker of LV injury.

This study builds on existing literature that has demonstrated DE-CMR to improve LVT detection as compared to echo alone.^{19, 32} Though its advantages are well described, CMR remains unfeasible as a screening tool for LVT for all comers with MI. In addition to substantial cost issues, CMR is not widely available in clinical practice and technical considerations (breath holds and supine positioning in an enclosed space) render CMR impractical for many post-MI patients. Findings of this study indicate that that non-contrast echo with strain analyses can be used to effectively stratify post-MI patients with the highest risk for LVT, thereby allowing for selection of at risk patients in whom further testing with contrast echo and/or DE-CMR is warranted.

It is important to recognize that no echos in our dataset were tailored for strain analysis. Despite this, dedicated post processing of archived data yielded strain results that correlated with MI size, and incrementally improved predictive utility of echo for LVT. The notion that advanced post processing can improve diagnostic capability of a given imaging test has been shown in our prior research.^{8, 25, 33} Among patients undergoing cine-CMR, we have shown automated segmentation to improve assessment of diastolic function and to stratify heart failure symptom status.^{25, 33} Among post-MI patients, we have shown that cine-CMR segmentation (i.e. regional apical EF) provides a structural risk factor for LVT.⁸ Our current study extends logically on this approach, demonstrating that longitudinal strain quantification, obtained through advanced post-processing of routine images, provides a marker of MI size that stratifies likelihood of LVT above and beyond conventional echo indices (ejection fraction and wall motion).

Several limitations should be noted. First, our study assessed patients with anterior MI so as to study a cohort known to be at high risk for LVT. While this cohort also allowed assessment of LV function and MI size, further research is needed to test whether our observed associations between echo-quantified strain and CMR indices are equivalent in patients with anterior or lateral MI. Second, it is possible that echo-based indices would have performed even better were circumferential strain quantified. On the other hand, circumferential strain typically requires dedicated image acquisition and thus may not be widely applicable to conventional echo data as routinely acquired clinically or in epidemiologic research studies. Finally, while our study tested echo strain in relation to CMR, it should be noted that prognostic utility of strain was not directly evaluated. Given that both CMR evidenced MI size and thrombus have been shown to stratify prognosis,

current data provides a logical basis for subsequent population-based research to test prognostic utility of myocardial strain in post-MI patients.^{34–36}

In conclusion, findings of this study demonstrate that longitudinal strain – as quantified via post-processing of routine, non-tailored, echo – provides a marker of MI size that incrementally improves stratification for CMR-evidenced LVT. Future studies are warranted to test whether magnitude of strain impairment differs between areas of infarction and ischemia, and whether strain-based indices enable prediction of LV remodeling and adverse prognosis among CAD population-based cohorts.

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Figure 1. Typical Example

Representative example of longitudinal echo-based strain analysis in a patient with anterior MI. Echo derived strain curves (acquired from 2-, 3-, 4-chamber apical long axis images) and resultant polar map shown on right. DE-CMR tissue characterization shown on left (green arrow denotes hyperenhancement [MI], yellow arrow denotes thrombus). Note progressive strain impairment in basal → mid → apical LV, corresponding to transmural MI pattern as well as presence of LV thrombus.

Figure 2. Global Longitudinal Strain in Relation to Myocardial Infarct Size
GLS (mean±standard deviation) among population-based quartiles partitioned based on CMR-evidenced MI size. Note stepwise decrements in GLS, paralleling increased MI size ($p<0.001$).

Figure 3. Regional Strain and Wall Motion Scores Stratified Based on Left Ventricular Thrombus

Regional LV strain (top) and corresponding visual wall motion scores (bottom) among patients with and without LV thrombus (data reported as mean±standard deviation). Note that mean apical strain was 2-fold lower among patients with LV thrombus (29 ± 11 vs. $60\pm29\%$; $p<0.001$), paralleling differences in apical wall motion score (13 ± 5 vs. 8 ± 6 ; $p=0.02$).

Figure 4. Receiver Operating Characteristics Curves

ROC curves for global (left) and regional (right) strain and wall motion score in relation to LV thrombus as identified by DE-CMR. As shown, regional apical strain yielded highest overall diagnostic performance for DE-CMR evidenced LV thrombus.

Table 1

Clinical and Imaging Characteristics

	Overall (n=74)	Abnormal GLS (n=53)	Normal GLS (n=21)	p
CLINICAL				
Age (year)	54 ± 11	55 ± 12	52 ± 9	0.34
Male gender	68 (92%)	49 (93%)	19 (91%)	1.0
Body surface area	2.0 ± 0.25	2.0 ± 0.26	2.0 ± 0.22	0.20
CAD risk factors				
Hypertension	35 (47%)	25 (47%)	10 (48%)	0.97
Hypercholesterolemia	34 (46%)	25 (47%)	9 (43%)	0.74
Diabetes mellitus	18 (24%)	13 (25%)	5 (24%)	0.95
Tobacco use	20 (27%)	14 (26%)	6 (29%)	0.85
Family history	23 (31%)	15 (28%)	8 (38%)	0.41
Prior myocardial infarction	8 (11%)	7 (13%)	1 (5%)	0.43
Prior coronary revascularization	9 (12%)	8 (15%)	1 (5%)	0.43
Cardiovascular medications				
Beta-blocker	73 (99%)	52 (98%)	21 (100%)	1.0
ACE-inhibitor/Angiotensin receptor blocker	54 (73%)	40 (76%)	14 (67%)	0.44
HMG CoA-reductase inhibitor	70 (95%)	52 (98%)	18 (86%)	0.07
Antithrombotic medications				
Aspirin	72 (97%)	51 (96%)	21 (100%)	1.0
Warfarin	6 (8%)	4 (8%)	2 (10%)	0.66
Thienopyridine	67 (91%)	47 (89%)	20 (95%)	0.67
MI treatment strategies				
Primary PCI	60 (81%)	44 (83%)	16 (76%)	0.52
Primary thrombolysis	13 (18%)	9 (17%)	4 (19%)	1.0
Intra-aortic balloon pump	14 (19%)	12 (23%)	2 (10%)	0.32
Chest pain to revascularization interval (h)	12.3 ± 13.2	13.9 ± 14.5	8.1 ± 8.1	0.03

Bold font corresponds to p value <0.05

Table 2

Global Strain in Relation to Global Infarct Size and Contractile Function

	Overall	Abnormal GLS	Normal GLS	P	R	P
Infarct size						
Creatinine phosphokinase	2427 ± 2266	3073 ± 2427	981 ± 695	<0.001	0.52	<0.001
Q wave area	3598 ± 2843	4285 ± 2850	1636 ± 1732	0.002	0.44	0.001
Myocardial infarct size						
Myocardial infarct size (% LV)	18.0 ± 9.7	21.6 ± 8.5	8.8 ± 5.9	<0.001	0.66	<0.001
Infarct transmuralty (% LV)	19.4 ± 12.5	23.7 ± 11.1	7.7 ± 7.7	<0.001	0.62	<0.001
Global LV structure and contractile function						
Cardiac magnetic resonance						
Ejection fraction (%)	50.6 ± 12.2	45.8 ± 10.4	62.8 ± 6.7	<0.001	0.79	<0.001
Stroke volume (ml)	79.4 ± 17.0	75.5 ± 15.6	89.1 ± 17.0	0.001	0.47	<0.001
End-diastolic volume (ml)	162.2 ± 40.3	169.7 ± 40.8	143.4± 32.7	0.01	0.41	<0.001
End-systolic volume (ml)	82.8 ± 37.2	94.1 ± 36.5	54.2 ± 19.8	<0.001	0.65	<0.001
Myocardial mass (gm)	141.8 ± 34.1	142.5± 34.5	140.0 ± 34.0	0.78	0.14	0.25
Echocardiography						
Ejection fraction (%)	47.2 ± 11.0	42.9 ± 9.6	57.8 ± 6.1	<0.001	0.70	<0.001
End-diastolic diameter (cm)	5.7 ± 0.42	5.8 ± 0.42	5.6 ± 0.40	0.03	0.34	0.004
End-systolic diameter (cm)	4.3 ± 0.74	4.5 ± 0.77	3.9 ± 0.39	0.001	0.50	<0.001
Myocardial mass (gm)	187.1 ± 41.0	188.4 ± 44.7	183.9± 30.3	0.67	0.25	0.04

Table 3

Regional Strain in Relation to Regional Infarct Transmurality

	Overall	Regional Transmural Infarct +	Regional Transmural Infarct –	p
Regional strain				
Base	98.1 ± 23.0	77.7 ± 17.8	100.0 ± 22.6	0.02
Mid	92.0 ± 25.1	80.5 ± 18.8	112.8 ± 21.4	<0.001
Apex	56.1 ± 28.9	50.2 ± 27.7	83.0 ± 16.5	<0.001

Table 4

Regional LV Strain in Relation to Conventional Contractility Indices

	Correlation coefficient		Linear regression equation
Basal left ventricle			
	r	p	
Cine-CMR			
Volumetric ejection fraction	0.53	<0.001	y = 0.21x + 32.9
Echocardiography			
Regional wall motion score	0.45	< 0.001	y = −0.08x + 10.6
Mid left ventricle			
Cine-CMR			
Volumetric ejection fraction	0.60	<0.001	y = 0.33x + 20.9
Echocardiography			
Regional wall motion score	0.68	<0.001	y = −0.15x + 20.3
Apical left ventricle			
Cine-CMR			
Volumetric ejection fraction	0.71	<0.001	y = 0.45x + 17.5
Echocardiography			
Regional wall motion score	0.69	<0.001	y = −0.12 x + 13.1

Table 5

Echocardiography Diagnostic Performance for Detection of LV Thrombus

	Sensitivity	Specificity	Accuracy	PPV	NPV
Apical strain [*]	100% (10/10)	54% (34/63)	60% (44/73)	26% (10/39)	100% (34/34)
Total strain [§]	100% (10/10)	48% (31/64)	55% (41/74)	23% (10/43)	100% (31/31)
Apical wall motion score [°]	100% (10/10)	44% (28/64)	51% (38/74)	22% (10/46)	100% (28/28)
Total wall motion score [¶]	100% (10/10)	34% (22/64)	43% (32/74)	19% (10/52)	100% (22/22)

^{*} cutoff 50 (for aggregate apical segments)
[§] cutoff < 294.1 (for aggregate 16 segments)
[°] cutoff 3.0 (for aggregate apical segments)
[¶] cutoff 5.0 (for aggregate 17 segments)