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Potential Prediction of Myocardial Viability Using Strain/Strain Rate at Low-Dobutamine Stress Echocardiography on a Segment-by-segment Basis

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ABSTRACT

Background: It is crucial to determine if the myocardium with highly impaired function is irreversibly harmed or reversibly dysfunctional in acute myocardial infarction. Although dobutamine-induced wall motion improvement is suitable for assessing viability, it is vulnerable to significant inter- and intra-observer variability because it is subjective.

Objectives: It was determined whether peak longitudinal strain (PLS) and peak longitudinal strain rate (PLSR) in individual myocardial segments with low dose dobutamine stress echocardiography (LDDSE) could diagnose myocardial viability in patients with acute ST segment elevation myocardial infarction using delayed contrast-enhanced cardiac magnetic resonance (DE-CMR) as a reference (STEMI).

Patients and Methods: This study included 60 individuals who had been in the hospital for at least three months after acute myocardial infarction. LDDSE with delayed contrast-enhanced cardiac magnetic resonance and offline deformation indices analysis was performed on all patients.

Results: There were 268 segments having significant resting wall motion anomalies available for the final analysis. Dobutamine-induced peak longitudinal strain was greater in viable than non-viable segments in all investigated individual myocardial segments (< 0.001 for mid inferoseptum, p=0.001 for mid inferolateral, and < 0.001 for all other segments). Furthermore, dobutamine-induced peak longitudinal strain rate was significantly lower in non-viable segments compared to viable segments within the studied individual myocardial segments (p< 0.001 for basal antro-septum, < 0.001 for apical inferior, <0.001 for mid inferolateral, < 0.001 for mid antrolateral, <0.001 for mid inferoseptum, <0.001 basal anterior, <0.001 for basal inferolateral, <0.001 basal inferoseptum, and < 0.001 for all other segments).

Conclusion: Dobutamine-induced peak longitudinal strain and strain rate could predict myocardial viability segment by segment in those suffering from acute ST segment elevation myocardial infarction.

Keywords: Myocardial viability, Strain rate, Strain, Acute myocardial infarction, Stress Echocardiography

INTRODUCTION

Because reperfusion therapy saves viable myocardium only after an AMI, it is critical to determine if a myocardium with considerably reduced function is permanently damaged or reversibly malfunctioning. Relying on the factors which promote ischemia-induced myocardial dysfunction, the time it takes to regain myocardial function following reperfusion might range from a few hours to many months (stunning, hibernation, or subendocardial infarction) (1). Low-dose dobutamine stress echocardiography (LDDSE) offers the advantages of being practicable, safe, and affordable, as well as having good diagnostic and prognostic accuracy. It is commonly used to assess viability after AMI. Wall motion improvement during LDDSE is specific for predicting viability, but because it is subjective, it is prone to substantial inter-and intra-observer variability [(2)].

Deformation rate (strain rate) and myocardial deformation (strain) permit a multi-dimensional evaluation of myocardial mechanics (radial, circumferential function, and longitudinal), as well as the detection of modest regional function problems that do not compromise global left ventricular function. This could be because strain rate imaging has a lower load dependence and gives a more accurate picture of contractility. Global cardiac displacement has no influence, and the anchoring impact of nearby wall segments obstructs two-dimensional visual inspections. Deformation indices are more objective and quantitative measures for assessing cardiac contraction, and, therefore may be more useful than wall motion analyses [(3)].

To overcome subjectivity of dobutamine stress echocardiographic (DSE) and making benefit from the ability of speckle tracking (STE) to detect subtle myocardia dysfunction, some researchers looked at the possibility of adding strain rate and strain depending on speckle tracking to DSE's protocol. They observed that STE offers an additional benefit in detecting mild myocardial damage and coronary artery disease in those suffering from cardiovascular risk factors and even the extent of myocardial scarring following either an acute or chronic myocardial infarction, and in detecting microvascular injury. Although the results were promising, the absence of uniformity and reference cutoffs makes it difficult to use in clinical settings [(4)]. More and more studies are required to establish reference cutoffs.

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The study used delayed contrast-enhanced cardiac magnetic resonance (DE-CMR) as a reference to determine whether LDDSE can detect myocardial viability in those suffering from acute ST segment elevation myocardial infarction utilizing peak longitudinal strain rate (PLSR), as well as peak longitudinal strain (PLS) in individual myocardial segments (STEMI).

PATIENTS AND METHODS

The current research was a prospective follow-up investigation. This study involved 60 patients who presented to Aswan Heart Center (AHC) and Aswan University Hospitals for the assessment of myocardial viability at least 3 months following acute STEMI and met the inclusion criteria. This research was carried out between March 2019 and June 2020. Using G*Power 3 software, an estimated minimum sample of 58 STEMI patients was required to identify an effect size of 0.2 in the mean PLS/PLSR between viable and non-viable segments with an error probability of 0.05 and 80 percent power on a two-tailed test. Individuals aged >18 years with STEMI who received fibrinolytic therapy in acute setting with no further early invasive intervention at least 3 months before presentation with some segmental wall motion abnormalities (SWMA) at the anatomical distribution of infarcted artery by 2-D echocardiography, were included. On the other hand, patients with previous MI, bundle branch block and atrial fibrillation, clinically obvious congestive heart failure, severe hemodynamic instability, early post-infarction unstable angina, mechanical complications of MI, technically inadequate echocardiographic imaging (more than two non-analyzable segments in the infarct zone), and significant valvular/congenital heart disease/any myocardial disease, other than ischemia, were excluded.

Study tools: Baseline data, comprising socio-demographics, hypertension, diabetes mellitus, smoking, and family history, were acquired from the participants or their parents. The participants were exposed to:

2-D speckle tracking strain and strain rate with LDDSE (Figure 1):

LDDSE was performed with a 3.5-MHz transducer on a Philips 4D (GE Healthcare, Horton, Norway). According to the recommendations of the American Society of Echocardiography (ASE) for Stress Echocardiography Performance, Interpretation, and Application 2007 (5), dobutamine infusions began at 2.5 ug/kg body weight/min for 3 minutes, then increased to 5 ug/kg body weight/min for another 3 minutes, and finally to 10 ug/kg each minute (maximum dose). During the picture capture, the dobutamine infusion rate was constant (10 ug/kg/min). Standard views were taken at the beginning and end of the infusion regimen. Beta-blockers were discontinued 48 hours prior to the stress and restarted afterward. Images were scanned and saved as cine-loop files for further study and analysis. Heart rate and blood pressure were monitored at baseline, 3 minutes, 6 minutes of dobutamine stress, and 3 minutes after the infusion ended. At the beginning and end of each step, a 12-lead ECG was collected.

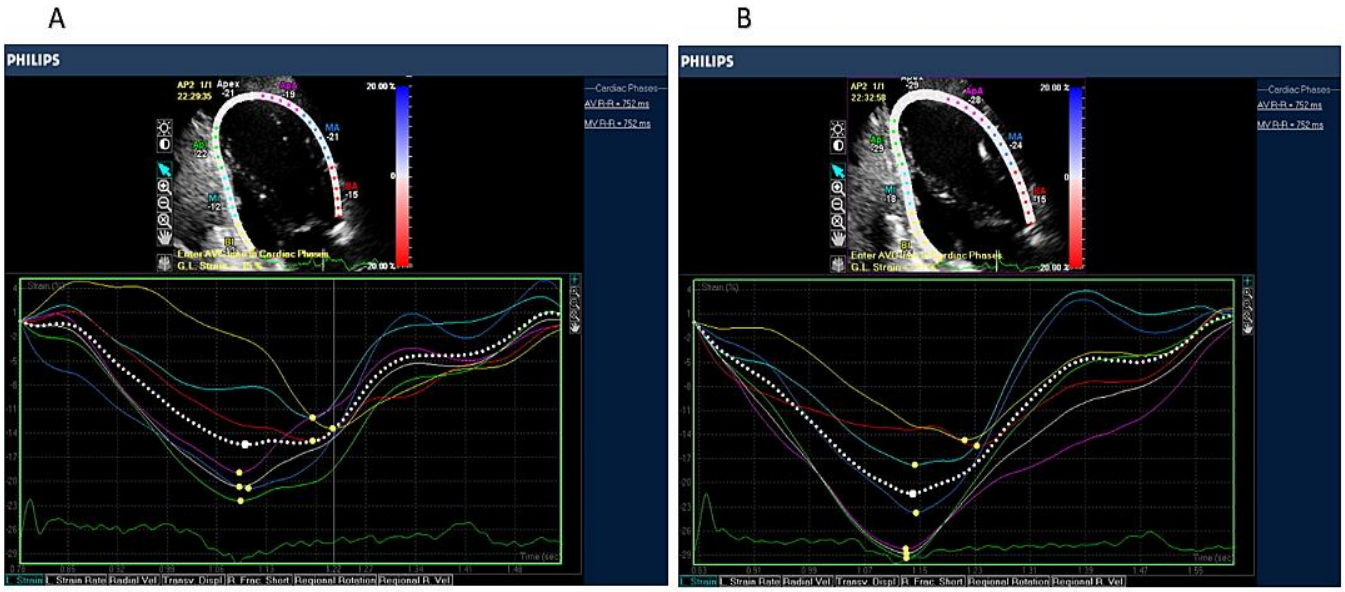

Figure (1): Speckle-tacking echocardiography was utilized to measure longitudinal myocardial strain in cases with right coronary artery disease (RCA). Apical two-chamber at rest (A) Apical two-chamber at low dosage dobutamine (B).

The 17-segment model was used to quantify myocardial strain and strain rate with speckle tracking echocardiographic (STE) at low dose dobutamine (10 ug/kg/min) and at rest independently for each segment. Longitudinal deformation (apical four chambers, apical three chambers, and apical two chambers) was measured using STE.

Only 17 segments with baseline substantial WMA (dyskinesia, akinesia, or marked hypokinesia) near the anatomical distribution of the infarcted artery were included in our investigation. In each view, the STE strain and strain rate were monitored during one cardiac cycle.

To obtain the results, a machine-generated frame was used with saved images from different viewpoints. Throughout the cardiac cycle, the software tracked speckles frame by frame. The machine first used automatic traces to obtain the final observation, and if the traces did not coincide with cardiac borders, they were manually changed to match. Then, the automated software-generated traces reflecting regional strain, and strain rate, were utilized to determine peak systolic strain, and strain rate. Finally, the LDDSE and speckle tracking analyses were carried out by a single observer (Aml Soliman), unaware of the DE-CMR results.

DE- CMR (Figure 2):

DE-CMR was conducted three months following the STEMI. A Siemens IRA 1.5 T system was used to obtain magnetic resonance pictures. A 17-segment LV model was employed for DE-CMR analysis. Short-axis, scouts, 2-chamber, 3-chamber, 4-chamber cine acquisitions, early gadolinium improvement (within the first 1-3 mins following contrast infusion) to find a microvascular obstruction indicating no LV thrombi as well as no-reflow, and late gadolinium enhancement (5:15 minutes after contrast infusion) to determine transmurality utilizing phase-sensitive inversion recovery sequences method. Viability is existent when transmurality is less than 50 percent of the affected segment's area, and it is missing when it is more than 50 percent (6).

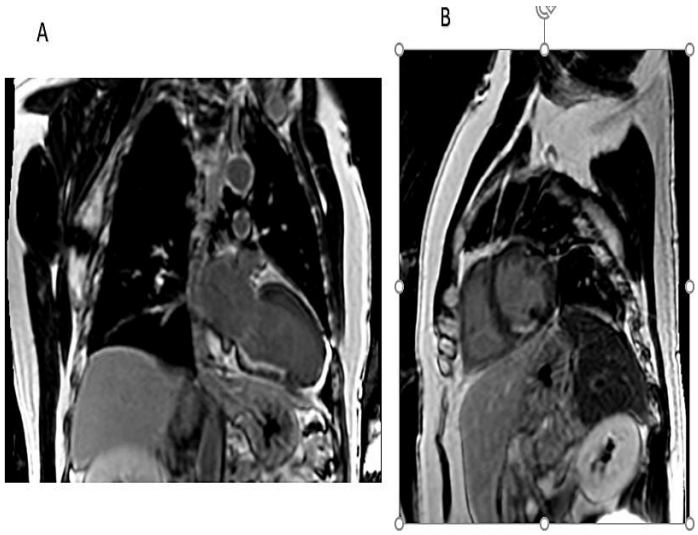

Figure (2): DE-CMR images in an individual with RCA cutoff diagram (the same individual in Figure 1). PSIR two-chamber (A). PSIR short axis (B).

Ethical consent:

This research was approved by the Medical Faculty, Aswan University Institutional Research Board (IRB) before the start of the study (IRB No. 222/3/18). The research complied with the World Medical Association Helsinki's Declaration. After receiving complete information about the study's objectives and methodology, all participants were asked to sign a written consent form, and their participation had no effect on the level of care provided. Confidentiality and anonymity were assured. The study was in line with the consort statement of the study execution ethics.

Statistical analysis

IBM-SPSS 24.0 (IBM-SPSS Inc., Chicago, IL, USA) was utilized for data analysis. Data were presented as median (Range), means (SD), and numbers (%). To compare means, the t-test was utilized. Validity data were calculated, comprising specificity, sensitivity, and negative and positive predictive values –PPV and NPV-. A Receiver Operating Characteristic curve (ROC) analysis was employed to assess the most appropriate cutoff values for various strain parameters. A p-value < 0.05 was significant statistically.

RESULTS

A total of 60 patients demonstrated at least 3 months following STEMI participated in the current study after fulfilling the inclusion/exclusion guidelines. In addition, the participants were treated with fibrinolytic therapy in acute setting with no further early invasive intervention, as summarized in **(Table 1).**

Table (1): Baseline characteristics of the studied sample

Variable	Category	n = 60
Age in years	• **Mean ± SD**	60.28 ± 9.9
	• **Median (Range)**	60 (40 – 77)
Sex	• **Male**	48 (80%)
	• **Female**	12 (20%)
DM	• **No**	38 (63.3%)
	• **Yes**	22 (36.7%)
HTN	• **No**	32 (53.3%)
	• **Yes**	28 (46.7%)
Smoking	• **No**	29 (48.3%)
	• **Yes**	31 (51.7%)
Family History	• **No**	44 (73.3%)
	• **Yes**	16 (26.7%)
Types of infarction	• **Anterior STEMI**	35 (58.3)
	• **Inferior STEMI**	22 (36.7%)
	• **Posterior STEMI**	3 (5%)
LVEF%	• **Mean ± SD**	45.62 ± 7.1
	• **Median (Range)**	46 (30 – 60)
LVEDD	• **Mean ± SD**	5.67 ± 0.7
	• **Median (Range)**	5.9 (4 – 7)
LVESD	• **Mean ± SD**	3.11 ± 0.7
	• **Median (Range)**	3.1 (2 – 5)

There were 278 segments available for study with resting substantial wall motion anomalies at the anatomical distribution of the infarcted artery. Due to insufficient image quality, seven segments could not be evaluated. No equivalent non-viable segments were not found for the basal anterolateral segments (3-seg.), and hence they were omitted from the final analysis. In all segments, DE-CMR image analysis was possible. As a result, there were 268 segments available for final analysis.

The distribution of non-viable segments vs. akinetic viable was illustrated in Figure (3). (as assessed by DE-CMR).

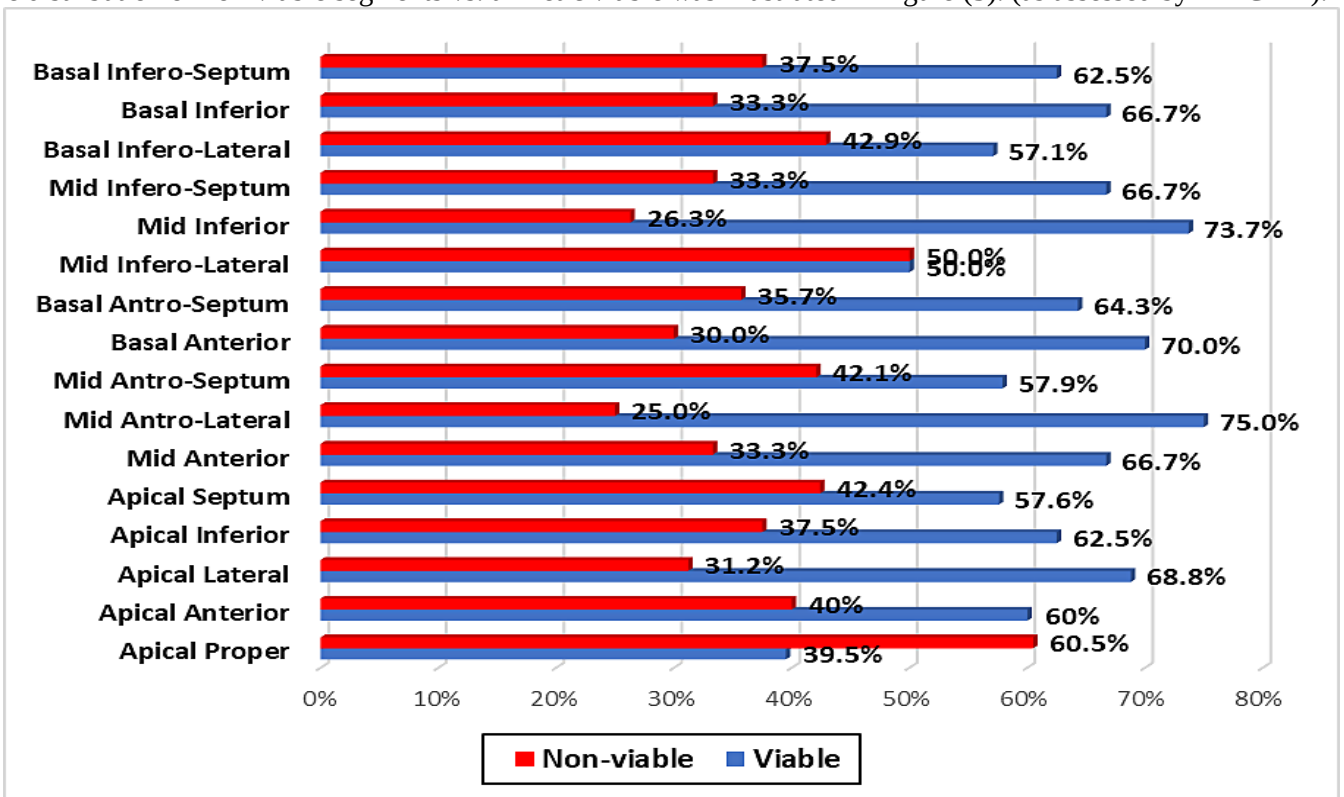

Figure (3): Non-viable and Viable segments distribution.

At rest and after a mild dosage of dobutamine, the average values of peak longitudinal strain (PLS) were statistically significantly low in non-viable compared to viable segments. Similarly, viable segments had significantly higher mean values of peak longitudinal strain rate (PLSR) at rest and following low dose dobutamine than non-viable segments. Furthermore, after dobutamine stress, the mean change in PLS (ΔPLS) was significantly lower in non-viable segments than in viable segments. Similarly, after dobutamine stress, the mean change in PLSR (ΔPLSR) was significantly lower in non-viable segments than in viable segments (Table 2).

Table (2): Comparison of various strain parameters by STE in non-viable and viable myocardial segments

Parameter	Viable (n = 161)	Non-Viable (n = 107)	*P-value*
Peak Longitudinal Strain (%)			
• **Mean ± SD**	-13.56 ± 2.6	-7.57 ± 1.7	< **0.001**
Peak Longitudinal Strain Rate			
• **Mean ± SD**	-0.76 ± 0.1	-0.53 ± 0.1	< **0.001**
Dobutamine Stress Peak Longitudinal Strain (%)			
• **Mean ± SD**	-18.49 ± 3.4	-7.70 ± 1.8	< **0.001**
Dobutamine Stress Peak Longitudinal Strain Rate			
• **Mean ± SD**	-0.97 ± 0.2	-0.54 ± 0.1	< **0.001**
Change in Peak Longitudinal Strain (%)			
• **Mean ± SD**	-4.67 ± 1.5	-0.26 ± 0.1	< **0.001**
Change in Peak Longitudinal Strain Rate (%)			
• **Mean ± SD**	-0.27 ± 0.1	-0.03 ± 0.01	< **0.001**

Furthermore, in all individual myocardial segments tested, dobutamine-induced PLS was considerably higher in viable versus non-viable segments (Table 3).

Table (3): Dobutamine-induced strain values in individual myocardial segments

Segment	Viable	Non-Viable	*P-value**
• **Apical-proper**	-19.07 ± 2.7	-7.87 ± 2.1	< **0.001**
• **Apical-anterior**	-17.06 ± 1.9	-8.67 ± 1.7	< **0.001**
• **Apical-lateral**	-18.82 ± 2.9	-8.20 ± 0.8	< **0.001**
• **Apical-inferior**	-17.80 ± 3.3	-7.83 ± 1.6	< **0.001**
• **Apical-septum**	-19.63 ± 2.6	-7.64 ± 1.3	< **0.001**
• **Mid-anterior**	-19.82 ± 1.7	-8.33 ± 2.3	< **0.001**
• **Mid-antrolateral**	-19.33 ± 1.5	-9.00 ± 0.1	< **0.001**
• **Mid-inferolateral**	-17.50 ± 2.4	-7.00 ± 1.4	**0.001**
• **Mid-inferior**	-18.71 ± 1.3	-6.80 ± 1.6	< **0.001**
• **Mid-infero-septum**	-18.50 ± 2.5	-7.50 ± 1.1	**0.006**
• **Mid-antro-Septum**	-19.36 ± 0.8	-8.63 ± 1.9	< **0.001**
• **Basal-anterior**	-18.14 ± 2.0	-6.67 ± 2.1	< **0.001**
• **Basal-inferolateral**	-17.75 ± 1.5	-6.67 ± 0.6	< **0.001**
• **Basal-inferior**	-18.00 ± 1.8	-5.17 ± 0.8	< **0.001**
• **Basal-infero-septum**	-16.80 ± 0.8	-7.00 ± 0.1	< **0.001**
• **Basal-antro-septum**	-17.44 ± 2.6	-7.60 ± 0.9	< **0.001**

Moreover, in all individual myocardial segments tested, dobutamine-induced PLSR was significantly greater in viable segments than in non-viable segments (Table 4).

Table (4): Dobutamine-induced strain rate in individual myocardial segments

Segment	Viable	Non-Viable	*P-value**
• **Apical-proper**	-1.05 ± 0.2	-0.49 ± 0.1	**< 0.001**
• **Apical-anterior**	-0.99 ± 0.2	-0.54 ± 0.2	**< 0.001**
• **Apical-lateral**	-0.91 ± 0.2	-0.55 ± 0.1	**< 0.001**
• **Apical-inferior**	-0.87 ± 0.2	-0.65 ± 0.1	**0.018**
• **Apical-septum**	-1.05 ± 0.2	-0.51 ± 0.1	**< 0.001**
• **Mid-anterior**	-1.02 ± 0.2	-0.58 ± 0.1	**< 0.001**
• **Mid-antrolateral**	-0.98 ± 0.2	-0.62 ± 0.1	**0.002**
• **Mid-inferolateral**	-0.95 ± 0.2	-0.57 ± 0.1	**0.030**
• **Mid-inferior**	-0.98 ± 0.1	-0.51 ± 0.1	**< 0.001**
• **Mid-infero-septum**	-0.99 ± 0.2	-0.47 ± 0.1	**0.015**
• **Mid-antro-Septum**	-0.95 ± 0.1	-0.61 ± 0.1	**< 0.001**
• **Basal-anterior**	-0.90 ± 0.1	-0.52 ± 0.2	**0.004**
• **Basal-inferolateral**	-0.92 ± 0.1	-0.49 ± 0.1	**0.005**
• **Basal-inferior**	-0.94 ± 0.1	-0.40 ± 0.1	**< 0.001**
• **Basal-infero-septum**	-0.84 ± 0.1	-0.49 ± 0.1	**0.003**
• **Basal-antro-septum**	-0.84 ± 0.2	-0.61 ± 0.1	**0.026**

ROC curve analysis was depicted for identification of the optimal cutoff with the highest accuracy in discriminating viable from non-viable myocardium for different stress parameters in all investigated individual myocardial segments. Cutoff values for the PLS extending from –11 to –18.5 % identified viability in apical segments, from –12 to –15% in mid- sectors, and from –11.5 to –12 % in basal segments, respectively (Table 5).

Table (5): Cutoff values for dobutamine-induced strain which indicate viability in individual myocardial segments

Segment	Cut-off	Sensitivity	Specificity	PPV	NPV
• **Apical-proper**	-18.5	80%	53%	63%	73%
• **Apical-anterior**	-13.0	100%	94%	94.5%	100%
• **Apical-lateral**	-11.0	81%	80%	90%	67%
• **Apical-inferior**	-12.5	80%	66%	80%	66%
• **Apical-septum**	-13.0	89%	85%	89%	85%
• **Mid-anterior**	-14.5	83%	83%	91%	71%
• **Mid-antrolateral**	-13.5	83%	100%	100%	67%
• **Mid-inferolateral**	-12.0	75%	100%	100%	80%
• **Mid-inferior**	-13.0	85%	80%	92%	67%
• **Mid-infero-septum**	-12.5	75%	100%	100%	67%
• **Mid-antro-Septum**	-15.0	81%	87%	90%	78%
• **Basal-anterior**	-12.0	71%	66%	83%	50%
• **Basal-inferolateral**	-11.5	75%	66%	75%	67%
• **Basal-inferior**	-10.5	75%	83%	90%	62.5%
• **Basal-infero-septum**	-11.5	80%	66%	80%	67%
• **Basal-antro-septum**	-11.5	77%	60%	78%	60%

Additionally, a cutoff value is from – 0.68 to – 1.3 s^{-1} for the PLSR identified viability in apical segments, from – 0.71 to – 0.78 s^{-1} in mid- segments, while a cutoff value is from – 0.66 to – 0.74% identified viability in basal segments (Table 6).

Table (6): Cutoff values for dobutamine-induced strain rate that predict viability in individual myocardial segments

Segment	Cut-off	Sensitivity	Specificity	PPV	NPV
• **Apical Proper**	-1.3	86%	87%	81%	90%
• **Apical Anterior**	-0.79	88%	91%	94%	84%
• **Apical Lateral**	-0.70	90%	80%	90%	67%
• **Apical Inferior**	-0.85	90%	83%	90%	83%
• **Apical Septum**	-0.68	84%	92%	94%	81%
• **Mid Anterior**	-0.76	75%	66%	81%	57%
• **Mid Antrolateral**	-0.76	66%	50%	80%	33%
• **Mid Inferolateral**	-0.74	75%	50%	60%	67%
• **Mid Inferior**	-0.71	78%	60%	84%	50%
• **Mid Inferoseptum**	-0.72	100%	100%	100%	100%
• **Mid Antro-Septum**	-0.78	90%	75%	83%	85%
• **Basal Anterior**	-0.72	85%	67%	86%	67%
• **Basal Inferolateral**	-0.74	75%	66%	75%	67%
• **Basal Inferior**	-0.66	83%	67%	83%	66%
• **Basal Inferoseptum**	-0.68	100%	66%	83%	100%
• **Basal Antro-Septum**	-0.72	88%	80%	88%	80%

DISCUSSION

MI is frequently complicated with congestive heart failure (CHF). That has a high morbidity and mortality rate [(7)]. Coronary revascularization in those cases can result in clinical and outcome improvement, as well as reversal of LV remodeling, giving rise to the concept of viable myocardium, which is used to identify patients whose recovery of LV function and improved prognosis outweighs the risk of revascularization [(7)].

CMR, LDDSE, single-photon emission computed tomography (SPECT), and positron emission tomography (PET), are among the imaging modalities presently approved to determine cardiac viability. The use of CMR is limited by its cost, availability, and lengthy study, whereas PET and SPECT are also limited by their costs, availability, and radiation exposure. The subjectivity of LDDSE underestimates its value [(8,9)]. Adding speckle tracking-based PLS and PLSR to LDDSE conventional protocol is applicable and can overcome its subjectivity, increase reproducibility, and hence its value in predicting myocardial viability [(10)].

In the future, dobutamine-induced PLS and PLSR by being available, reproducible, accurate, radiation and contrast free, and inexpensive, could be the standard imaging modality to assess myocardial viability, especially in developing countries where the cost and availability of other modalities are serious problems. So, to overcome subjectivity and improve accuracy, some research investigated adding deformation indices (depending on speckle tracking or tissue Doppler) to the low dose dobutamine stress echocardiographic (LDDSE) technique (11). They compared deformation indices with either LDDSE or PET.

STE based deformation indices were adopted in the current study as they have no angel dependency [(11-13)]. DE-CMR was approved to be the gold standard for measuring myocardial viability [(6)]. In the current study, deformation indices were compared against DE-CMR because of its availability in the study area.

Primary percutaneous coronary intervention (PCI) is currently the recommended reperfusion method for individuals with an acute STEMI. However, fibrinolytic therapy is still used in many areas, including the study area, either due to the high cost of primary PCI or a lack of local expertise. All current study cohort are patients with STEMI that were treated with fibrinolytic therapy in acute setting with no further early invasive intervention as we thought they would benefit the most from revascularization if they have viable myocardium.

The current study findings revealed that post dobutamine PLS and PLSR were significantly higher in viable segments. **Gong et al. (14)** observed that longitudinal strain rate (LSR), and longitudinal strain (LS) at rest, as well as LDDSE, were independent predictors of viable myocardium in 42 hospitalized cases with AMI and left ventricular systolic failure (left ventricular ejection fraction< 50 percent).

Moreover, we found that post dobutamine PLS and PLSR were significantly high in each studied individual myocardial segment in a viable group. This is in line with **Ismail *et al.* [(2)],** who were the first and only to identify viability on individual segment basis, but these findings were more consistent. They evaluated 60 patients who underwent a myocardial viability assessment at least four weeks after a STEMI. They found that dobutamine-induced S and SR were considerably higher in viable segments than in non-viable segments. This was true for the majority of individual cardiac segments (10 out of 16 for S and 11 out of 16 for SR). In contrast, they used a tissue Doppler-based strain, strain rate, and a 16-segment model.

The current investigation also found that viable segments had much higher resting PLS and PLSR than non-viable segments. These findings contradicted those of Chan et al. (15). They studied 80 cases with chronic ischemic LV dysfunction aged 63 years and found that subendocardial infarct (viable) segments had significantly lower PLS and PLSR than normal myocardium. However, insignificant differences were observed between subendocardial (viable) and transmural infarct segments (non-viable). These results were in line with **Bansal *et al.* [16].** They examined 55 individuals with left ventricular systolic dysfunction (left ventricular ejection fraction < 0.45) and ischemic heart disease who had dobutamine stress echocardiography for myocardial viability assessment and later had their hearts revascularized. They discovered that resting peak longitudinal S and SR were much higher in viable segments, but that was limited in the anterior circulation, unlike humans.

Another important finding was that both ΔPLS and ΔPLSR with low-dose dobutamine were significantly high in viable segments. The findings were inconsistent with **Bansal *et al.* [16],** who observed no significant difference between non-viable and viable segments as regard both changes, and with **Sharma *et al.* [17],** who found a significant increase in SR but not S with low dose dobutamine. The cutoff value for ΔPLS that best discriminate viable from non-viable myocardium was lower in our study (-1.50% vs -1.9%) with higher sensitivity and specificity, whereas the cutoff value for ΔPLSR was similar in both studies (-0.20 s^{-1}) with higher sensitivity but lower specificity in our study. Likewise, **Hoffmann *et al.* [18]** found that only ΔPLSR at LDDSE is significantly lower in non-viable compared to viable segments, but they did not determine cutoff value that best discriminate non-viable from viable myocardium.

Limitations of the study:

The current research findings are challenging to generalize due to the non-randomized method used. STE is a restriction for this project because it relies on image quality and frame rate. Another disadvantage is that the low frame rate makes it difficult to characterize regional myocardial motion accurately and reduces the regional strain map's overall temporal resolution. Unlikely, image resolution is dependent on the scan line density that is reduced by the increase in the frame rate. Also, inter-observer variability was not assessed, which is considered as one of the study limitations. As the myocardial infarction scar extends from endocardium to epicardium depends on transmurality of infarction, so layer-specific strain may be more beneficial in detecting the exact extent of scar. This could be another limitation of this study.

CONCLUSION

In those suffering from acute ST segment elevation myocardial infarction, dobutamine-induced peak longitudinal strain and strain rate predict myocardial viability segment by segment.

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