The International Journal of Cardiovascular Imaging (2019) 35:339–346
https://doi.org/10.1007/s10554-018-1498-7

ORIGINAL PAPER

The predictive value of global longitudinal strain on late infarct size in patients with anterior ST-segment elevation myocardial infarction treated with a primary percutaneous coronary intervention

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Received: 3 August 2018 / Accepted: 7 November 2018 / Published online: 14 November 2018

Abstract
Late infarct size (IS) after ST segment elevation myocardial infarction (STEMI) is a determinant of subsequent mortality. Late Gadolinium enhancement in cardiac magnetic resonance imaging (LGE-CMRI) is the gold standard for IS measurement, however, it is not readily accessible in many areas. We aimed to evaluate the value of early baseline 2D-echocardiographic global longitudinal strain (GLS) for the prediction of late IS after STEMI. From October 2017 to July 2018, we studied 100 patients with their 1st anterior STEMI treated with primary percutaneous coronary intervention. Baseline GLS calculation was performed within 48 h of admission. In addition, the average value of the nine segments supplied by the LAD was assessed separately (anterior GLS). Infarct size was assessed 3 months later using LGE-CMRI, and large infarcts were defined as ≥ 20% LV myocardium covered by scar. Based on CMRI, we defined two groups; 57 patients with large infarcts (group I) and 43 patients with small infarcts (group II). Both groups were matched in all baseline demographics and risk factors. There was a good and significant correlation between GLS and late IS ($r = -0.840$, $P < 0.001$). This correlation was even higher for anterior GLS ($r = -0.867$, $P < 0.001$). ROC analysis showed a cut-off point of GLS (− 13%) that identified large late IS with a sensitivity and specificity of 66.7% and 88.4% respectively (AUC = 0.85). For anterior GLS, the cut-off point was − 9.6% (Sensitivity 94%, specificity 86%, AUC = 0.9). We concluded that baseline GLS significantly predicts late IS after anterior STEMI.

Keywords Anterior STEMI · Cardiac magnetic resonance · Global longitudinal strain · Infarct size

Introduction

Despite recent advances and the widespread adoption of Primary Percutaneous Coronary Intervention (PPCI) as the modality of choice for emergent reperfusion in STEMI, some irreversible damage to the myocardium supplied by the infarct-related artery (IRA) is unavoidable in many patients [1]. Infarct size (IS) is one of the major determinants for short- and long-term major adverse cardiac events (MACEs) after STEMI [2]. Therefore, a great focus has been directed in recent years towards evaluating various techniques for IS estimation. Generally, late gadolinium enhancement (LGE) in cardiac magnetic resonance (CMR) is considered now to be the gold standard for quantitative IS determination [3]. However, widespread use of CMR in daily practice isn't still a reality as it is not easily accessible, expensive, needs long scanning times in addition to other patient-related factors including—but not limited to—relative contraindications in renal impairment and with certain implantable cardiac devices [4]. Accordingly, the focus of many researchers shifted towards finding readily accessible, reproducible and less expensive bedside tools for estimating IS. Measuring myocardial strain using advanced echocardiographic techniques such as the global longitudinal stain (GLS) by speckle-tracking technology has been shown to have high correlation with IS as measured by CMR for patients with 1st time STEMI [5]. Some research work pointed out this strong correlation in the *acute* phase of STEMI [6, 7]. Importantly, the *late* IS, measured 3 months following the

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acute phase of STEMI (otherwise known as *final* IS) is also linked to some significant complications such as life-threatening arrhythmias and hospitalizations for heart failure and it has been shown to provide strong independent prognostic information incremental to any traditional risk stratification parameter [8, 9]. Very few studies using very small sample sizes have examined the role of baseline GLS obtained during index hospitalization for prediction of late IS. Therefore, we thought that it may be interesting if we could explore the predictive value of baseline GLS on late IS among a group of patients with 1st time anterior STEMI treated contemporarily by PPCI.

Patients and methods

Study population

This single-center prospective observational study included 100 consecutive patients admitted with anterior STEMI to coronary care unit at Benha University Hospital, Egypt in the period from October 2017 to July 2018. *Key inclusion criteria* were: patients aged 18–80 years, first time anterior STEMI eligible for reperfusion therapy in the form of uncomplicated PPCI. While *key exclusion criteria* were: patients with prior STEMI or revascularization, those with severe valvular disease, patients with contraindications to MRI. All patients signed an informed consent and the study was approved by local ethics committee. We aimed to test the correlation between GLS and LGE for identification of IS in this category of patients.

Baseline evaluation

All patients had review of medical history, including demographic criteria, risk factor of CAD, history of co-morbidities. Physical examination, ECG, laboratory investigations were done on admission.

Primary PCI

A loading dose of 300 mg acetylsalicylic acid and 600 mg clopidogrel were given pre-procedure. Un-fractionated heparin (UFH) of 10,000 units' bolus dose was given after sheath insertion. The procedure was done according to the standard technique for coronary angiography and PCI. Transfemoral approach was done in all patients using 6 Fr sheaths. Diagnostic coronary angiography was done to explore non-infarct related artery. XB or Judkin left guide catheters were used to cannulate the left system. Thrombus aspiration and glycoproteins inhibitors (Eptifibatide or Tirofiban intracoronary bolus followed by intravenous infusion for 12 h) were used in lesions with heavy thrombus burden and/or impaired TIMI flow after the procedure (to the operator's discretion). The operator determined the length and diameter of implanted stents. Sheaths were removed 4 h post procedure. According to local protocol, PPCI was done for culprit-only revascularization.

Echocardiographic examination

Echocardiographic examination was performed using commercially available ultrasound system (*Philips EPIQ 7 Ultrasound System*) equipped with 3.5 MHz phased-array transducer. Baseline echocardiographic study was done within 48 h of admission. All exams were performed by two experienced operators blinded all clinical data. Before execution of the study and for reproducibility of measurements, these two experienced operators repeated ten measurements of left ventricular ejection fraction (LVEF), wall motion score index (WMSI), and GLS. Differences in measurements by the two observers were obtained for estimation of interobserver variability. The same observers repeated the ten measurements after a 2-month interval, and intraobserver variability was calculated.

The image was obtained at held end-expiration. LVEF was calculated using the Simpson's biplane method in accordance with the European Association of Echocardiography and the American Society of Echocardiography recommendations [10]. WMSI was assessed using the 17-segment model [10]. For the two-dimensional speckle-tracking echocardiography (2D-STE) image technique, sector depth and size were optimized to achieve perfect visualization of all LV myocardium in the three standard apical views (4-, 2-, and long-axis view) with a frame rate between 60 and 100 fps. End-systole was defined by the aortic valve closure in the apical long axis view. The regions of interest were manually outlined at end-systole by outlining the endocardial borders in the apical 4, 2, and 3 chamber views. Peak systolic longitudinal myocardial strain was automatically calculated throughout the myocardium for each LV apical view and reported spatially from base to apex and circumferentially in a polar plot map using a color-coded parametric representation. According to the American Society of Echocardiography recommendations [10], we divided the LV into 17 segments. The global longitudinal strain (GLS) was calculated by taking an average of all peak systolic segmental strain values from the three standard apical views. For calculation of anterior GLS, we separately calculated the average of the nine segments supplied by the LAD which are segments 1, 2, 7, 8, 12, 13, 14, 15, and 16. Longitudinal peak strain values were averaged over three consecutive cardiac cycles [11] (Fig. 1).

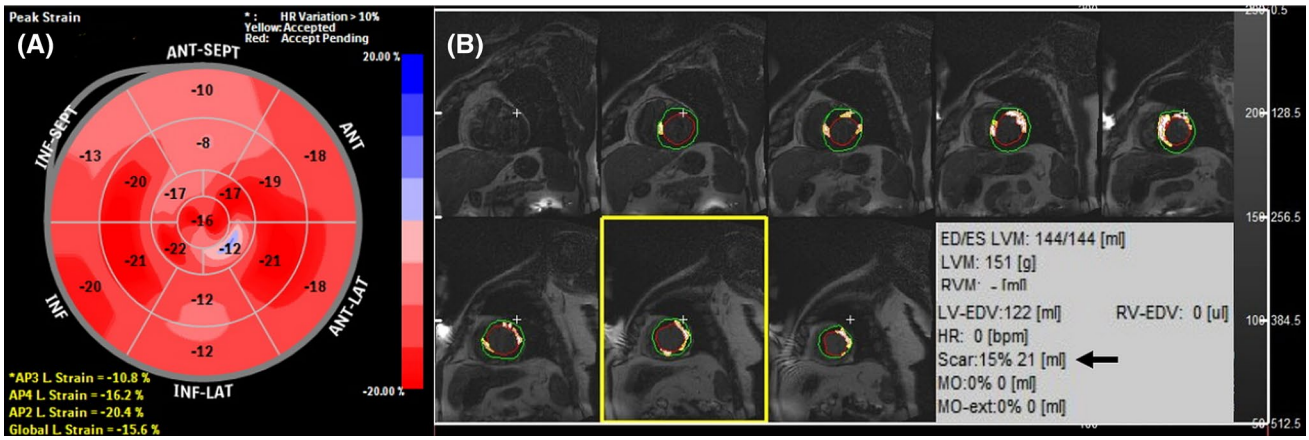

Fig. 1 **A** Bull's eye polar map of baseline GLS of case no. 20. Calculated GLS = − 15.5% and anterior GLS = − 15.4%. Baseline LVEF measured by Simpson = 55%. **B** Stack of short-axis CMRI images of LV in late enhancement with the enhancing scar delineated using semiautomated method. Scar percentage to the LV mass (black arrow) is generated automatically and displayed to the right of the figure

Cardiac magnetic resonance

The cardiac MRI (CMRI) was obtained 3 months after infarction using MAGNETOM Aera 1.5 T MRI scanner (*Siemens Healthcare GmbH, Henkestr, Germany*). A group of images were acquired using ECG gated breath hold technique pre and post-contrast. The pre-contrast images included: contiguous stack of short axis images followed by acquisition of vertical long axis, and horizontal long axis images using 2d balanced steady-state free-precession (True FISP). The post-contrast delayed enhancement contiguous stack of short axis images of LV were acquired 12–20 min after administration of IV contrast medium (*gadopentetate dimeglumine, Magnevist* 1 mmol/ml) using Phase sensitive inversion recovery (PSIR) single shot Trufisp, then the pre and post -contrast images were analyzed by Segment software for cardiac image analysis version 2.0 R4840 (*Medviso AB, Griffelvagen, Lund, Sweden*). The left ventricular mass was calculated from pre-contrast images by semi-automated tool after delineation of both endocardial and epicardial surfaces, while the scar percentage to the left ventricular mass was calculated from post-contrast images by specialized semi-automated tool with some manual corrections by delineating area of hyperintense late gadolinium enhancement in contrast to dark myocardium. According to the definition proposed by Sjøli et al. [6], we used a myocardial scar mass covering 20% of the LV as a cut-off point to differentiate large and small scars. (Fig. 1). Based on this cut-off point, enrolled patients were divided into two groups:

Group I: Those with large infarcts (≥ 20% of LV mass).
Group II: Those with small infarcts (< 20% of LV mass).

Sample size calculation

Sample size was calculated using *MedCalc software* v.18.2.1 based on previous study done by Grabka et al. [8]. The study reported Area Under Curve (AUC) equal to 0.83 of GLS for the prediction of large infarction. Total sample size of 22 patients (12 and 10 patients in large & small infarction groups respectively) will be a minimum. Type I and type II errors were adjusted at 0.05 and 0.2 respectively.

Statistical analysis

Data management and statistical analysis were done using SPSS vs.25. Numerical data was summarized as means and standard deviations or medians and ranges. Categorical data was summarized as numbers and percentages. Comparisons between two groups were done using independent t test or Mann Whitney U test for normally and non-normally distributed numeric variables respectively. Categorical data was compared using Chi square test of Fisher exact test if appropriate. Pearson's correlations were done between infarct size at 3 months and baseline GLS and AGLS. "r" is the correlation coefficient. It ranges from − 1 to + 1. − 1 indicates strong negative correlation, + 1 indicates strong positive correlation while 0 indicates no correlation. Linear regression analysis was done for the prediction of infarct size at 3 months. Regression coefficients "β" with 95% confidence intervals were calculated for predictors. ROC analysis was done for baseline GLS and AGLS for prediction of infarct size more than 20%. Area under curve with 95% confidence intervals, best cutoff points and diagnostic indices including sensitivity and specificity were calculated. Logistic regression analysis was performed for predictors of Infarct size more than 20%. Odds ratios with 95% confidence intervals

were calculated for all predictors. All P values were two sided. P value less than 0.05 was considered significant.

Results

Study population

One hundred and five patients were enrolled. Up to the time of CMRI, two patients withdrew their consents, one patient died due to out-of-hospital cardiac arrest, one patient developed acute kidney injury and was therefore excluded, and one patient dropped out due to inability to contact. Of the remaining 100 patients left for the final analysis, the mean (SD) age was 62 ± 10 years, 68% were males, 25% had DM, 54% were hypertensives, 49% were smokers, 45% had known dyslipidemia, 20% had family history of premature coronary artery disease (CAD), 17% had past history of CAD. Between groups analysis showed no statistically significant differences in all baseline characteristics. (Table 1).

Electrocardiography

Twenty-three patients had extensive anterior STEMI (22.8% versus 23.3% in groups I and II respectively, P = 1), 47 patients had anterior STEMI (47.4% vs. 46.5% in groups I and II respectively, P = 1), 23 patients had anteroseptal STEMI (22.8% vs. 23.3% in groups I and II respectively, P = 1) and 7 patients had anterolateral STEMI (7% in both groups, P = 1). The mean (SD) maximum baseline ST segment elevation among whole study population was 5 ± 2 mm and single-lead ST resolution (STR) after PPCI reperfusion was 66 ± 18%. Between groups analysis yielded a significantly higher maximum baseline ST elevation in group I than group II (6 ± 1 vs. 3 ± 2 mm respectively, P < 0.001). Moreover, single-lead STR after PPCI was significantly better in group II when compared to group I (72 ± 19 vs. 61 ± 16% respectively, P = 0.002).

Table 1 Baseline criteria of study groups

		Group I (n = 57) N (%)	Group II (n = 43) N (%)	P value
Age (years)	Mean ± SD	64 ± 11	60 ± 8	0.06
Gender	Males	38 (66.7)	30 (69.8)	0.724
DM		15 (26.3)	10 (23.3)	0.726
HTN		32 (56.1)	22 (51.2)	0.621
Smoking		29 (50.9)	20 (46.5)	0.665
Known dyslipidemia		25 (43.9)	20 (46.5)	0.792
FH of premature CAD		11 (19.3)	9 (20.9)	0.84
PH of IHD		10 (17.5)	7 (16.3)	0.868

DM diabetes mellitus, *FH* family history, *HTN* hypertension, *IHD* ischemic heart disease, *PH* past history

PPCI

Total ischemic time was 127 ± 24 min and first medical contact (FMC)-to-device time was 62 ± 7 min. Seventy-two percent had single vessel CAD, 33% had two-vessel CAD and 44% had three-vessel CAD. During PPCI, balloon dilatations were used in 57% of patients, thrombus aspiration devices were used in 14%, and stents were implanted in 91% (86% bare-metal stents 'BMS' and 6% drug-eluting stents 'DES'). The mean stents(s) length was 26 ± 7 mm and the mean stent(s) diameter was 3.05 ± 0.45 mm. Intra-procedural complications were reported in 5% of cases in the form of three cases with no-reflow, 1 case with stent edge dissection and 1 case with unstable ventricular arrhythmia. Of note, glycoprotein IIb/IIIa antagonists were used in 8% of cases. The preprocedural median (range) TIMI-flow was 0 (0–III) and postprocedural was III (II–III). Between groups analysis revealed a significantly longer total ischemic and longer FMC-to-device times in group I compared to group II (142 ± 20 vs. 117 ± 21 min for total ischemic time and 65 ± 6 vs. 60.7 min for FMC-to-device time, P < 0.001 for both). No statistically significant differences between groups were reported regarding all other PPCI procedural details. (Table 2).

Echocardiography

The interobserver variabilities were 5.6 ± 3.1% for LVEF, 4.2 ± 2.3% for WMS, and 6.8 ± 3.6 for GLS. Intraobserver variabilities were 4.6 ± 2.5% for LVEF, 5.7 ± 3.2% for WMS, and 5.8 ± 3.4% for GLS. The test–retest variability of GLS was 5.0 ± 2.1% (maximum 80.8, minimum − 112.3).

For the entire study population, the mean (SD) baseline end-systolic volume (ESV) was 61 ± 10 ml, end-diastolic volume (EDV) was 103 ± 11 ml, LVEF was 53 ± 3%, WMSI was 1.3 ± 0.3, tissue-doppler E′ (TDI E′) velocity at medial mitral valve (MV) annulus was 0.8 ± 0.1 m/s. The mean (SD) baseline GLS was − 13.8 ± 2.8% and anterior GLS was − 8.9 ± 2%. Table 3 illustrates between groups analysis regarding all baseline echocardiographic parameters. Notably, both GLS and anterior GLS were significantly higher in group II (small infarcts) when compared to group I (large infarcts) (− 15.8 ± 2.3 vs. 12.3 ± 2.1% for GLS and − 10.5 ± 1.7 vs. 7.7 ± 1.1% for anterior GLS in groups II and I respectively, P < 0.001 for both).

GLS as a predictor of late infarct size

The mean (SD) CMRI total late-infarct size at 3 months for the whole study population was 21 ± 7% (26 ± 4% in group I

Table 2 STEMI and PPCI procedural details

		Group I (n = 57) N (%)	Group II (n = 43) N (%)	P value
Max troponin T level (ng/ml)	Mean ± SD	14.0 ± 2.6	9.8 ± 3.2	< 0.001
Total ischemic time (min.)	Mean ± SD	142 ± 20	117 ± 21	< 0.001
FMC-to-device time (min.)	Mean ± SD	65 ± 6	60 ± 7	< 0.001
No of vessels affected, n (%)	One vessel CAD	15 (26.3)	12 (27.9)	0.877
	Two vessel CAD	20 (35.1)	13 (30.2)	
	Three vessel CAD	22 (38.6)	18 (41.9)	
Balloon use		33 (57.9)	24 (55.8)	0.835
Thrombus aspiration		8 (14.0)	6 (14.0)	0.991
Stent use		51 (89.5)	40 (93.0)	0.539
Type of stent	BMS	48 (94.1)	38 (95.0)	1.0
	DES	3 (5.9)	2 (5.0)	
Average stent(s) length (mm)	Mean ± SD	26 ± 7	27 ± 8	0.691
Average stent(s) diameter (mm)	Mean ± SD	2.98 ± 0.41	3.15 ± 0.49	0.075
Intra procedural complications	No reflow n (%)	2 (3.5)	1 (2.3)	–
	Dissection n (%)	0 (0.0)	1 (2.3)	
	Perforation n (%)	0 (0.0)	0 (0.0)	
	Unstable arrhythmia n (%)	1 (1.8)	0 (0.0)	
Glycoprotein IIb/IIIa inhibitors		6 (10.5)	2 (4.7)	0.460
TIMI flow before	Median (range)	0 (0–III)	0 (0–III)	0.654
TIMI flow after	Median (range)	III (II–III)	III (II–III)	0.433

BMS bare metal stent, *DES* drug eluting stent, *FMC* first medical contact, *GP* glyco-proteins, *TIMI* thrombolysis in myocardial infarction

Table 3 Baseline echocardiographic parameters in study groups

	Group I (n = 57)		Group II (n = 43)		P value
	Mean	SD	Mean	SD	
Baseline ESV (ml)	63	11	59	8	0.06
Baseline EDV (ml)	108	10	95	6	< 0.001
Baseline EF (%)	52	2	54	3	< 0.001
Baseline WMSI	1.4	0.3	1.1	0.3	< 0.001
Baseline TDE′ velocity (m/s)	0.8	0.2	0.9	0.1	< 0.001
Baseline GLS (%)	− 12.3	2.1	− 15.8	2.3	< 0.001
Baseline AGLS (%)	− 7.7	1.1	− 10.5	1.7	< 0.001

ESV end-systolic volume, *EDD* end-diastolic volume, *EF* ejection fraction, *WMSI* Wall Motion Score Index, *GLS* global longitudinal strain, *AGLS* anterior global longitudinal strain, TDE′ Tissue Doppler E′

and 14 ± 4% in group II, P < 0.001). There was a statistically significant high correlation between baseline GLS and late infarct size assessed by CMRI. This correlation was higher for baseline anterior GLS (r = − 0.867, P < 0.001) than for baseline GLS (r = − 0.840, P < 0.001) (Fig. 2). Linear regression analysis using LGE in CMRI as a dependent factor and both baseline GLS and anterior GLS as independent factors showed that for every point estimate reduction of baseline GLS, there's a corresponding increase in LGE of 0.673% (CI 1.363–2.009), P < 0.001, and for every point estimate reduction of baseline anterior GLS, there's a corresponding increase in LGE of 0.269% (CI 0.495–1.405), P < 0.001. With the receiver operating characteristic curve, the cut-off point of − 13% for GLS was an indicator of at least 20% scar of LV mass in LGE with 66.7% sensitivity and 88.4% specificity (area under the curve = 0.855 [CI 0.784–0.925], P < 0.001). For segments supplied by LAD, the cut-off value of anterior GLS for the prediction of large infarct was − 9.6% (sensitivity 94%, specificity 86%, and area under the curve = 0.9 [CI 0.828–0.973], P < 0.001) (Fig. 3). An adjusted multivariate logistic regression analysis using large late infarct size (≥ 20% of LV mass) as a dependent factor showed both baseline GLS and anterior GLS to be among the significant independent predictors for occurrence of large late infarcts (OR 1.73, CI 1.02–2.95, P = 0.042 for GLS and 8.85, CI 1.85–41.6, P = 0.006 for anterior GLS) (Table 4).

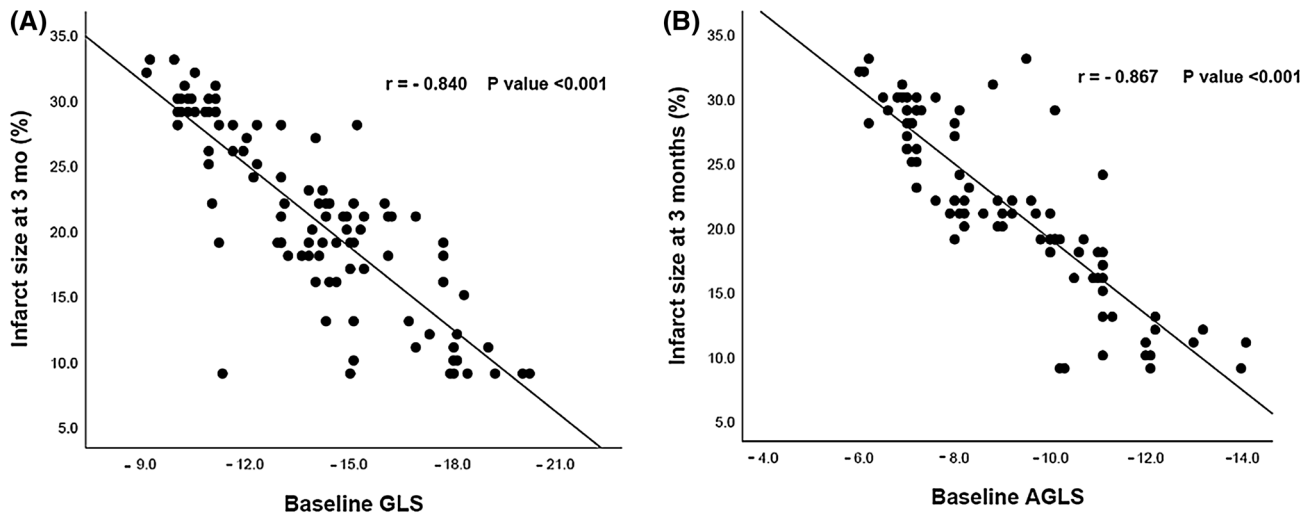

Fig. 2 Correlation between percentage of LV myocardium covered by scar with baseline GLS (**A**) and baseline anterior GLS (**B**)

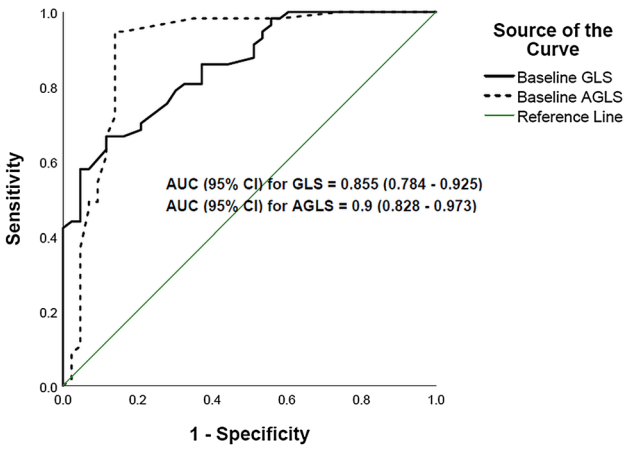

Fig. 3 Receiver operating characteristics curve for prediction of large late infarct size based on the baseline global longitudinal strain (GLS) and baseline anterior GLS. Areas under curve with their 95% confidence intervals (CIs) are provided

Table 4 Predictors of large late infarct size

	OR	95% CI for OR	P value
Max troponin T level (ng/ml)	1.915	(1.215–3.016)	0.005
Max baseline ST elevation (mm)	2.937	(1.108–7.786)	0.03
Baseline GLS	1.73	(1.02–2.95)	0.042
Baseline AGLS	8.85	(1.85–41.6)	0.006
Baseline EF (%)	1.795	(1.06–3.03)	0.03

GLS global longitudinal strain, *AGLS* anterior global longitudinal strain, *EF* ejection fraction, *OR* odds ratios, *95% CI* 95% confidence interval

Discussion

In simple words, infarct size following STEMI is a major determinant of subsequent heart failure and mortality [12]. Being linked with some significant late complications such as serious arrhythmias and hospitalizations due to heart failure, *late* infarct size (measured few months after STEMI) is of particular concern in this context [9]. Many cardioprotective therapies have been studied in 62 randomized controlled trials (RCTs) for reduction of IS using LGI-CMRI as the gold standard for IS measurement (15% of these RCTs used 3-months LGE-CMRI for follow-up) [13]. However, CMRI may not be ready for the prime time due to many issues such as expense, accessibility and long duration of scans [4]. This becomes true in some world developing countries with burdened economies such as Egypt. Therefore, we strongly believe that any non-invasive, rapid and relatively cheap techniques that provide reasonable accuracy for infarct size estimation could be a great addition to the therapeutic armamentarium of STEMI.

Speckle tracking echocardiography (STE)-derived GLS is a little bit similar to the CMRI technique knows as 'myocardial tagging' that is used to evaluate deformation of individual myocardial segments, and the results of both techniques are in line with each other [14, 15].

In this study, we found that both baseline GLS and anterior GLS (done for segments supplied by the LAD separately) significantly predict large late infarct size at 3-months follow up. Moreover, there was a pretty good and statistically significant correlation between both baseline GLS values and infarct mass (expressed as scar percentage to LV myocardium).

Most studies in the literature that evaluated the role of GLS for prediction of IS utilized early (rather than late) IS as an endpoint, and some of them evaluated patients with NSTEMI (rather than STEMI). Moreover, 3D-GLS (with its notoriously known lack of standardization) has been explored by some. All these factors translated into a relatively high degree of heterogeneity across studies leading to inability to draw firm conclusions [5]. On the other hand, very few studies have evaluated the predictive power of 2D-GLS on the final late infarct size > 1 month after 1st STEMI. Grabka et al. enrolled 39 patients with their 1st time anterior STEMI and found a good correlation between 2D-GLS and late infarct size using CMRI (r = 0.62, CI 0.38–0.78). They also reported a cut-off point of baseline GLS of − 12.3% to predict large infarct size (> 20%) with a sensitivity and specificity of 85% and 96% respectively [8]. Another study by Bière et al. in 2014 evaluated 41 STEMI patients (anterior and inferior) and showed a good correlation between 2D-GLS and late infarct size by CMRI (r = 0.61, CI 0.37–0.77) [16].

Our study, although its findings are in agreement with other previous studies, has some strengths. First, we provide -through a formal calculation- the largest sample size ever reported for studies evaluating the role of GLS for prediction of *late* infarct size after STEMI [5]. Second, we separately assessed the GLS of segments supplied by the LAD to nullify any influence of multi-vessel disease on the validity of our results. Third, instead of evaluating the whole population of STEMI irrespective to the location of infarction and reperfusion modalities (a relatively heterogeneous population) as done previously by some investigators, we opted to providing a unique dataset by focusing exclusively on a very homogenous high-risk population with their 1st anterior STEMI treated contemporarily by PPCI.

Although lack of assessment of circumferential strain may be suggested as a limitation to this study, we believe (among others) that global circumferential strain (GCS) lacks the high predictive power provided by GLS as shown in many studies [5]. This is surprising in light of the fact that circumferential strain at the inner endocardium (early affected in MI) is basically higher than that of the outer epicardium (later affected in MI). However, a simple techniqal reason for this discrepancy is that the ultrasound beam of the probe is longitudinal, thus it's inherently parallel to the GLS and this helps to provide a better resolution in the longitudinal direction. In contrast, GCS is measured in the short-axis view, thereby it is plagued by the inevitably low resolution in the apical and basal levels. Better algorithmic developments in the future may improve the utility of GCS in this domain.

Finally, we preferred to utilize 2D-GLS rather than 3D-GLS for two main reasons. First, results coming from studies utilizing both 2D and 3D-GLS [17, 18] indicate that the performance of 2D-GLS is better, even when compared to CMRI findings. This is because 2D technique allows for a multi-layered assessment in contrast to the single full-thickness layer assessment provided by 3D technique. Therefore, if we consider the fact that strain change in the longitudinal direction is mainly caused by oblique subendocardial fibers (calling for a single and separate layer assessment), one could realize easily that 3D technology might not be the correct choice. Second, 2D-GLS is becoming more widely accessible than 3D-GLS. Accordingly, we thought that any relevant findings from our study would be readily and rapidly reflected on daily practice, a main value of any clinical research.

Conclusion

Semiautomated calculation of early baseline GLS significantly predicts and correlates well with late infarct size at 3-months follow up in patients with anterior STEMI treated by PPCI. This represents a promising simpler and readily accessible tool whenever CMRI is not available.

Acknowledgements We appreciate the invaluable insights of Dr. Mohamed Bendary for statistical data analysis. We also greatly acknowledge Dr. Tarek Afifi for his efforts in preparation of CMRI procedures.

Compliance with ethical standards

Conflict of interest The authors declare that they have no competing interests.

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