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SYSTEMATIC REVIEWS

Global myocardial strain assessment by different imaging modalities to predict outcomes after ST-elevation myocardial infarction: A systematic review

Abhishek Shetye, Sheraz A Nazir, Iain B Squire, Gerald P McCann

Abhishek Shetye, Sheraz A Nazir, Iain B Squire, Gerald P McCann, Department of Cardiovascular Sciences, University of Leicester, Glenfield Hospital, Leicester LE3 9QP, United Kingdom

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Correspondence to: Dr. Gerald P McCann, National Institute of Health Research Career Development Fellow/Honorary Consultant Cardiologist, Department of Cardiovascular Sciences, University of Leicester, Glenfield Hospital, Groby Road, Leicester LE3 9QP,
United Kingdom. gerry.mccann@uhl-tr.nhs.uk
Telephone: +44-116-2583977

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Abstract

AIM: To conduct a systematic review relating myocardial strain assessed by different imaging modalities for prognostication following ST-elevation myocardial infarction (STEMI).

METHODS: An online literature search was performed in PubMed and OVID® electronic databases to identify any studies that assessed global myocardial strain parameters using speckle-tracking echocardiography (STE) and/or cardiac magnetic resonance imaging (CMR) techniques [either myocardial tagging or feature tracking (FT) software] in an acute STEMI cohort (days 0-14 post-event) to predict prognosis [either development of major adverse cardiac events (MACE)] or adverse left ventricular (LV) remodelling at follow-up ($\geqslant$ 6 mo for MACE, $\geqslant$ 3 mo for remodelling). Search was restricted to studies within the last 20 years. All studies that matched the pre-defined search criteria were reviewed and their results interpreted. Due to considerable heterogeneity between studies, meta-analysis was not performed.

RESULTS: A total of seven studies (n = 7) were identified that matched the search criteria. All studies used STE to evaluate strain parameters - five (n = 5) assessed global longitudinal strain (GLS) (n = 5), one assessed GLS rate (GLS-R) (n = 1) and one assessed both (n = 1). Three studies showed that GLS independently predicted the development of adverse LV remodelling by multivariate analysis - odds ratio between 1.19 (CI: 1.04-1.37, P < 0.05) and 10 (CI: 6.7-14, P < 0.001) depending on the study. Four studies showed that GLS predicted the development of MACE - hazard ratio (HR) between 1.1 (CI: 1-1.1, P = 0.006) and 2.34 (1.10-4.97, P < 0.05). One paper found that GLS-R could significantly predict MACE -

HR 18 (10-35, $P < 0.001$) - whilst another showed it did not. GLS < -10.85% had sensitivity/specificity of 89.7%/91% respectively for predicting the development of remodelling whilst GLS < -13% could predict the development of MACE with sensitivity/specificity of 100%/89% respectively. No suitable studies were identified that assessed global strain by CMR tagging or FT techniques.

CONCLUSION: GLS measured acutely post-STEMI by STE is a predictor of poor prognosis. Further research is needed to show that this is true for CMR-based techniques.

Key words: Strain; Speckle tracking; Tagging; Feature tracking; Myocardial infarction; Major adverse cardiac events; Remodelling

Core tip: Global myocardial strain is an objective measure of cardiac function. It can be assessed using post-processing analysis on different imaging modalities such as speckle-tracking echocardiography (STE) and cardiac magnetic resonance imaging (CMR) - tagging and feature tracking. We performed a systematic review that showed global longitudinal strain (GLS) measured acutely by STE following ST-elevation myocardial infarction (STEMI) predicted clinical outcomes and adverse left ventricular remodelling, a surrogate marker of poor prognosis. No relevant studies were found for CMR techniques. GLS may refine risk stratification in the STEMI population but further work is needed to support this.

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INTRODUCTION

Ischaemic heart disease (IHD) presents a significant burden to healthcare services and is one of the leading causes of death worldwide[1]. Acute myocardial infarction (MI) results from spontaneous coronary artery occlusion due to thrombus formation as a result of plaque rupture and subsequent platelet aggregation - most commonly seen with the background of IHD[2]. ST-elevation myocardial infarction (STEMI) is an acute emergency that requires prompt reperfusion by either primary percutaneous coronary intervention (PPCI) or thrombolysis, ideally within two hours of symptom onset[3].

Timely reperfusion has led to a reduction in mortality from acute MI[4]. However, despite receiving current best therapy, a significant number of patients still develop complications post-MI that includes new-onset heart failure (HF)[5] - 20.4% of patients develop HF on admission and 8.6% subsequently[6]. The incidence of HF has increased over the past few decades[7] and it is especially prevalent amongst the elderly[8]. Long-term mortality from HF still remains high, even with the best contemporary pharmacological and non-pharmacological interventions[9]. The increase in HF incidence may partly be a result of improved survival post-MI, albeit with greater morbidity in some survivors.

Outcomes after STEMI

Major adverse cardiac events: Major adverse cardiac events (MACE) are often used in cardiovascular studies as a measure of clinical outcomes after STEMI. It is an umbrella term that includes a variety of measures - including all-cause mortality, hospital readmission due to HF, recurrence of MI, need for revascularisation, and occurrence of stroke. Demographic features associated with poor outcomes post-STEMI include age[10], diabetes[11], hypertension[12], infarct location (*i.e.*, anterior MI)[13], large infarct size (IS)[14] and presence of microvascular obstruction[15].

"Hard events" such as mortality are the best markers of outcome. However, these are relatively rare occurrences and so require a considerable sample size to demonstrate statistically significant association with a biomarker, or effects of intervention[16] and some authors believe that studies reporting these need to have a sample size of $n > 1000$ to be statistically robust[17]. Such large, multi-centre trials are challenging to conduct and need to be carried out over a considerable period of time in order to accrue the required sample sizes and numbers of events. Consequently, surrogate markers of poor outcome such as adverse left ventricular (LV) remodelling can be used in lieu of hard outcomes with much smaller sample sizes to achieve statistically significant results.

Adverse LV remodelling: Adverse LV remodelling post-MI is thought to be the main process underpinning the development of HF and is defined as: "A change in size, shape and function of the heart resulting from cardiac load or injury"[18]. It is a complex process that progresses over a period of weeks to months post-infarct (Figure 1). Adverse LV remodelling post STEMI can be defined as either an increase in end-diastolic volume (EDV) of > 20% or end systolic volume (ESV) of > 15%, at follow-up compared to baseline. However, there is no consensus on which definition is better. Several cellular, extracellular, inflammatory, and neuro-hormonal pathways have been implicated to play a role in development of LV remodelling; these include neutrophils[19], macrophages[19], collagen fibres[20], various metallo-proteinases[20] and activation of the sympathetic nervous system along with the renin-angiotensin-aldosterone system (RAAS)[7,18] amongst others. The exact role of

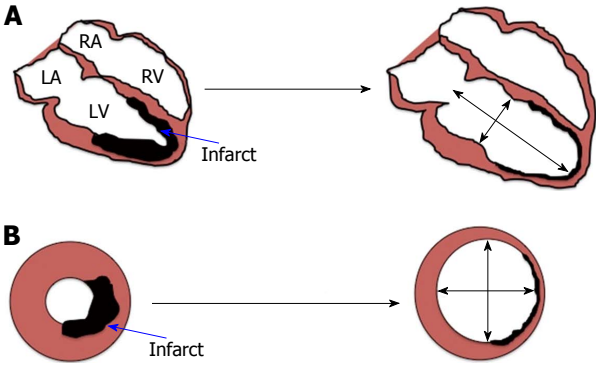

Figure 1 Development of adverse left ventricular remodelling post-myocardial infarction in (A) long axis view and (B) short axis view. LA: Left atrium; LV: Left ventricle; RA: Right atrium; RV: Right ventricle.

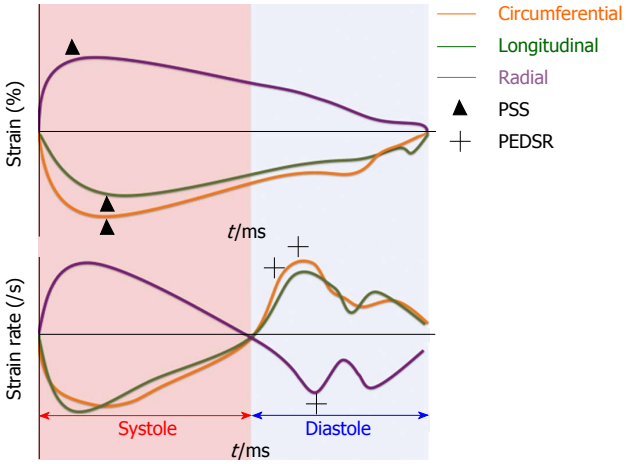

Figure 3 Strain and strain rate values as a function of time - peak systolic strain and peak early diastolic strain rate are annotated. PSS: Peak systolic strain; PEDSR: Peak early diastolic strain rate.

these components has not yet been elucidated and there is still some controversy over the initial trigger of remodelling[21]. There is good evidence to suggest that angiotensin-converting enzyme inhibitors, angiotensin receptor antagonists, and aldosterone antagonists attenuate the process of adverse remodelling by inhibiting RAAS[18].

Early identification of high-risk patients who are likely to undergo adverse LV remodelling may allow targeted therapeutic intervention in these patients to counteract remodelling processes. Parameters that reflect myocardial dysfunction can potentially be utilised to help identify such patients as cardiac function is often affected post-MI, which usually precedes development of overt HF.

LV dysfunction post-infarction

Traditionally, the systolic phase of the cardiac cycle is often used as a measure of LV function in a clinical setting. A region of myocardium affected by an infarct may have impaired contractility due to death of myocytes in that zone. Ejection fraction (EF) is the most commonly used method to assess systolic function and a

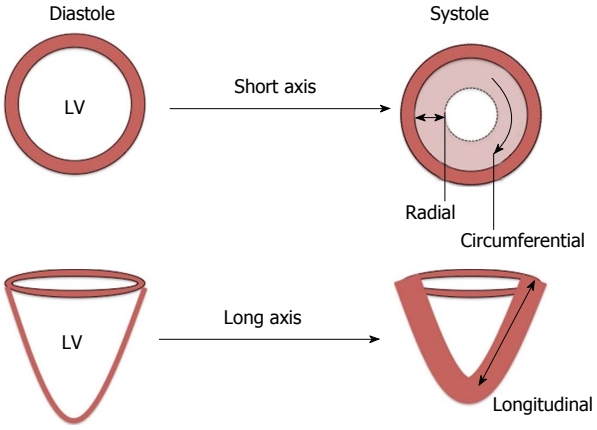

Figure 2 Myocardial contraction in three vectors - circumferential, longitudinal and radial. LV: Left ventricle.

reduced EF, commonly measured by echocardiography, is known to be associated with a poor outcome[22]. However, EF is relatively insensitive to regional differences in myocardial function and has been shown to be a poor predictor of late myocardial dysfunction when measured acutely after reperfusion therapy[23]. Wall Motion Score Index (WMSI) has also been used in addition to EF but it has the inherent shortcoming of being a subjective measure based on the experience of the assessor. WMSI is based on either the 16-segment[24] or the 17-segment model[25] of the LV.

An infarct is also thought to affect LV compliance by increasing wall stiffness and hence reducing active relaxation of the myocardium - this can cause diastolic dysfunction[26]. Recent evidence suggests that diastolic dysfunction post-MI measured by echocardiography confers a poor outcome[27,28].

The optimal marker of LV dysfunction would: (1) Be objective and "angle independent"; (2) Be sensitive to myocardial dysfunction early after an MI; (3) Offer an evaluation of both regional and global LV contractility; (4) Provides an assessment of both systolic and diastolic heart function; and (5) Be reproducible and easy to measure.

Myocardial strain

Strain is defined as the change in length of an object relative to its original length[29]. In the heart, myocardial strain is a sensitive measure of contractility. Strain can be calculated at both the segmental and global level and in the three axes of myocardial contraction - circumferential, longitudinal and radial (Figure 2). Strain rate (SR) measures the change in strain for a given vector as a function of time and can also be assessed. Systolic and diastolic strain rates vary throughout the cardiac cycle (Figure 3).

Anatomically, myocardial fibres are orientated longitudinally in the sub-endocardium and circumferentially in the mid-myocardium[30]. This suggests that longitudinal strain (LS) can provide a reflection of sub-endocardial function whilst circumferential strain can inform mid-myocardial function. Radial strain, whilst

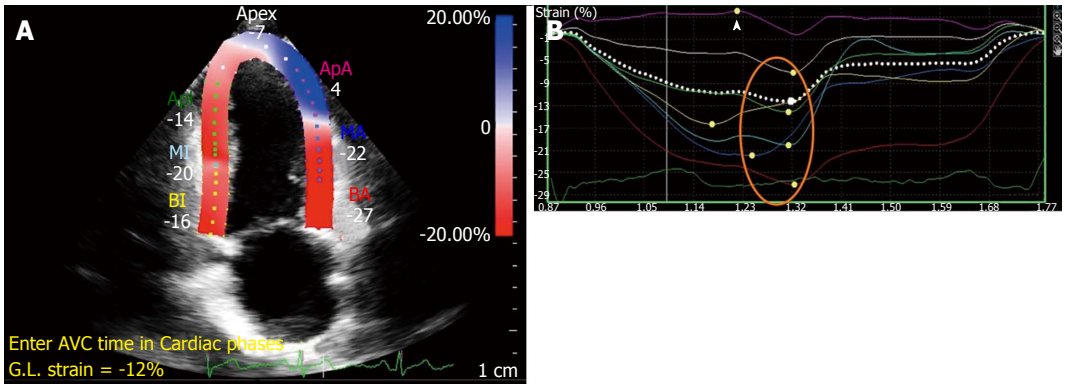

Figure 4 Peak systolic strain calculated by speckle-tracking echocardiography. A: Segmental strain after definition of endocardial and epicardial contours; B: Graphical illustration of segmental peak systolic strain - normal values annotated by orange circle, impaired strain by arrowhead.

being potentially informative of myocardial contraction in the short axis, has been shown to have high intra- and inter-observer variability[31] making it unsuitable for routine clinical practice. Peak systolic strain (PSS) is commonly used to assess myocardial contraction whilst peak early diastolic strain rate (PEDSR) is a marker of diastolic function[32]. Consequently, strain/diastolic strain rate assessment provides a comprehensive evaluation of myocardial contractility and compliance.

Myocardial Strain/strain rates can be assessed by a number of different imaging modalities - most frequently by echocardiography, but also by cardiac magnetic resonance imaging (CMR).

Echocardiography

Tissue Doppler imaging can assess myocardial strain but this technique is extremely angle dependent and has been superseded by speckle tracking echocardiography (STE)[33,34]. The ultrasonic images obtained by echocardiography consist of a large number of "speckles" which have individual properties[35]. These "acoustic markers"[34] can be identified and tracked as they move from one frame to the other throughout the cardiac cycle. Endocardial and epicardial borders are pre-defined by the operator and each speckle within this region of interest (ROI) is tracked. The tracking of such movement can be used to derive measures of strain[36] and strain rate[33]. STE is entirely a post-processing analysis. The only minor requirements are a short duration of breath holding by the patient so that respiratory motion does not affect the tracking of cardiac motion and a high frame rate to optimize temporal resolution.

Common echocardiographic imaging protocols include the acquisition of two-, four-chambered and three chamber views from which global LS (GLS) is derived (Figure 4). Short axis views allow circumferential and radial strain to be derived but it is difficult to accurately obtain global measures due to uncertainty of the imaging plane location.

STE-derived global strain parameters in the setting of an acute STEMI have shown good reproducibility - intra- and inter-observer variability of 0.92 and 0.85 by Intra-class Correlation Coefficient (ICC) respectively[37]. Repeatability is a measure of the "variation in repeat measurements made on the same subject under identical conditions made within a short period of time over which the underlying value can be considered to be constant"[38]. It is another method of establishing reliability. However, no studies to date have reported the repeatability of global strain measured by STE in acute STEMI.

CMR

CMR is another non-invasive imaging modality and is an alternative method of imaging to echocardiography. CMR can be used in the diagnosis, risk-stratification, and prognosis of a number of cardiac disorders[39,40], including acute MI[41-43]. Typically, strain is assessed on CMR using specialised myocardial tissue tagging sequences that involves the superimposition of horizontal and vertical lines on a cine image that appear in the form of a "grid"[44]. These grids or "tags" are formed onto the tissue by changing the local magnetisation through the use of selective radiofrequency saturation pulses perpendicular to the plane of image acquisition[45]. Tags deform along with the myocardium through the cardiac cycle and this deformation can be used to assess strain. Tagged images are commonly acquired using spatial modulation of magnetisation (SPAMM)[46] and complementary SPAMM sequences[47]. Post-processing analysis of tagged data can be performed using Harmonic phase analysis[48] and local sine wave modelling[49] and they have been shown to have good agreement[50]. Tagging has been validated against other invasive methods of strain assessment such as sonomicrometry[51] and has been used in a variety of animal models[52-54]. Tagging-derived strain parameters have a good intra- and inter-observer variability - ICC of 0.8 for both - along with acceptable test-retest repeatability - ICC of 0.74[55].

Tagging sequences however involve relatively long breath holds that may be difficult in the context of a recent STEMI. In addition, analysis is also labour-intensive and time-consuming[56]. Tagging, particularly with SPAMM sequences, cannot reliably calculate diastolic strain as the tags fade after systole especially at the 1.5 T field strength[45,57]. This can be overcome

Table 1 Advantages and disadvantages of speckle-tracking echocardiography *vs* cardiac magnetic resonance imaging

Advantages	Disadvantages
Cheaper than CMR scan	Cannot acquire SAX views easily - needed to calculate circumferential strain
Can be performed at the bedside	Cannot routinely obtain stress imaging as part of acquisition protocol
Short duration: 10-20 min for STE *vs* 45-60 min for CMR	Not possible to ascertain infarct size, oedema, microvascular obstruction
Significant contraindications for CMR - for example, pacemaker/ICD, brain aneurysmal clip, claustrophobia, eGFR < 30 mL/min per 1.73 m^2 - *vs* almost none for STE	CMR has much higher spatial resolution than STE. Consequently, a greater percentage of images are analysable by CMR than STE

CMR: Cardiac magnetic resonance imaging; eGFR: Estimated glomerular filtration rate; ICD: Insertable cardioverter defibrillator; SAX: Short axis; STE: Speckle-tracking echocardiography.

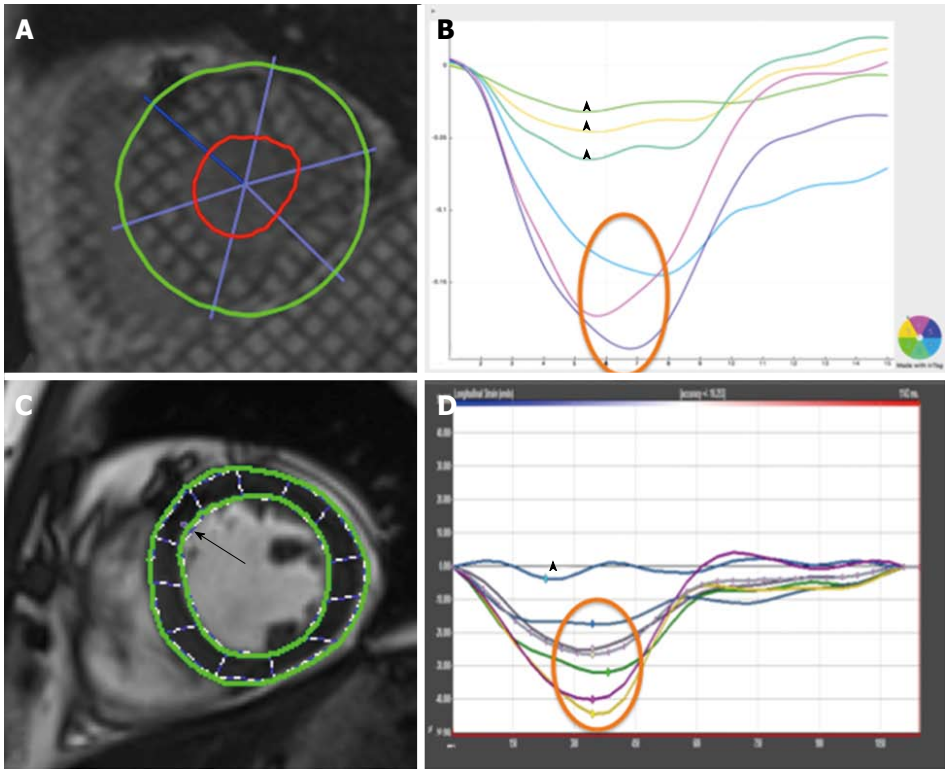

Figure 5 Comparison of tagging (A and B) and feature tracking (C and D) for evaluation of global circumferential strain - normal peak systolic strain annotated by orange circle, impaired peak systolic strain by arrowhead.

by using a stronger magnetic field strength (3.0 T) and Steady State Free Precession (SSFP) sequences[45]. However, true reproducibility is poor at 3.0 T CMR[58]. This may in part be due to the fact that by 3.0 T CMR images are also more susceptible to artefacts due to increase in inhomogeneity within the magnetic field[59].

To overcome the issues of tagging, myocardial motion tracking through the cardiac cycle on routinely acquired cine SSFP sequences can be performed by means of the novel feature tracking (FT) software[60]. FT is analogous to STE - endocardial and epicardial borders are defined and then subsequently propagated through the cardiac cycle. The software tracks the motion of the defined ROI from one frame to the next - PSS and PEDSR can be derived from this motion[60]. FT has shown excellent reproducibility - intra- and inter-observer variability of 0.988 and 0.971 in terms of ICC[61] - and acceptable test-retest repeatability - ICC of 0.77[56] - for PSS. Additionally, PSS by FT can predict global recovery of LV function in terms of EF[62].

Figure 5 illustrates a comparison of global circumferential strain (GCS) evaluation by tagging and FT.

STE vs CMR to assess strain

STE has several advantages over CMR in the assessment of strain (Table 1). There is good agreement between STE-derived and CMR derived global values of strain - this is true both for tagging[63,64] and FT[65]. This suggests that these methods could be used interchangeably in the assessment of global strain. A detailed comparison of different imaging modalities to be used in the setting of an acute MI can be found elsewhere[36].

Aims of systematic review

Global myocardial strain can objectively evaluate LV dysfunction post-STEMI and can be measured by

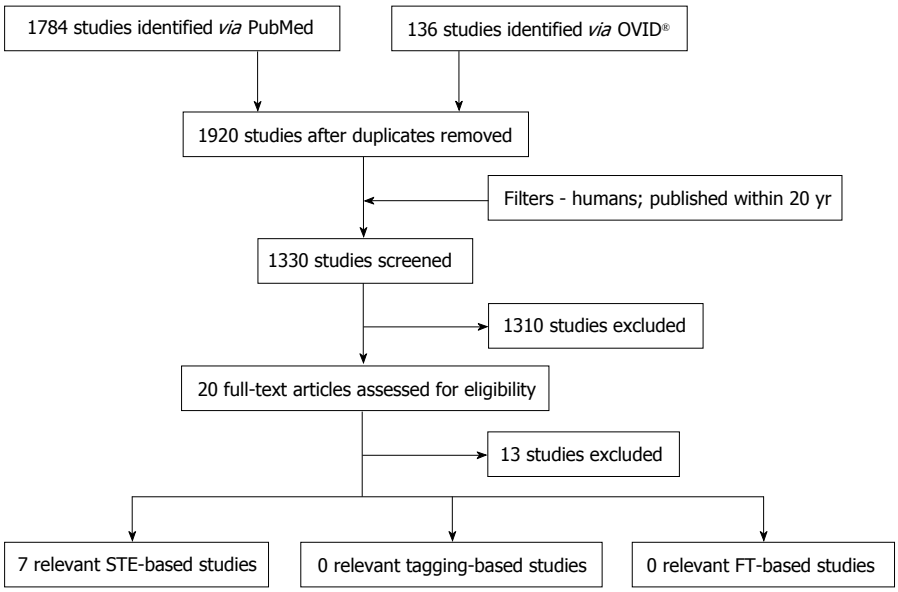

Figure 6 Flowchart illustrating the search for relevant studies. FT: Feature tracking; STE: Speckle-tracking echocardiography.

Table 2 Eligibility criteria for systematic review

Type of characteristic	
Population type	Acute STEMI
Measured parameters	Global longitudinal and/or circumferential strain and/or strain rate - PSS or strain rate (PSS-R) or PEDSR
Imaging modalities	STE or cardiac MRI tagging or cardiac MRI FT
Timeframe for baseline scan	Days 0-14 post-STEMI
Outcomes reported	MACE or adverse LV remodelling
Timeframe for follow-up	MACE - ⩾ 6 mo Adverse LV remodelling - s ⩾ 3 mo
Year published	Within the last 20 yr

STEMI: ST-elevation myocardial infarction; PSS: Peak systolic strain; PEDSR: Peak early diastolic strain rate; STE: Speckle-tracking echocardiography; FT: Feature tracking; MACE: Major adverse cardiac events; LV: Left ventricular; MRI: Magnetic resonance imaging.

STE and CMR techniques with good reproducibility and repeatability. We looked to review the literature for studies that evaluated the ability of global strain measured acutely post-STEMI by either STE or CMR to predict either MACE or development of adverse LV remodelling.

MATERIALS AND METHODS

We followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses protocol[66].

Eligibility criteria

Table 2 highlights the eligibility criteria for the review. Studies were limited to acute STEMI patients to represent the setting of an acute MI - NSTEMI patients were excluded since the diagnosis is more complex, heterogeneous presentations and that their subsequent management is based on risk-stratification[67]. There was no limitation placed on the management of the STEMI - both in terms of method of revascularisation (PPCI or thrombolysis) and success/failure. Strain parameters were restricted to peak systolic GCS and GLS and PEDSR in the same two vectors. Both segmental strain values and radial strain parameters were excluded since they both have been shown to have poor intra- and inter-observer variability[31,58]. We limited the timeframe for the baseline scan to be 0-14 d post- to limit the effects of subsequent remodelling. The timeframe for outcome measures were ⩾ 3 mo for adverse LV remodelling (since it is a dynamic process that takes months to fully develop[68]). Minimum follow-up time for development of MACE was six months. We included studies that quoted either changes in EDV or ESV.

Search protocol

The literature search was performed in PubMed and OVID® electronic databases. The final date on which the online search was performed was January 27th, 2015 (Table 3) for list of keywords used.

Study selection

Figure 6 highlights the process of study selection. Initial electronic search yielded 1920 studies; 1330 remained after addition of relevant filters. The titles and abstracts of these studies were then screened to assess for eligibility for inclusion in the systematic review (Table 2). A majority of the studies were deemed inappropriate for inclusion based on the aforementioned criteria (n = 1310). The remaining 20 papers were further scrutinised by searching for and evaluating the full-text article. A further 13 studies were excluded - some did not actually assess strain at all (n = 4), some assessed torsion (n = 3), three had included NSTEMI patients and the rest did not have full-text articles available as they were presented as posters (n = 3). Consequently,

Table 3 Keywords used for search of electronic databases

"Cardiac MRI" OR "CMR" OR "magnetic resonance imaging [MeSH Term]" OR "cardiac magnetic resonance" OR "feature tracking" OR "tissue tracking" OR "tagging" OR "tag" OR "tagged" OR "SPAMM" OR "CPSAMM" OR "HARP" OR "SinMOD" OR "Echocardiography [MeSH Term]" OR "Speckle tracking", "2D speckle" OR "3D speckle" OR "two dimensional speckle" OR "three dimensional speckle". MIs were searched using "myocardial infarction [MeSH Term]" OR "acute MI" OR "STEMI" OR "ST elevation". Strain was searched using "strain" OR "myocardial strain" OR "strain rate" OR "deformation" OR "myocardial deformation" OR "systolic" OR "diastolic" OR "PSS" OR "PEDSR" OR "longitudinal" OR "circumferential". Outcomes were searched using "Predict" OR "Outcome" OR "Risk" OR "Prognosis" OR "Logistic Models [MeSH Term]" OR "risk" OR "multivariable" OR "multivariate" OR "odds" OR "MACE" OR "mortality [MeSH Term]" OR "remodelling" OR "remodelling" OR "adverse" OR "cardiac" OR "left ventricular"
Note: MeSH terms were only available on PubMed

MRI: Magnetic resonance imaging; CMR: Cardiac magnetic resonance imaging; STEMI: ST-elevation myocardial infarction; PSS: Peak systolic strain; PEDSR: Peak early diastolic strain rate.

there were seven studies that matched our inclusion criteria for the review.

RESULTS

Strain measured by STE

Seven STE-based studies that matched our inclusion criteria were found (Table 4) highlights studies that assessed global strain to predict adverse LV remodelling and Table 5 highlights studies that used global strain to predict MACE. Six studies reported peak systolic longitudinal strain parameters to predict outcomes - only one study used diastolic strain. All the patients were treated with PPCI.

Multivariate analyses in all the studies have shown that peak systolic GLS can independently predict both adverse LV remodelling and MACE. Such analyses have shown that this is independent of factors such as age, diabetes, location of infarct, EF and WMSI. One study showed that global longitudinal SR (GLS-R) also had significant impact on prognosis[69] - patients with impaired GLS-R, and GLS, were 18-times more likely to suffer from composite endpoint of mortality, readmission due to HF, revascularisation, or re-infarction. One study showed that a cut-off GLS > -12.5% (*i.e.*, LV unable to contract more than 12.5% of its original length in the longitudinal vector) could predict development of remodelling - OR 1.19 (1.04-1.37), $P < 0.05$, sensitivity/specificity of 69%/79%[70]. Another showed a cut-off of GLS = 10.85% - OR 0.39 (0.26-0.57), $P < 0.01$, sensitivity/specificity of 89.7%/91.7%[71]. A cut-off for prediction of MACE ranged from GLS > -13% [HR = 2.34 (1.10-4.97), $P < 0.05$, sensitivity/specificity of 100%/89%][72] to GLS > -9.55% [OR = 0.56 (0.34-0.91), $P = 0.02$, sensitivity/specificity of 83.3/83.5%][71].

PEDSR was only measured in one study[73]. There was no significant difference in PEDSR in between patients that reached clinical endpoints and those that did not.

Strain measured by CMR

There were no studies that used CMR-based strain measurement techniques - either tagging or FT - to predict outcomes post-STEMI that matched our eligibility criteria.

DISCUSSION

This systematic review has shown that certain strain parameters measured by STE - namely, GLS[70-72,74,75] and GLS-R[69] - are independent predictors of adverse outcomes post-STEMI. Impaired GLS can predict both clinical endpoints and adverse LV remodelling, a surrogate marker of poor prognosis. When combined with routine clinical functional parameters such as EF and WMSI, strain provides incremental value in the prognostication of STEMI patients.

However, studies that monitored "hard" events such as mortality could not match the large sample size of $n > 1000$ that some authors believe is important for the evidence to be considered statistically robust[17]. Only one of the studies we assessed had such a large sample size - but the authors monitored remodelling and not "hard" events[74].

Some of the studies that monitored MACE had only a small number of patients that had reached their defined endpoints. Despite this, they constructed models for multivariate analysis that included a large number of independent variables (in addition to GLS). It is believed that one variable should be added for every 10 events to ensure that the regression estimates have reasonable precision[76]. Therefore, all of these studies may have included an inappropriately high number of variables to assess independent predictors of clinical endpoints and the models are likely to suffer from over-fitting.

PEDSR does not seem to provide any benefit at predicting these outcomes although has only been assessed in one study. Consequently, further studies are surely needed to determine if diastolic dysfunction has any role to play in prognostication after a STEMI[27].

Data in this review is limited to GLS measured by STE. We cannot comment on whether GCS is of any added value or has similar predictive properties as GLS since no studies assessed these two parameters together.

Evidence suggests that GLS measured by STE is related to IS[37,77]. The question remains as to whether GLS provides additional information to IS in post-

Table 4 All studies that have used speckle-tracking echocardiography-based strain to predict adverse left ventricular remodelling

Ref.	Age (yr)	Sample size (male)	Baseline ejection fraction (%)	Timeframe baseline scan	Timeframe follow-up scan(s)	Definition of adverse remodelling	Other parameters in multivariate model	Results	Limitations
Bochenek *et al*[70]	59.6 ± 10.3	66 (53)	49.7 ± 9.2	4-6 d post-infarct	3 mo	EDV > 20%	Diabetes Anterior MI Leuk. Count Time to reperfusion WMSI Max. Trop ST-elevation max pre-PCI	22 patients remodelled; GLS can predict LV remodelling - OR = 1.19 (1.04-1.37), $P < 0.05$ - shown by multivariate analysis GLS > -12.5% can predict remodelling - AUC = 0.77 for ROC, sensitivity/specificity of 69%/79% respectively	Only longitudinal strain measured. Too many variables in multivariate analysis
Joyce *et al*[74]	60 ± 12	1041 (792)	47.0 ± 9.0	2 d post-PPCI	3 and 6 mo	EDV ⩾ 20%	Male sex LAD infarct Max. Trop Discharge heart rate LA volume index WMSI	GLS > -15% can predict remodelling at 3 and 6 mo *vs* GLS < -15% (both $P < 0.001$): OR = 6.7 (2.8-11) for 3 mo; OR = 10 (6.7-14) for 6 mo	Only longitudinal strain measured; Prognostic data divided categorically - *i.e.*, GLS > -15% or < -15%; Excluded patients with re-infarction before follow-up and cardiogenic shock - could potentially have been used as another endpoint
Cong *et al*[71]	59.9 ± 11.6	127 (103)	51.8 ± 5.1	1 d post-PPCI	6-9 mo	ESV ⩾ 15%	Anterior MI Time to reperfusion ΣST before PPCI ΣST post-PPCI Raised CK-MB/Trops Baseline ESV/EF WMSI	41 patients developed remodelling; GLS predicted remodelling - OR = 0.39 (0.26-0.57), $P < 0.01$; GLS = -10.85% had sensitivity/specificity of 89.7%/91.7% respectively by ROC to predict remodelling	Only longitudinal strain measured; Too many variables in the multivariate analysis

AUC: Area under curve; CK: Creatine kinase; EDV: End diastolic volume; ESV: End systolic volume; GLS: Global longitudinal strain; LA: Left atrium; LAD: Left anterior descending; LV: Left ventricle; OR: Odds ratio; PPCI: Primary percutaneous coronary intervention; ROC: Receiver operator characteristic; WMSI: Wall motion score index; ΣST: Sum of ST-elevation.

STEMI prognostication and it can only be adequately answered using CMR. However, no studies were found that showed global strain measured by CMR could predict development of remodelling or MACE.

Limitations

We could rule out publication bias - unpublished data were not included as part of our review and could possibly affect our results, especially if it contradicted the seven studies that were assessed. We did not include three search results that were presented as posters since we could not access either the poster itself or the full-text articles associated with it. Regardless, we do not feel this exclusion would significant affect the results of the review since the titles of all three posters stated that GLS could predict post-STEMI outcomes. There is outcome data available in strain measured by TDI but we decided to exclude it from our review since its major limitation of "angle dependence" has been superseded by STE.

Conclusion

Global longitudinal strain when measured by STE is an independent predictor of both adverse LV remodelling and MACE after STEMI and provides incremental prognostic value when combined with traditional LV functional parameters such as EF and WMSI. No such data exist for CMR, but this modality could inform us as to whether strain provides prognostic data in addition to IS.

Table 5 All studies that have used speckle-tracking echocardiography-based strain to predict major adverse cardiac events

Ref.	Age (yr)	Sample size (male)	Baseline ejection fraction (%)	Timeframe baseline scan	Follow-up period	Outcome measures	Other parameters in multivariate model	Results	Limitations
Antoni *et al*[69]	60 ± 12	759 (517)	46.0 ± 8.0	2 d post-PPCI	21 ± 13 mo	GLS and/or GL-strain rate to predict: A: Mortality; B: Composite of revascularisation/ readmission for HF/re-infarction	Age (A) HTN (A) Multi-vessel disease (A/B) Peak Trop (A) QRS duration (A/B) EF (A/B) Severe MR (A) Smoking (B) Diabetes (B)	179 patients reached one or more endpoints; GLS independent predictor of all-cause mortality - HR = 1.2 (1.1-1.3), $P = 0.002$; GLS-R independent predictor of B endpoints - HR = 22 (11-48), $P < 0.001$; Both GLS and GLS-R independent predictors of combined A and B endpoints - HR = 1.1 (1 -1.1, $P = 0.006$) and 18 (10-35, $P < 0.001$) respectively	Sample size $n < 1000$ - potentially not large enough to predict "hard" events like mortality; Only longitudinal strain measured; SR analysis feasible in only 89% of segments
Shanks *et al*[73]	59.7 ± 11.6	371 (288)	45.2 ± 8.0	2 d post-PPCI	17.3 ± 12.2 mo	GL-PEDSR to predict: Mortality; Readmission for HF; Re-infarction; Revascularisation	EF TIMI 0-1 ESV-index Iso-volumetric relaxation SR	Combined clinical endpoints occurred in 84 patients; GL-PEDSR does not predict clinical outcomes	Sample size potentially too small to assess "hard" endpoint such as mortality; No measure of GLS; Only longitudinal parameters obtained
Woo *et al*[72]	64.4	98 (65)	52.6 ± 12.0	Pre-PPCI and 3 d post-PPCI	13.1 ± 3.8 mo	GLS to predict: Mortality; Readmission for HF	Initial Trop Initial NT-pro BNP EF (baseline) WMSI (follow-up) E/e'sr EF (follow-up) WSMI (follow-up)	7 patients developed endpoints; Pre-PPCI GLS predictor of outcomes - HR = 1.41 (1.01-1.98), $P < 0.05$; Post-PPCI GLS more likely to predict outcomes - HR = 2.34 (1.10-4.97), $P < 0.05$; Pre-PPCI GLS < 14% had sensitivity/specificity of 85%/75% respectively - post-PPCI GLS < 13% of 100%/89%	Very small sample size; Only longitudinal strain measured; Too many variables in multivariate analysis
Munk *et al*[78]	63.1	576 (446)	50.0 ± 10.0 (without composite endpoint), 47.0 ± 12.0 (with composite endpoint)	1 d post-PPCI	24 (IQ range 13-61) mo	GLS to predict: Mortality/re-infarction/ stroke/hospitalisation for HF; Crude mortality	EF WMSI ESV-index (Separately and in combination with each other)	162 patients experienced composite endpoints; GLS alone predicted outcomes within 1 yr post-MI - HR = 1.2 (1.12-1.29), $P < 0.01$; GLS alone could not predict outcomes later than 1yr post-MI	GLS could only be obtained in 74% of 576 patients - 26% excluded due to poor image quality (no difference in event rates, however); Only longitudinal strain measured
Cong *et al*[71]	59.9 ± 11.6	127 (103)	51.8 ± 5.1	1 d post-PPCI	16.9 ± 1.6 mo	GLS to predict: Mortality; Development of HF	Anterior MI Time to reperfusion ΣST before PPCI ΣST post-PPCI Raised CK-MB/Trops Baseline ESV/EF WMSI	GLS predicted outcomes - OR = 0.56 (0.34-0.91), $P = 0.02$; GLS > -9.55% had sensitivity/specificity of 83.3%/83.5% respectively	Sample size could potentially be too small to significantly predict "hard" events such as mortality

CK: Creatine kinase; ESV: End systolic volume; GLS: Global longitudinal strain; HF: Heart failure; OR: Odds ratio; HR: Hazard ratio; HTN: Hypertension; IQ: Inter-quartile range; MR: Mitral regurgitation; PEDSR: Peak early diastolic strain rate; PPCI: Primary percutaneous coronary intervention; TIMI: Thrombolysis in myocardial infarction; WMSI: Wall motion score index; ΣST: Sum of ST-elevation; MI: Myocardial infarction.

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COMMENTS

Background

Left ventricular (LV) dysfunction is an important determinant of prognosis following ST-elevation myocardial infarction (STEMI). Routinely used measures of LV dysfunction such as ejection fraction (EF) may not be able to detect subtle changes in cardiac function. Myocardial strain describes the relative change in length of myocardium through the cardiac cycle and is an objective measure of LV function. It can be measured during both systole and diastole and hence provides a reflection of both systolic and diastolic LV contractility. Acutely measured strain post-STEMI may help in predicting markers of poor prognosis [such as development of adverse LV remodelling or major adverse cardiac events (MACE)] at follow-up.

Research frontiers

Strain can be assessed using post-processing speckle-tracking echocardiography (STE) or cardiac magnetic resonance imaging (CMR)-based techniques [such as tagging or novel feature tracking (FT) software]. Such techniques can quantify strain at a segmental and global level and may provide additional information to LV volumes and EF.

Innovations and breakthroughs

This is the first paper to review the literature and present all the studies that have assessed acutely measured global strain parameters to predict markers of outcome post-STEMI. Three studies have shown that global longitudinal strain (GLS) measured by STE is a predictor of adverse remodelling following STEMI whilst four studies have shown that it can predict MACE at follow-up. Therefore, GLS may be a useful clinical measure of identifying patients at a "high risk" of developing poor outcomes. There were also no CMR-based studies assessing strain and its relation to prognosis following STEMI.

Applications

GLS may help improve risk stratification following STEMI but further studies are required to show that this improves outcome.

Terminology

Myocardial strain describes the relative change in length of myocardium through the cardiac cycle -GLS is a measure of LV contractility in the longitudinal vector; STE is an echocardiography-based post-processing software that analyses myocardial deformation parameters (such as global strain) by tracking the motion of "speckles" from one frame to another through the cardiac cycle; Tagging is a post-processing CMR-based software that evaluates strain on tagged sequences - examples of such sequences include spatial modulation of magnetisation (SPAMM) and complementary SPAMM; FT is a post-processing software that assesses strain on cine steady-state free precession images, a type of sequence that is routinely acquired during a clinical CMR scan.

Peer-review

The article is interesting, well-written and supported by updated references.

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