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Usefulness of Two-Dimensional Strain Echocardiography to Predict Segmental Viability Following Acute Myocardial Infarction and Optimization Using Bayesian Logistic Spatial Modeling

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Abstract

Viability assessment following acute myocardial infarction (MI) is important to guide revascularization. Two-dimensional strain echocardiography (2DSE) was shown to predict viability but the methodology assumed strain in each segment is independent of contiguous segments. We tested the hypotheses that segmental strain post-MI are spatially correlated and that using Bayesian approach improves prediction of non-viable myocardium. 21 subjects (58±12 years, 6 females) with ≥2 weeks MI underwent 2DSE and late gadolinium enhancement (LGE) cardiac magnetic resonance imaging within 48-hours of each other. The heart was divided into 16 segments and longitudinal, radial and circumferential strains were measured using software. Using similar segmentation, LGE was measured and segments with >50% LGE were considered nonviable. Spearman analyses assessed spatial correlation of strain and receiver operating characteristic curve analysis was used to determine prediction of non-viable myocardium without and with Bayesian logistic spatial conditionally autoregressive (CAR) model. There is significant spatial correlation in strain and LGE, especially in the apex. Longitudinal strain was the best predictor of non-viability and was impaired in non-viable myocardium (-12.1 ± 0.6 , -8.0 ± 0.6 and $-4.6 \pm 1\%$ for 0, 1-50, >50% LGE, respectively, $p < 0.001$). Use of CAR model improved the area under the curve for detection of non-viable myocardium (0.7 to 0.94). A CAR probabilistic score of 0.17 had 88% sensitivity and 86% specificity for detecting non-viable myocardium. In conclusion, longitudinal strain from 2DSE can predict myocardial viability following MI and exploiting spatial correlations in segmental strain using Bayesian CAR enhances the ability of 2D strain to predict non-viable myocardium.

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Keywords

myocardial infarction; strain echocardiography; viability; Bayesian analysis

Introduction

Two-dimensional strain echocardiography (2DSE) is a simple, cost-effective tool that utilizes speckle tracking of myocardial segments to measure tissue deformation or strain. In animal¹ and human subjects^{2,3}, 2DSE was shown to be sensitive and specific in detecting myocardial viability and is superior to conventional methods of left ventricular (LV) function evaluation for early detection of dysfunction^{4,5}. However, the variability and range of strain values in infarcted and normal regions within an individual and among different patients make it difficult to rely on a single individual cutoff value that would reliably dichotomize viable from non-viable myocardium. Since acute myocardial infarction (MI) results in abnormality in contiguous segments supplied by the involved coronary artery, the pathology has a spatial distribution that is ignored by current methods of analysis that treat the strain of individual segments as if they are independent of others. This limitation could potentially be ameliorated by exploiting spatial correlation of strain; in essence, giving greater weight to abnormal strain in a segment if adjacent segments are similarly abnormal. We hypothesize that segmental strain following MI demonstrates spatial correlation and that exploiting these correlations using Bayesian probabilistic approach will enhance 2DSE's ability to identify non-viable myocardium. Our aims are to determine segmental correlation in 2D strain and to compare the ability of 2DSE to predict nonviable myocardium without and with logistic spatial conditionally autoregressive (CAR) models that accounts for spatial correlations.

Methods

Twenty one subjects (58 ± 12 years, 6 females) who were diagnosed to have myocardial infarction from coronary artery disease by standard criteria (symptom, electrocardiography and cardiac enzymes) at least 2 weeks prior to recruitment were enrolled. The 2 weeks post-infarction period was selected to provide greater certainty that the amount of late gadolinium enhancement (LGE) on cardiac magnetic resonance imaging (CMR) post-infarct is stable because prior reports show that in the acute setting, the extent of LGE was greater than infarct size due to the additional contribution of edema and inflammation⁶. All subjects gave informed consent and the study was approved by the local Institutional Review Board. Patients who were claustrophobic, had ferromagnetic materials or significant renal dysfunction that contraindicated gadolinium administration were excluded. No patient was excluded for poor acoustic echocardiographic quality. The magnetic resonance imaging and echocardiography were performed either on the same day or within 48 hours of each other.

Two-dimensional echocardiography in the short axis (base or mitral level, mid or papillary muscle level and apex) as well as standard apical 4-, 2- and 3-chamber views were obtained using General Electric (Waukesha WI) Vivid-7 echo and M3S transducer at frame rates of 80-100 frames per second with second harmonic imaging. The data were then transferred for post-processing using Q analysis software in an EchoPACS workstation (General Electric).

Radial and circumferential strains were obtained from the short axis slices by outlining the endocardial border and selecting the myocardial width. The software then derived radial and circumferential strain for each segment utilizing standard American Society of Echocardiography (ASE) designation⁷. The radial and circumferential strains were correlated with LGE on CMR obtained in short axis slices.

Longitudinal strain was obtained from the apical long axis slices (2-, 4- and 3-chamber views). The user selected the basal mitral annular and apical regions; the software then traced the endocardial borders and myocardial width which the user had the option of manually adjusting as necessary to delineate the myocardium. The software then automatically calculated segmental longitudinal strain using standard ASE segmentation. The longitudinal strain was correlated with LGE on CMR obtained from similar long axis slices (Figure 1). Analyses of strain were performed by a cardiologist blinded to LGE CMR results.

For CMR, a General Electric 1.5 Tesla CV magnetic resonance imaging scanner with 8-channel cardiac coil was used. Infarct size was assessed by measuring the amount of LGE in a myocardial segment. LGE is a well-validated method of measuring infarct size that highly correlated with histologic measurement^{6,8,9} and is an established method for assessment of myocardial viability^{6,8,9}. Gadolinium (0.1 mmol/kg of gadolinium gadodiamide, GE Healthcare or gadobenate dimeglumine, Bracco Diagnostics) was injected and image acquisition was started after ~10 minutes post-injection. Cardiac gated inversion-recovery prepared gradient echo pulse sequence was used with the following parameters: field of view 38-42 cm, matrix size 256×192-256, slice thickness 7-8 mm, interslice gap of 2-3 mm (for short axis slices), inversion time from 175-300 ms adjusted to null normal myocardial signal, number of excitations 1-2 and 2-R-R intervals. The optimal inversion time that nulled the myocardium was determined by acquiring multiple images of a midventricular slice using different inversion times.

Images were obtained in both standard short axis slices (base just below mitral valve level to apex) as well as orthogonal long axis slices that corresponded to standard echocardiographic apical long axis views (4-, 2- and 3-chamber). Segmental designation utilized standard American Society of Echocardiography criteria⁷. Analysis of CMR images was performed by a CMR expert who was blind to echocardiography strain data.

The extent of LGE was measured in the short axis as well as long axis views. For short axis slices, ReportCard software (General Electric, Waukesha WI) was used to manually delineate the left ventricular endocardial border, epicardial border and posterior insertion point of the right ventricle to the left ventricle. Using a threshold of 2 times the minimal signal intensity in the myocardial slice, the software automatically selected regions with increased signal intensity (LGE) that corresponded to the infarct region. In rare cases, the threshold was increased up to 3 times the minimal signal intensity for the myocardial slice if the reader clearly thought that the lower threshold incorrectly designated normal regions as abnormal. This semiquantitative approach avoided spurious overestimation of infarct size and more closely followed clinical practice. The software then calculated the % area of infarct for each standard segment (total 16 segments). The short axis-derived late gadolinium enhancement was used to compare to short axis-derived radial and circumferential strain for 2DSE.

Long axis views orthogonal to the short axis were also obtained with standard 4-, 2- and 3-chamber views similar to apical views on echocardiography (Figure 1). The left ventricle was divided into 3 equal segments (basal, mid and apical) and the transmural extent of LGE was measured for each segment. Transmural extent was designated as the greatest width of subendocardial LGE in a segment divided by the segment width x 100%. The transmural extent of LGE on long axis was used to compare long axis-derived longitudinal strain for 2DSE.

Values are presented as means±standard error of mean for normally distributed data or median and 25-75% range for strain data that are not normally distributed. Strain values

were compared among groups using one-way analysis of variance for normally distributed data and Kruskal-Wallis analysis for non-normally distributed data (Sigmastat 3.5, Systat Corporation). Pairwise comparison was performed using Student-Newman-Keuls method (normally distributed data) or Dunn's method (non-normally distributed data). Correlation analyses were performed using Spearman's method.

The primary statistical goal was the prediction of the presence of LGE in > 50% of the myocardial segment on CMR (a level clinically accepted as threshold for non-viability¹⁰) using 2DSE derived radial, circumferential and longitudinal strain. The primary statistical difficulty is that measurements in different segments exhibited a spatial correlation structure. For example, as shown in Figure 2, segments within the apex are generally correlated. In order to account for this spatial correlation, we employed a hierarchical Bayes model using conditionally autoregressive (CAR) models¹¹. We modeled the probability of a particular segment having > 50% LGE on CMR using the Bayesian logistic CAR model with radial, circumferential, and longitudinal strain as covariates. Using the deviance information criterion (DIC), we selected the final model as follows:

$$\begin{aligned} \text{Logit}(p_{ij}) &= \alpha + \beta_i + \theta_j + \gamma \times \text{Longitudinal}_{ij} \\ &, \alpha \propto 1 \\ &, \beta_i \sim N(\mu_\beta, \tau_\beta) \\ &, \theta_j \sim \text{NormalCAR}(A, W, \tau) \\ &, \gamma \sim N(0, 0.001) \mu_\beta \sim N(0, 0.001) \\ &, \tau_\beta \sim \text{Gamma}(0, 0.001) \\ &, \tau \sim \text{Gamma}(0.5, 0.0005), \end{aligned}$$

where $i=1,2,\dots,21$ indexes patients and $j=1,2,\dots,16$ indexes segments within a patient. The $\text{Logit}(x)$ is the logit function and the $N(a, b)$ indicates the normal distribution with mean a and precision b . The $\text{Gamma}(c, d)$ is the gamma distribution. The θ_j 's represent spatial random effects that induce correlation amongst spatially neighboring heart segments. The β_i 's are patient-specific random effects allowing for changes in the baseline probability of >50% LGE for a particular patient. 'A' represents an adjacency matrix that defines the neighboring spatial structure of the different heart segments. Finally 'W' denotes a weight matrix where each element was set to 1. We also conducted sensitivity analyses of our results to the prior choices made. In general, we found that our results were rather insensitive to the choice of prior distributions, producing only slight variation in the predictive capability of the model. The spatial CAR models were fitted using the WinBUGS software¹², while all other analyses were performed using the R Statistical Software Language^{13,14}.

Results

The infarcts were in the left anterior descending artery territory in 6 (28%), circumflex in 1 (5%), right coronary artery in 2 (10%), two vessels in 5 (24%) and 3 vessels in 7 (33%). The infarct volume by LGE on CMR comprised $19.8 \pm 7.5\%$ of the myocardial volume. The mean segmental extent of LGE was $18 \pm 22\%$ on long axis views and $21 \pm 18\%$ on short axis slices ($p=\text{NS}$). Per subject, there were 2 ± 2 segments with >50% transmural extent of LGE. Late gadolinium enhancement demonstrated spatial correlation in contiguous segments, especially in the apical region (Figure 2A).

There were significant differences in longitudinal (overall $p < 0.001$), radial ($p < 0.001$) and circumferential strain ($p < 0.04$) among segments with no, 1-50% or >50% transmural infarct (Figure 3). Receiver operating characteristic (ROC) curve analysis of segmental strain demonstrated that longitudinal strain was better at predicting >50% LGE than radial or circumferential strain (AUC 0.7, 0.33, and 0.62, respectively) when each segment value was assumed to be independent of the others'. When the CAR model was utilized taking into account spatial correlation in segmental strain, longitudinal strain was the strongest predictor of >50% LGE (posterior mean 1.135, 95% C.I. 1.06-1.22); radial and circumferential strain did not provide significant additive predictive value to longitudinal strain. Using the CAR model, the area under the curve improved to 0.924 in prediction of >50% LGE (Figure 4). Using likelihood ratio to determine the cutoff value that would provide the optimal combination of sensitivity and specificity, longitudinal strain of -3.3% and CAR probabilistic score of 0.17 had 50% and 88% sensitivity, and 80% and 86% specificity, respectively, for detecting non-viable myocardium.

Discussion

There are 2 novel findings in this study. One, the spatial relations of segmental strain was quantified for the first time in post-MI patients and the contiguous spatial correlation in strain corresponded to similar spatial correlation in extent of segmental LGE on CMR, especially in the apex. Second, exploiting these spatial relations in segmental strain allowed a Bayesian approach CAR modeling to improve the ability of longitudinal strain to predict segmental myocardial non-viability with enhanced sensitivity and specificity.

Viability assessment post-MI is important for prognostication and more practically, to assess candidacy for surgical or percutaneous revascularization^{15,16}. Multiple studies of various imaging modalities demonstrated that revascularization of coronary arteries supplying non-viable myocardium did not result in functional improvement or improved survival^{15,17,18}. There are several methods of evaluating viability that have been validated using clinical and functional outcomes. Cardiac magnetic resonance imaging utilizes the presence of LGE; gadolinium is an extracellular contrast agent that has a higher volume of distribution in infarcted tissue compared to normal myocardium¹⁹. LGE closely corresponded to histologic infarct size and distribution^{6,8,9} and extent of LGE has been shown to predict segmental viability following revascularization^{6,8,9}. In practice, >50% extent of LGE in a segment is an arbitrary cutoff used by clinicians to define non-viability, based on the original study by Kim and colleagues¹⁰ that has since been validated by others¹⁹⁻²⁵. Cardiac MRI, however, is not widely available and cannot be used in patients with ferromagnetic materials, claustrophobia or abnormal renal function. Alternative techniques to assess viability, including 18-fluorodeoxyglucose positron emission tomography, thallium single photon emission computed tomography and dobutamine stress echocardiography are also limited by exposure to radiation, poor spatial resolution, need for an inotropic agent, subjectivity or cost¹⁵⁻¹⁸.

Two-dimensional strain echocardiography is a new technique to assess myocardial mechanics²⁶⁻²⁸. This method directly measures myocardial deformation or strain in the longitudinal, radial and circumferential directions. It has been validated with histology to be sensitive and specific in detecting myocardial dysfunction in various animal models of cardiomyopathy, including doxorubicin injury⁴ and ischemic cardiomyopathy^{1,5}, with excellent sensitivity and specificity in predicting viability post-MI¹. Strain derived from 2DSE was also useful in predicting functional recovery post-MI in human subjects that underwent revascularization^{2,3}. Viability assessment by 2DSE has advantages over current methods in that it is simple, portable, cost-effective and by its quantitative nature, is less subjective.

Strain values, however, demonstrate a wide range in both noninfarcted segments (LS: 10.3 to -29%, median -13%) as well as infarcted segments (>50% LGE LS: 12 to -30%, median -8%). This overlap in strain values between viable and nonviable segments reduces the sensitivity and specificity of the test, and if segments are considered independent of each other, the area under the curve for predicting nonviability was only 0.7 for longitudinal strain. A unique approach to overcome this limitation is to exploit the spatial correlation of strain among myocardial segments using probabilistic methods. Coronary artery disease has a predisposition to regional segmental distribution, depending on which coronary arteries are involved. In addition, there is usually apical involvement: this region is at risk following occlusion of the coronary artery at any point, be it proximal, mid or distal. First, we quantified this spatial correlation in segmental strain and then using Bayesian CAR methods created a model that assigned probability scores to each segment. This weighting, in essence, relied not only on the raw strain value in each segment, but also accounted for the strain of adjacent as well as remote segments in determining the probability that the segment is non-viable. Greater weight was given if contiguous segments were abnormal and remote or non-adjacent segments had higher values, thereby utilizing normal uninvolved remote segments as intrinsic controls. By using this probability score, instead of the raw longitudinal strain value, the ability to predict segments with >50% LGE was improved. Similar to the approach used in this study but using a different methodology, the Bayesian probabilistic modeling has been utilized in predicting cancer risk or recurrence from mathematical risk models of cancer genetics and survival outcomes^{29,30}. As far as we know, this report is the first to utilize Bayesian methods in segmental myocardial abnormalities.

The study has several limitations. One, the sample size is small and the model needs validation in a larger number of subjects, an area of future research. Second, sensitivity/specificity analysis is affected by the prevalence of the condition being tested. Thus, the results may not apply to patients with lesser or greater prevalence of non-viable segments. Thus the incremental value of Bayesian probabilistic approach over 2D strain should be tested in various CAD population groups. Despite this limitation, however, the specific group tested in this study represents the patient population where revascularization decisions need to be made, making it a clinically relevant group. In addition, we utilized LGE on CMR as a gold standard for non-viability. Indeed LGE has been well-validated using histologic infarct size gold standard as well as functional recovery^{6,8,9} in the evaluation of myocardial viability, making it a good surrogate marker of viability. However, the more clinically relevant gold standard outcome is segmental functional recovery following revascularization; this again, is an area for further study. Lastly, the echocardiographic and CMR images may potentially not be exactly co-registered. We tried to mitigate this limitation by utilizing standard ASE segmentation, ensuring that the short axis views are orthogonal to the long axis by acquiring left ventricular images that are as close to a round shape as possible, and replicating standard 4-, 2- and 3-chamber views seen on echo on the long axis CMR images.

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Figure 1.

Late gadolinium enhancement on CMR and longitudinal strain on 2DSE. The subject presented with occlusion of the left anterior descending coronary artery. A, C and E demonstrate near-transmural late gadolinium enhancement consistent with non-viability from scar. The corresponding long axis views on 2DSE are shown in B, D and F with parametric mapping of the longitudinal strain. The color map scale is shown in the right upper corner of the image. Areas of non-viability from CMR demonstrate reduced longitudinal strain.

Correlation Analyses

Figure 2.

Spatial correlation of late gadolinium enhancement and strain. Spearman correlation analyses of late gadolinium enhancement (A), longitudinal (B), circumferential (C) and radial (D) strain among 16 myocardial segments (x and y axes) are shown and gray scale-coded with scale shown on the right side. LGE and strain demonstrate spatial relationships among contiguous segments that is more pronounced in the apical region.

Figure 3.

Strain and extent of late gadolinium enhancement. Longitudinal (A, $p<0.001$), radial (B, $p<0.001$) and circumferential strain (C, $p<0.04$) were significantly different in regions with >50%, 1-50% and no infarct. The relationship was also seen in circumferential strain (C) but strain was only significantly different between segments with >50% versus 1-50% transmural LGE.

Receiver Operating Characteristic Curve Analysis

Figure 4. Receiver operating characteristic curve analysis showing that the area under the curve for Bayesian analysis using CAR model is greater than 2DSE longitudinal strain alone for predicting >50% transmural LGE on CMR, thus signifying superior sensitivity and specificity.