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Acute myocardial infarction: Susceptibility-weighted cardiac MRI improves detection of reperfusion hemorrhage

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Introduction

Acute ST elevation myocardial infarction (STEMI) usually occurs as a consequence of thrombotic occlusion of an epicardial coronary artery and is a leading cause of heart failure and cardiovascular mortality. A wavefront of ischaemic necrosis propagates from the endocardial to epicardial myocardium and the aim of reperfusion therapy is to restore coronary flow and limit infarct size. The preferred means to achieve this is primary percutaneous coronary intervention (PPCI) (1), however the process of restoring blood flow to ischaemic myocardium can itself induce ischaemia-reperfusion injury which may paradoxically reduce the beneficial effects of myocardial reperfusion (2, 3). A candidate marker for reperfusion injury is the presence of intra-myocardial haemorrhage (IMH) which is a specific finding that occurs only after flow has been restored to severely ischaemic myocardium (4–6).

Haemorrhage is a frequent complication following reperfusion of acute myocardial infarction and is an independent predictor of adverse left ventricular (LV) remodelling regardless of the initial infarct size (7). Blood degradation products within the infarct can be detected on T2 and T2* weighted imaging as a region of signal loss, but low image contrast and motion artefact at longer echo times limit their sensitivity (8–10). Susceptibility weighted imaging (SWI) exploits magnetic susceptibility differences between tissues,

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resulting in a phase change between regions containing paramagnetic deoxygenated blood products and surrounding tissue (10, 11).

The aim of this study was to assess whether cardiac SWI could improve the conspicuity of myocardial haemorrhage as a marker of acute ischaemia-reperfusion haemorrhage.

Materials and Methods

Participants

The study was approved by the hospital research ethics committee and all participants gave written informed consent. We prospectively investigated 30 patients (30 male; mean age 53 years) who had undergone PPCI within the previous 7 days.

The inclusion criteria were men and women aged 18–85 with an ECG diagnosis of acute ST-elevation myocardial infarction and coronary intervention within 10 hours of symptom onset. Exclusion criteria were contraindications to cardiac magnetic resonance (CMR) imaging, previous myocardial infarction (MI) or heart failure, clinical instability, significant arrhythmias, pregnancy or lactation. All patients had presented within 24 hours of the onset of chest pain for treatment of a first myocardial infarction. The diagnosis of MI required ST elevation of >1mm in two contiguous ECG leads and chest pain onset within 12 hours of presentation (up to 24 hours in case of ongoing symptoms).

Phantom model of haemorrhage

To simulate the presence of blood products serial dilutions of a carbohydrate-coated, superparamagnetic iron oxide (SPIO) nanoparticle containing 30 mg/ml of iron (Ferumoxyl, Takeda, Osaka, Japan) were prepared using 0.05ml, 0.1 ml, 0.15 ml, 0.2 ml, 0.25 ml and 0.3 ml of the agent in 100 ml deionized water with the addition of a gelling agent (Sigma-Aldrich, St. Louis, MO) (12). The agarose solutions were poured into 50 ml tubes and placed in a hexagonal array surrounded by a pure agar solution with a control tube without Ferumoxyl at the centre. Imaging was performed to assess the effect of iron concentration on SWI and magnitude-only reconstructions.

Image acquisition

The CMR studies were performed on a 1.5T Philips Achieva system (Best, The Netherlands). The maximum gradient strength was 33 mT/m and the maximum slew rate was 160 mT/m/ms. A five-element cardiac phased-array coil was used for signal reception.

Scout images were obtained and used to plan a short axis stack of balanced steady-state free precession cine images of the LV from base to apex.

Myocardial oedema was imaged by using a breath-hold black-blood T2-weighted turbo spin-echo sequence with spectral pre-saturation with inversion recovery (SPIR) fat suppression with the following parameters: TR two R-R intervals, TE 100 ms; voxel size, 1.9 x 2.6 x 10 mm; flip angle, 90°; section thickness, 10 mm; and bandwidth, 467 Hz/pixel.

A dual-inversion black-blood gradient multi-echo T2* sequence was acquired as a single short axis section positioned at the mid-point of the infarct in the long-axis direction using the following parameters: TR 17 ms, TE 2.3–16.1 ms; seven echoes obtained; change in echo time, 2.3 ms; voxel size, 1.7 x 2.8 x 10 mm; flip angle, 20°; section thickness, 10 mm; and bandwidth, 1387 Hz/pixel. Cardiac triggering was set for mid-diastole to reduce motion artefact. The SPIO phantom was positioned in the bore such that the tubes were perpendicular to B0. A single transverse plane through the phantom was acquired using the same parameters and reconstructions as for the clinical imaging.

An intravenous bolus of gadolinium dimeglumine (Magnevist; Bayer Schering Pharma, Berlin, Germany) was administered at a dose of 0.2 mmol/kg via a power-injector (Spectris Solaris; Medrad, Indianola, Pa). Early enhancement was performed within 1 minute of contrast injection using a 3 dimensional inversion-recovery breath-hold sequence of the whole LV using the following parameters – TR 4.3 ms, TE 1.4 ms; voxel size 1.4 x 1.4 x 8 mm, flip angle 15°, slice thickness 8mm, bandwidth 266 Hz/pixel, inversion time 400 msec. Late gadolinium enhancement (LGE) was performed 10 minutes after contrast injection using a 2D inversion-recovery sequence using the following parameters - field of view, 320 x 320 mm; matrix 200 x 147; TR 5.5 ms, TE 2.5 ms; in-plane resolution 1.6 x 2.2 mm; section thickness 8 mm. The inversion time was adjusted to null the signal from normal myocardium using a Look-Locker method.

Image processing

The technique of SWI exploits phase shifts in the high-pass filtered (HPF) image to produce a phase mask, which yields an increase in image contrast (10). Image processing of the complex data from the gradient echo sequence was performed off-line using analysis software (Matlab 2012, Mathworks, Natick, MA). In the phantom experiment circular regions of interest (ROI) were drawn around each tube in the array including the interface with the background region. In the clinical images ROI were drawn around the infarcted and remote myocardium on the gradient echo images, defined by the hyperintense zone on the corresponding LGE images, by an observer with 10 years of CMR experience. HPF images were reconstructed to remove low spatial-frequency variations in phase and the mean absolute phase in radians was determined for each ROI. A phase mask was created from the HPF phase map using a triangular function raised to the 4th power. The original modulus image was multiplied by the phase mask to produce susceptibility-weighted images for each TE. CNR was calculated at each echo time by comparing the signal intensities within infarcted and remote tissues as follows:

$$CNR = \frac{|\overline{SI}_{remote} - \overline{SI}_{infarct}|}{SD(SI_{remote})}$$

where $\overline{SI}_{infarct}$ is the mean signal intensity within the infarct ROI and $\overline{SI}_{remote}$ is the mean signal intensity within the remote ROI.

A pixel-by-pixel analysis was performed of the multi-echo T2*-weighted modulus images with fitting to a simple monoexponential decay using the following equation:

$M(TE) = M_0 e^{-TE/T2^*}$ where $M(TE)$ is the magnitude signal intensity at echo time TE and $T2^*$ the decay constant (13).

Patients were classified into those with or without haemorrhage (independently of the findings on $T2^*$ or SWI) by a consensus of two experienced readers. The presence or absence of a hypointense zone within the infarcted territory on early enhancement or LGE that corresponded with focal hypointensity on $T2$ -SPIR within ischaemic myocardium was used to identify haemorrhage. While gradient echo itself is the most sensitive sequence for identifying blood products these features on $T2$ spin-echo and post-contrast imaging will identify nearly all cases of haemorrhagic microvascular damage (14). Infarct size was semi-automatically quantified using segmentation software (Segment CMR version 1.9, Medviso, Sweden) (15).

Volumetric assessment of the left ventricle was performed using analysis software (CMRtools, Cardiovascular Imaging Solutions, London, UK). LV mass, end diastolic volume and end systolic volume were calculated and indexed to body surface area.

Inter-observer agreement was performed by two independent readers, with 10 and 8 years of CMR experience respectively, reviewing randomized source and SWI reconstructions to determine the first echo at which a focal area of hypointensity was discernible. The observers were allowed to window and magnify the images as required.

Statistical Analysis

Normally distributed data were reported as mean $\pm$ one standard deviation (SD) or otherwise as median and interquartile range (IQR). The data were analysed using mixed models (SAS 9.3, SAS Institute, Cary, NC) to account for the correlation between echo numbers within patients and reconstruction methods. Log transformation of phase was used as the data were skewed. Other data were compared using a Wilcoxon test for paired samples or a Mann-Whitney test for independent samples. Inter-observer agreement was assessed using the linearly weighted kappa statistic. All tests were two-sided and a P value $<.05$ was considered indicative of statistical significance.

Results

Phantom study

An image of the SPIO phantom array is shown in figure 1. The solutions had $T2^*$ decay constants of 5.1 ms, 6.3 ms, 7.6 ms, 9.0 ms, 13.3 ms and 21.0 ms, with the control tube's $T2^*$ being 63.4 ms. Phase increased with echo time and SPIO concentration ($P<.0001$). CNR was greater for SWI reconstructions than magnitude-only source images for all echo times ($P<.0001$).

Clinical data

CMR was performed at a mean of 3.5 ± 2.3 days (1-7 days) post-PPCI and imaging was well tolerated by all subjects. The differences between patient groups are shown in Table 1. The coronary vessels that received intervention were – left anterior descending $n=19$, right

coronary artery $n=9$ and circumflex $n=2$. Transmural oedema was present in all subjects on the T2-SPIR images and was in the same anatomical region as the LGE. No patients had late enhancement or oedema in more than one territory. T2* in haemorrhagic infarcts was shorter than in non-haemorrhagic infarcts (33.5 ms (24.9 – 43.0) vs 49.9 ms (44.6 – 67.6); $P=.0007$). The difference between T2* in non-haemorrhagic infarcts and all remote segments was not significant (49.9 ms (44.6 – 67.6) vs 44.9 ms (38.8 – 51.4); $P=.16$).

Susceptibility effects within haemorrhagic myocardium caused a progressive phase change compared to a smaller oscillatory phase variation in remote myocardium (Figures 2 and 3). There was a significant effect of echo number on phase ($P<.0001$) and also a significant difference between the phase in haemorrhage and non-haemorrhage groups ($P=.0016$) (figure 4). SWI reconstruction had a significant effect on improving CNR at all echo times (echoes 1 - 5, $P<.0001$; echo 6, $P=0.01$; echo 7, $P=0.02$) (figure 5). The median echo number at which haemorrhage was first visible by the observers was less for SWI compared to source images (echo 2 vs echo 5, $P=.0002$) with inter-observer kappa coefficients of 0.85 and 0.80 for the respective reconstructions.

Discussion

In this study we have demonstrated that cardiac SWI is a feasible technique for detecting reperfusion haemorrhage following PPCI at 1.5T. Blood products within haemorrhagic myocardium have different susceptibilities to adjacent tissue and their signal develops a phase change as echo time increases. This results in an improvement of contrast on phase-masked magnitude images and visibility of haemorrhagic ischaemia-reperfusion injury at shorter echo times.

IMH is emerging as one of the most powerful predictors of poor outcome following PPCI for STEMI and is associated with adverse ventricular remodelling, arrhythmic risk and major cardiovascular events (16–18). Macroscopic haemorrhage does not occur in the absence of reflow (19) and as such it may provide an imaging biomarker of lethal reperfusion injury which is paradoxically develops after the restoration of coronary blood flow (20). Haemorrhage occurs within myocardium that has already progressed to the stage of severe ischaemic necrosis when reflow is re-established (21, 22) and is typically observed following revascularization of transmural infarctions with microvascular damage (23–25). Blood degradation products within haemorrhagic tissue, mainly intracellular deoxyhaemoglobin and methaemoglobin, cause magnetic field inhomogeneities leading to a loss of phase coherence as echo time increases (26).

Previous work has shown that IMH causes a phase change in myocardium and that susceptibility effects can be detected at 3.0T field strength (27, 28). In this study we have translated SWI techniques that are widely used in neuroimaging to detect micro haemorrhages in traumatic brain injury and applied them to cardiac imaging making full use of both the magnitude and phase data (29). The phantom study demonstrated the potential of this technique at 1.5T by demonstrating that a phase change occurs which is dependent on both echo time and the concentration of SPIO. The clinical study also showed that reperfusion haemorrhage was associated with a phase change consistent with local

susceptibility effects of paramagnetic blood products (27). The phase change increased progressively within the range of echo times used by conventional T2*-weighted multi-echo sequences (30, 31). This phase change allows a significant improvement of contrast to be obtained from SWI reconstructions compared to conventional magnitude-only imaging. Combining both magnitude and phase data in these fully susceptibility-weighted datasets offers an appealing method for maximizing sensitivity for the detection of myocardial haemorrhage.

Our study has shown that SWI can be readily adopted in routine cardiac clinical imaging at 1.5T and as well as post-infarct imaging may have other uses where assessment of focal tissue iron is required such as in stem cell tracking (32). Further research will be required to determine the role of cardiac SWI in risk stratification post-STEMI and whether the detection of smaller micro-haemorrhages is predictive of a poorer outcome. While the presence of MVO or reperfusion haemorrhage does not presently influence patient management it may have a role in assessing cardio-protective strategies that aim to limit the consequences of ischaemia-reperfusion injury (33).

Study limitations

We examined a small number of subjects in this feasibility study but this was sufficient to demonstrate a clinically significant difference in image contrast using SWI. Susceptibility artefacts present on T2*-weighted images, occurring at the inferolateral wall due to venous blood or at the heart-lung interface, will be exaggerated on SWI. However, as these are typically epicardial they can be readily distinguished from myocardial haemorrhage. A characteristic of SWI is that if the size of the susceptibility variation is smaller than a voxel then its signal will interfere against that of neighbouring voxels to create a strong partial volume effect. As we did not have histopathologic correlation we were not able to assess how accurately the area of signal loss corresponded to extravasated red cells. Similarly, the phantom study was not of the same physical dimensions as a human infarct and the boundary susceptibility effects will be different. As we were comparing two gradient echo sequences for the direct assessment of tissue blood products we stratified patients by the combination of T2 hypointensity and microvascular obstruction as an indicator of those likely to have reperfusion haemorrhage. Although this will identify 95% of subjects with haemorrhage a minority will only have blood products detectable on T2* weighted imaging (34). We were also not able to determine if SWI could detect haemorrhage that was not apparent using conventional sequences without a histological gold standard. We studied patients in the first week after STEMI and the maturation of blood degradation products may have influenced their signal characteristics. We included the whole infarct on our T2* measurements, and so this value will be longer than other studies evaluating only the haemorrhagic core. Due to the sample size and patient population all the subjects were male and further research is needed to establish if the technique is effective in a larger population.

In conclusion, cardiac SWI offers a new technique for improving the conspicuity of reperfusion haemorrhage that may be of value in assessing ischaemia-reperfusion injury following STEMI.

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The study was approved by the hospital research ethics committee and all participants gave written informed consent.

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Figure 1. Phantom array

The phantom array consisted of six tubes with increasing concentrations of superparamagnetic iron oxide solution (A – highest to F – lowest concentration) with a control tube at the centre. This was designed to model the effects of different concentrations of paramagnetic blood products.

Figure 2. Cardiac MR images of a 51 year old male patient following successful primary coronary intervention of a left anterior descending coronary artery occlusion.

A) A four chamber late gadolinium enhancement image shows an extensive area of microvascular obstruction within the interventricular septum (arrow heads). Apical mural thrombus of similar signal characteristics is also visible (arrow). B) A high-pass filtered phase image in the mid-ventricular short axis plane shows a region of phase change within the infarcted septum. C) A plot to show the variation of mean absolute phase with echo time (TE 2.3 – 16.1 ms, TE 2.3ms) within this patient's infarcted and remote myocardium.

Figure 3. Cardiac MR images in the mid-ventricular short axis plane of a 58 year old male patient following successful primary coronary intervention of a right coronary artery occlusion. A) Gradient echo image demonstrating subtle signal loss within the inferior wall (arrow). B) T2* parametric map showing focal area of shortened T2* relaxation (arrow). Pixels with a T2* < 20 ms are depicted in red. C) High pass filtered phase image demonstrating a phase change within the inferior and inferoseptal walls (arrows). D) Susceptibility weighted image demonstrating the enhanced contrast of haemorrhage within the infarct (arrows). E) Late gadolinium enhancement image demonstrating a comparable area of microvascular obstruction (arrows).

Figure 4. Bar chart of phase in infarcted myocardium

The bar chart shows the mean absolute phase at each echo time (TE 2.3 – 16.1 ms, TE 2.3ms) within infarcted myocardium in both haemorrhagic (n=16) and non-haemorrhagic (n=14) infarcts (with 95% confidence intervals).

Figure 5. Contrast between infarcted and remote myocardium.

A plot to show the contrast to noise ratios within infarcted and remote myocardium in haemorrhagic infarcts using susceptibility weighted and source data magnitude-only reconstructions (with 95% confidence intervals). An improvement in contrast was present at all echo times, but the effect was diminished at the longest echoes.