Characterizing Myocardial Edema and Hemorrhage Using Quantitative T2 and T2* Mapping at Multiple Time Intervals Post ST-Segment Elevation Myocardial Infarction

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Background—Accurate characterization of the longitudinal trends of myocardial edema and hemorrhage has been previously limited by subjective qualitative methods. We aimed to prospectively characterize the evolution of myocardial edema and hemorrhage post acute myocardial infarction using quantitative measures.

Methods and Results—Sixty-two patients were enrolled post primary percutaneous coronary intervention and underwent cardiovascular magnetic resonance on a 1.5-T scanner at 48 hours, 3 weeks, and 6 months. Myocardial edema and hemorrhage were assessed by T2 and T2* mapping, respectively, in both infarct segment (IS) and remote segment (RS). At 48 hours, T2 is higher in IS compared with RS (56.7 ms versus 43.4 ms; P<0.01). At 3 weeks T2 remains higher in IS compared with RS (51.8 ms versus 39.5 ms; P<0.01), and subsequently equalizes by 6 months (39.8 ms versus 39.5 ms; P=nonsignificant). T2 is also increased in RS at day 2 versus 3 weeks (43.4 ms versus 39.5 ms; P<0.01). At 48 hours T2* was reduced in IS compared with RS (32.4 ms versus 37.4 ms; P<0.01). At 3 weeks (IS, 37.7 ms versus RS, 38.4 ms; P=nonsignificant) and 6 months (IS, 37.3 ms versus RS, 38.2 ms; P=nonsignificant), T2* values were equal in both segments.

Conclusions—Quantification of myocardial edema and hemorrhage by T2 and T2* mapping is feasible post acute myocardial infarction and demonstrates that hemorrhage resolves faster than edema. Noninfarcted segments can also demonstrate edema in the acute phase possibly due to global hyperemia. (Circ Cardiovasc Imaging. 2012;5:566-572.)

Key Words: edema ■ hemorrhage ■ magnetic resonance imaging ■ myocardial infarction

Cardiovascular magnetic resonance (CMR) imaging has gained clinical importance in the noninvasive assessment of myocardial injury parameters post acute myocardial infarction. Histopathological assessment of infarcted myocardium demonstrates that edema, hemorrhage, and microvascular obstruction (MVO) are implicated in the remodeling process.1,2 Furthermore, these parameters have prognostic implications.3–6

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Quantitative T2 and T2* mapping techniques may offer increased accuracy in the detection of myocardial edema and hemorrhage by overcoming the known problems associated with T2-weighted imaging. Our group has recently shown that regional, longitudinal, and cross-subject comparisons of edema and hemorrhage are possible using quantitative T2 and T2* mapping techniques in a porcine model of post myocardial infarction remodeling.7 However, similar data in patients describing the natural time course of myocardial edema, hemorrhage, and MVO after an acute myocardial infarction are limited. One recent study has shown that infarct size and edema decreased significantly between day 2 and 1 week post reperfused acute myocardial infarction.8 Also, a canine study has shown that the extent of MVO and infarct size increased significantly over the first 48 hours after acute myocardial infarction.9

The knowledge of the relative resolution of myocardial damage and its impact on remodeling processes using quantitative techniques would be important in grading severity, evaluating treatment strategies, and potentially improving clinical outcomes. Therefore, the objective of our study was to prospectively characterize the evolution of myocardial edema by T2 quantification and myocardial hemorrhage by T2* quantification at both early and late time intervals post acute myocardial infarction.

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Methods

Study Population
Consecutive patients presenting to Sunnybrook Health Sciences Centre in Toronto with ST-segment elevation myocardial infarction (STEMI) between July 2009 and June 2011 were screened and prospectively enrolled. The main inclusion criteria was patients who met the standard diagnostic criteria for STEMI. All patients had undergone primary percutaneous coronary intervention as part of a regional program that triages patients with STEMI to our center for early revascularization. Exclusion criteria included significant arrhythmias, significant renal dysfunction (estimated glomerular filtration rate <30 mL/min), and typical contraindications to CMR imaging such as pacemakers and implantable cardioverter-defibrillators. Written consent was obtained from all patients, and the study was approved by the ethics review board of Sunnybrook Health Sciences Centre.

Periprocedural Protocol and Analysis
All patients were pretreated before revascularization with aspirin 162 mg and clopidogrel 600 mg. Choice of anticoagulant (intravenous heparin or bivalirudin) and optional use of glycoprotein IIb/IIIa inhibitor were left to operator discretion. In accordance to our institutional practice, thrombus aspiration was used routinely. Subsequent to the procedure, patients received aspirin, clopidogrel, β-blockers, angiotensin-converting enzyme inhibitors, angiotensin-receptor blockers, and statins as per standard of care.

Myocardial blush grade, thrombus score, and Thrombolysis in Myocardial Infarction flow grade were estimated visually by 2 experienced blinded observers.11–15 ST-segment resolution, defined as ≥70% reduction in the sum of the ST-segment elevation score between electrocardiograms obtained before primary percutaneous coronary intervention and immediately after the procedure, was also recorded.

CMR Acquisition Protocol
Each patient was imaged at 3 time points: within 48 hours, 3 weeks, and 6 months post STEMI. Studies were performed on a 1.5-T clinical scanner (Signa Twinspeed HDx; GE Healthcare, Waukesha, WI) with image acquisition performed using electrocardiographic triggering, an 8-channel receive coil and breath holds at end-expiration. Cardiac function was evaluated with 10 to 12 short-axis slices imaged with a balanced steady-state free-precession sequence in cine mode (FIESTA, GE Healthcare) with the following parameters: repetition time=3.7 ms; echo time=1.6 ms; flip angle=45°; slice thickness=8 mm; acquisition matrix=256×192; field of view=35 cm; bandwidth=125 kHz; and 20 cardiac phases per slice. T2 quantification was performed for characterization of myocardial edema using a previously validated free-breathing cardiac-gated spiral imaging sequence with T2 preparation and the following typical parameters: 5 short-axis slices prescribed on the basis of the angiographically determined occlusion territory; echo times=2.9, 24.3, 88.2, and 184.2 ms; 12 spiral interleaves with 4096 points each (within 48 hours), defined as the segment contralateral to the IS, by drawing manual contours over the corresponding maps and visually inspecting viability on the contrast-enhanced T1-weighted images. End-diastolic wall thickness has been shown to be able to identify and monitor the presence, extent, and resolution of myocardial edema after primary percutaneous coronary intervention.15,16 Therefore, we measured end-diastolic wall thickness in the IS as an indicator of myocardial edema in the acute phase and scar formation in the chronic phase.

Infarct size was quantified in a contrast-enhanced T1-weighted image using the full-width-half-maximum technique as previously described.17 whereas MVO was identified as a region of hypoenhancement within the region of the hyperenhanced infarct, 10 minutes post contrast administration. Both infarct size and MVO were expressed as a percentage of total myocardium. Data fitting for T2/T2* maps and infarct/MVO size computation were performed with custom-written scripts developed in MATLAB (The Mathworks, Natick, MA).

Statistical Analysis and End Points
End points were the degree of myocardial edema (T2 and end-diastolic wall thickness measurements) and myocardial hemorrhage (T2* measurement) in the IS compared with the RS over the course of the 3 time points. We also stratified patients by whether or not they had significant myocardial hemorrhage in the IS at the first time point (within 48 hours), defined as T2* <30 ms, and compared their T2 values over time in the IS and RS. The threshold of T2* <30 ms was chosen on the basis that this value was 2 SDs below the mean T2* measurement in the RS. Additional end points included the presence of MVO at the first time point, infarct size and parameters of left ventricular remodeling including left ventricular end-diastolic volume index, left ventricular end-systolic volume index, left ventricular stroke volume index, left ventricular ejection fraction, and IS systolic wall thickening at the final time point of 6 months post STEMI. Continuous data were expressed as means SD. For skewed variables, median and interquartile range were reported instead. Continuous data were analyzed in SAS version 9 (SAS Institute Inc, Cary, NC) using a mixed procedure, with adjustment for repeated time and segment of the same patients. A Tukey-Kramer adjustment for multiple comparisons was also applied. In analyses where the interaction between time and segment was significant, comparisons were also made by analyzing time and segment separately. When appropriate, continuous data were analyzed by the Student t test, specifically to compare parameters of left ventricular remodeling at 6 months, between patients who had hemorrhage or MVO acutely. Categorical variables were reported as frequencies and percentages and analyzed by the χ2 or Fisher exact test, as appropriate. All tests were 2-tailed, and statistical significance was accepted at P<0.05. Correlation analysis using Pearson correlation coefficient was done between the 2 measures of acute myocardial edema (T2 measurement and the difference in end-diastolic wall thickness between IS and RS).

Results
Ninety patients were screened and 62 patients were prospectively enrolled. Reasons for exclusion were: impaired renal function (5 patients), contraindications to CMR (4 patients), and patient refusal (19 patients). The demographics and clinical characteristics of the enrolled patients are presented in Table 1. The median symptom-to-balloon time and door-to-balloon time...
Conversely, 17 (28%) patients had MVO present without significant hemorrhage. Parameters of left ventricular remodeling at 6 months are listed in Table 3.

Figure 1 demonstrates the evolution of T2 values over time in both the IS and RS. At 48 hours, T2 is higher in the IS compared with the RS (56.7 ms versus 43.4 ms; \( P<0.01 \)) suggestive of myocardial edema. At 3 weeks T2 remains higher in IS compared with RS (51.8 ms versus 39.5 ms; \( P<0.01 \)) and subsequently equalizes by 6 months (39.8 ms versus 39.5 ms; \( P= \text{nonsignificant} \ [\text{NS}] \)). T2 is also increased in RS at day 2 versus 3 weeks (34.3 ms versus 39.5 ms; \( P<0.01 \)).

End-diastolic wall thickness, a marker of myocardial edema in the acute phase and a marker of myocardial scar formation in the chronic phase, had a similar evolution over time as T2 values (Figure 2). At 48 hours, IS end-diastolic wall thickness was higher (10.5 mm versus 7.0 mm; \( P<0.01 \)), but lower at 3 weeks (6.0 mm versus 8.0 mm; \( P<0.01 \)) compared with RS. At 6 months, myocardial edema had resolved and myocardial scar had formed in the IS resulting in a lower end-diastolic wall thickness (4.7 mm versus 8.5 mm; \( P<0.01 \)) compared with RS. At 48 hours, there was a moderate degree of correlation (\( r^2=0.72; \ P<0.01 \)) between the T2 measurement in the IS and the difference in end-diastolic wall thickness between IS and RS.

T2* evolution over time is depicted in Figure 3. At 48 hours, T2* was reduced in the IS compared with RS (32.4 ms versus 37.4 ms; \( P<0.01 \)). At 3 weeks (IS, 37.7 ms versus RS, 38.4 ms; \( P=\text{NS} \)) and 6 months (IS, 37.3 ms versus RS, 38.2 ms; \( P=\text{NS} \)), T2* values were equal in both segments.

We further stratified patients as to whether or not there was evidence of significant myocardial hemorrhage at the first time point of 48 hours (T2* <30ms) and examined their T2 measurements over time (Figures 4 and 5). Forty-one patients had no significant hemorrhage, whereas 21 patients did have. Patients without hemorrhage had a higher T2 value in the IS at 48 hours compared with 3 weeks (58.4 ms versus 51.9 ms; \( P<0.01 \)). In contrast, patients with hemorrhage had equivalent T2 values in the IS at both time points (53.4 ms versus 51.7 ms; \( P=\text{NS} \)). Both group of patients had similar T2 in the RS at 48 hours compared with 3 weeks (hemorrhage group: 43.7 ms versus 40.6 ms, \( P=\text{NS} \); no hemorrhage group: 43.6 ms versus 41.6 ms, \( P=\text{NS} \)).

At 6 months, patients with hemorrhage had a lower IS end-diastolic wall thickness (4.2 mm versus 5.7 mm; \( P=0.008 \), lower IS systolic wall thickening (23% versus 62%; \( P=0.001 \)), lower left ventricular ejection fraction (44.9% versus 55.3%; \( P=0.001 \)) compared with patients without hemorrhage. Other parameters of left ventricular remodeling including infarct size (16.3% versus 13.1%; \( P=0.43 \)), left ventricular end-diastolic volume index (81.8 mL/m² versus 75.2 mL/m²; \( P=0.40 \)), left ventricular end-systolic volume index (45.6 mL/m² versus 33.4 mL/m²; \( P=0.06 \)), and systolic volume index (35.5 mL/m² versus 41.8 mL/m²; \( P=0.13 \)) did not show a statistically significant difference between the 2 groups.

At 6 months, patients with MVO had a lower left ventricular ejection fraction (49.1% versus 57.6%; \( P=0.003 \)) compared with patients without MVO. Other parameters of left ventricular remodeling including infarct size (17.3% versus 9.2%; \( P=0.06 \)), IS end-diastolic wall thickness (4.6 mm versus

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### Table 1. Baseline Clinical Characteristics of the Study Population

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Total (n=62)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>60±11</td>
</tr>
<tr>
<td>Male, %</td>
<td>54 (87)</td>
</tr>
<tr>
<td>Diabetes mellitus, %</td>
<td>17 (27)</td>
</tr>
<tr>
<td>Hypertension, %</td>
<td>27 (44)</td>
</tr>
<tr>
<td>Dyslipidemia, %</td>
<td>16 (26)</td>
</tr>
<tr>
<td>Smoking, %</td>
<td>17 (27)</td>
</tr>
<tr>
<td>Prior myocardial infarction, %</td>
<td>2 (3)</td>
</tr>
<tr>
<td>Prior PCI, %</td>
<td>4 (6)</td>
</tr>
<tr>
<td>Prior CAGB, %</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Creatinine, umol/L</td>
<td>83±23</td>
</tr>
</tbody>
</table>

**Notes:** PCI indicates percutaneous coronary intervention; CAGB, coronary artery bypass grafting.

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### Table 2. Periprocedural Characteristics and Outcomes of the Study Population

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Total (n=62)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infarct artery, %</td>
<td></td>
</tr>
<tr>
<td>LAD</td>
<td>27 (44)</td>
</tr>
<tr>
<td>LCx</td>
<td>12 (19)</td>
</tr>
<tr>
<td>RCA</td>
<td>23 (37)</td>
</tr>
<tr>
<td>Multivessel disease, %</td>
<td>26 (42)</td>
</tr>
<tr>
<td>Thrombus score</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>22 (37)</td>
</tr>
<tr>
<td>3</td>
<td>24 (39)</td>
</tr>
<tr>
<td>4</td>
<td>5 (7)</td>
</tr>
<tr>
<td>Thrombus aspiration</td>
<td></td>
</tr>
<tr>
<td>Yes, %</td>
<td>37 (60)</td>
</tr>
<tr>
<td>No, %</td>
<td>25 (40)</td>
</tr>
<tr>
<td>Adjunctive pharmacologic therapy</td>
<td></td>
</tr>
<tr>
<td>Glycoprotein IIb/IIa inhibitor, %</td>
<td>36 (58)</td>
</tr>
<tr>
<td>Bivalirudin, %</td>
<td>26 (42)</td>
</tr>
<tr>
<td>Stent type</td>
<td></td>
</tr>
<tr>
<td>Bare</td>
<td>42 (68)</td>
</tr>
<tr>
<td>Drug</td>
<td>20 (32)</td>
</tr>
<tr>
<td>ST-segment resolution, %</td>
<td>36 (57)</td>
</tr>
<tr>
<td>Post PCI TIMI flow grade, %</td>
<td>60 (97)</td>
</tr>
<tr>
<td>Post PCI MBG, %</td>
<td>44 (71)</td>
</tr>
</tbody>
</table>

**Notes:** LAD indicates left anterior descending; LCx, left circumflex; RCA, right coronary artery; PCI, percutaneous coronary intervention; TIMI, Thrombolysis in Myocardial Infarction; and MBG, myocardial blush grade.
Table 3. Outcomes of Left Ventricular Remodeling in Study Population, Mean±SD

<table>
<thead>
<tr>
<th></th>
<th>Scan 1–48 Hours</th>
<th>Scan 2–3 Weeks</th>
<th>Scan 3–6 Months</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEDVI, mL/mm²</td>
<td>69.8 ± 13.9</td>
<td>71.8 ± 13.4</td>
<td>78.1 ± 18.5</td>
</tr>
<tr>
<td>LVESVI, mL/mm²</td>
<td>38.0 ± 10.8</td>
<td>37.1 ± 9.7</td>
<td>38.9 ± 14.1</td>
</tr>
<tr>
<td>LVSVI, mL/mm²</td>
<td>31.8 ± 6.7</td>
<td>35.0 ± 6.6</td>
<td>39.1 ± 9.5</td>
</tr>
<tr>
<td>LVEF, %</td>
<td>45.6 ± 6.7</td>
<td>49.9 ± 7.2</td>
<td>50.7 ± 9.4</td>
</tr>
<tr>
<td>IS-SWT, %</td>
<td>−1.9 ± 21.6</td>
<td>38.7 ± 32.3</td>
<td>43.6 ± 28.1</td>
</tr>
<tr>
<td>Infarct size, % of myocardium</td>
<td>22.5 ± 14.2</td>
<td>13.2 ± 6.9</td>
<td>14.4 ± 6.3</td>
</tr>
<tr>
<td>MVO, % of myocardium</td>
<td>4.9 ± 1.3</td>
<td>0.4 ± 0.1</td>
<td>0.0 ± 0.0</td>
</tr>
</tbody>
</table>

LVEDVI indicates left ventricular end-diastolic volume index; LVESVI, left ventricular end-systolic volume index; LVSVI, left ventricular stroke volume index; LVEF, left ventricular ejection fraction; IS-SWT, infarct segment systolic wall thickening; and MVO, microvascular obstruction.

Discussion

In the present study, we prospectively characterized the evolution of myocardial edema by T2 quantification and myocardial hemorrhage by T2* quantification at both early and late time intervals post acute myocardial infarction. We observed that myocardial edema is not resolved at 3 weeks post acute myocardial infarction, suggesting that the subacute phase of myocardial injury is ongoing. Conversely, myocardial hemorrhage resolves sooner (within 3 weeks) post STEMI. MVO is present in all patients with hemorrhage, but hemorrhage does not have to present with MVO. In the acute phase, the T2 measurement is elevated in the noninfarcted segment as well and resolves by 3 weeks, which may represent transient edema of the remote myocardium. Patients with hemorrhage or MVO had a trend toward worse left ventricular remodeling at 6 months compared with patients without either of these patterns of microvascular injury.

Several preclinical and human studies have demonstrated that T2 signal hyperintensity by CMR suggests increased myocardial water content. The T2 signal may increase quickly, that is, within 30 minutes of ischemia onset, even before detectable injury by troponin or late-gadolinium enhancement. Furthermore, the presence of myocardial edema after an acute coronary syndrome has been recently shown to be associated with a higher hazard of cardiovascular event or death within 6 months. Hence, characterizing the evolution of myocardial edema has important prognostic value, and perhaps this parameter of myocardial injury can be used to evaluate the efficacy of future therapies with the advantage of avoiding contrast administration.

Figure 1. Evolution of T2 measurement over time. T2 is higher in infarct segment (IS) compared with remote segment (RS) at 48 hours and 3 weeks. By 6 months, the T2 measurement has equalized.

Figure 2. Evolution of end-diastolic wall thickness (EDWT) measurement over time. EDWT is increased in infarct segment (IS) compared with remote segment (RS) at day 2 and decreases over the next 2 time points. The RS EDWT measurement increases over time.
Previous studies have used T2-weighted imaging with dark blood turbo spin-echo technique. Although frequently used, these techniques have limitations, which impairs their validity. Some of these limitations include the following: (1) surface coil intensity inhomogeneity leading to variability in myocardial signal, (2) subendocardial bright signal artifact caused by stagnant blood, (3) cardiac motion leading to reduced myocardial signal, and (4) subjective nature of qualitative T2-weighted imaging assessment which then poses significant challenges in tracking longitudinal changes in a robust manner. We used a quantitative technique to measure edema and recently T2 mapping has been shown by Verhaert et al to be a robust alternative to conventional T2-weighted imaging to detect myocardial edema in these patients. In their study, recent ischemic injury was quantitatively differentiated from remote myocardium by its higher T2 value in 96% of patients enrolled in the study, compared with 67% by T2-weighted imaging, highlighting the superiority of quantitative T2 mapping.

Our study offers the additional benefit of having longitudinal quantitative follow-up of T2 in both the IS and RS. Myocardial edema appears to be present in the IS even up to 3 weeks post STEMI and resolves by 6 months. Interestingly, the RS has higher T2 values acutely as well compared with the 3 weeks time point. This may be due to a global hemodynamic response at the time of myocardial injury and may even serve as a marker of extensive myocardial damage. Edema has been previously discussed as a generic component of the tissue response to acute injury and what we see in the RS in our study may indeed reflect that. This is not well described in the previous literature, perhaps because even histological techniques have previously failed to provide reliable qualitative or quantitative data on the presence and regional distribution of edema. However, due to the ability of water-sensitive CMR, visualization of myocardial edema in vivo is now possible.

We used an additional quantitative measure of edema, that is, end-diastolic wall thickness. The higher measurement in the IS at 48 hours may be attributable to myocardial edema and tissue swelling as demonstrated by the moderate degree of correlation between the IS T2 measurement and degree of end-diastolic wall thickness in the IS at this time point (Figure 3). The subsequent decline in the IS end-diastolic wall thickness is likely secondary to edema resolution and scar formation. The end-diastolic wall thickness increases over time in the RS, likely due to localized compensatory hypertrophy as a consequence of left ventricular remodeling.

Myocardial hemorrhage is considered to be a sign of severe microvascular injury characterized by vascular cell damage, with leakage of red blood cells from injured cells affecting mainly the midmyocardial layer. It is a common complication after successful myocardial reperfusion, affecting 25% of patients, and is an independent predictor of adverse left ventricular remodeling regardless of the initial infarct size, and a marker of late arrhythmic risk. As a result, documenting the presence and evolution of myocardial hemorrhage may have
similar prognostic implications and therapeutic applications like myocardial edema. Our findings show that myocardial hemorrhage has a much more rapid time course of complete resolution (<3 weeks) compared with edema, the cause of which is also unclear. Furthermore, 46% of patients with MVO in our study did not have significant hemorrhage suggesting that there may be other causes of MVO beyond hemorrhage.

We also observed that patients without hemorrhage showed a decline in their T2 signal between the acute and subacute phase, a finding one would expect with infarct healing and edema resolution. However, patients with hemorrhage had no change in their T2 signal over the course of 3 weeks. Two explanations may be contributing to this unexpected finding. Perhaps patients with hemorrhage have more extensive myocardial injury and edema, which persists for a longer period of time (>3 weeks). An alternative, but not mutually exclusive reason for our finding is that in the presence of myocardial hemorrhage, edema quantification by T2 mapping is hindered due to susceptibility effects and therefore we may be underestimating the T2 signal at the initial time point in these patients. This was also observed by Verhaert et al in a subset of their patients, where the core of the infarct was found to have a significantly lower T2 value compared with the periphery of the infarcted tissue. This was postulated by the authors to be possibly secondary to myocardial hemorrhage given that these areas corresponded to MVO and there is a known relationship between myocardial hemorrhage and MVO. Our study results are consistent with the authors’ hypothesis and provide direct proof that T2 shortening due to hemoglobin breakdown impairs the ability of quantitative T2 techniques to detect myocardial edema in the acute phase.

Six-month data demonstrated a trend toward worse left ventricular remodeling in patients with hemorrhage or MVO. Certain parameters of left ventricular remodeling did not reach statistical significance potentially due to insufficient power to detect these differences.

**Study Limitations**

The present study represents a single-center experience with a limited number of patients. However, the sample size in our study is similar to previously published CMR studies that have investigated myocardial edema, hemorrhage, and MVO in other clinical settings. The median peak creatine kinase rise in our study group was ≈1800 IU/L as the majority of our patients had large infarcts, which may limit the ability to generalize our results to patients with smaller infarcts. A systematic intra- and interobserver analysis was not performed for T2 and T2* measurements. However, signal fluctuations in these values generated by dynamic processes like edema and hemorrhage are far greater than those arising from measurement variability. Last, we used a surface coil which can decrease the sensitivity for detecting inferolateral edema and infarction with an opposite effect in the anteroseptal region, due to an inhomogeneous coil sensitivity profile.

**Conclusions**

The characterization of myocardial edema and hemorrhage by T2 and T2* quantification is feasible post acute myocardial infarction. The evolution of these myocardial injury parameters post STEMI is complex, with hemorrhage resolving faster than edema. Noninfarcted segments can demonstrate edema in the acute phase as well, perhaps serving as a surrogate marker of severity of myocardial injury. Myocardial hemorrhage can make edema quantification challenging in the acute phase due to susceptibility effects. Last, the presence of hemorrhage or MVO in patients with STEMI leads to potentially worse left ventricular remodeling.

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Disclosures

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CLINICAL PERSPECTIVE

Cardiovascular magnetic resonance imaging has gained clinical importance in the noninvasive assessment of myocardial injury parameters including myocardial edema, hemorrhage, microvascular obstruction, and infarct size post acute myocardial infarction. We prospectively characterized the evolution of these parameters post reperfused acute myocardial infarction at both early and late time points. We noted that edema is still present in intracted tissue at 3 weeks, whereas hemorrhage resolves faster. Noninfarcted segments can demonstrate edema in the acute phase as well, perhaps indicative of more severe myocardial injury. The presence of hemorrhage in the acute phase makes edema quantification challenging due to susceptibility effects. In addition, both hemorrhage and microvascular obstruction are associated with worse left ventricular remodelling. Gaining this knowledge about the temporal resolution of myocardial damage and its impact on remodeling processes using quantitative techniques is potentially important in grading severity, evaluating treatment strategies, and improving clinical outcomes.
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