Detecting Acute Coronary Syndrome in the Emergency Department With Cardiac Magnetic Resonance Imaging

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Background—Managing chest pain in the emergency department remains a challenge with current diagnostic strategies. We hypothesized that cardiac MRI could accurately identify patients with possible or probable acute coronary syndrome.

Methods and Results—The diagnostic performance of MRI was evaluated in a prospective study of 161 consecutive patients. Enrollment required 30 minutes of chest pain compatible with myocardial ischemia but an ECG not diagnostic of acute myocardial infarction. MRI was performed at rest within 12 hours of presentation and included perfusion, left ventricular function, and gadolinium-enhanced myocardial infarction detection. MRI was interpreted qualitatively but also analyzed quantitatively. The sensitivity and specificity, respectively, for detecting acute coronary syndrome were 84% and 85% by MRI, 80% and 61% by an abnormal ECG, 16% and 95% for strict ECG criteria for ischemia (ST depression or T-wave inversion), 40% and 97% for peak troponin-I, and 48% and 85% for a TIMI risk score ≥3. The MRI was more sensitive than strict ECG criteria for ischemia (P<0.001), peak troponin-I (P<0.001), and the TIMI risk score (P=0.004), and MRI was more specific than an abnormal ECG (P<0.001). Multivariate logistic regression analysis showed MRI was the strongest predictor of acute coronary syndrome and added diagnostic value over clinical parameters (P<0.001).

Conclusions—Resting cardiac MRI exhibited diagnostic operating characteristics suitable for triage of patients with chest pain in the emergency department. Performed urgently to evaluate chest pain, MRI accurately detected a high fraction of patients with acute coronary syndrome, including patients with enzyme-negative unstable angina. (Circulation. 2003;107:531-537.)

Key Words: emergency department ■ magnetic resonance imaging ■ myocardial infarction ■ ischemia ■ troponin

Public health initiatives appropriately encourage patients with 20 to 30 minutes of chest pain to seek immediate medical evaluation. Annually, more than 5 million patients visit emergency departments (ED) with chest pain, which leads to important diagnostic issues for ED physicians.

Acute coronary syndrome (ACS) spans a broad classification, including ST-elevation myocardial infarction (STEMI), non–ST-elevation myocardial infarction (NSTEMI), and unstable angina. On the basis of ECG and initial troponin levels, only a small percentage of patients with chest pain are clearly diagnosed on presentation as having ACS. Although cardiac enzymes are extremely sensitive to myocardial infarction (MI), by definition, these assays do not detect unstable angina. Furthermore, it may take 4 to 12 hours after presentation before cardiac enzymes are definitive. It is well recognized that the ECG can be completely normal in acute MI and, therefore, also in unstable angina.

Missing the diagnosis of ACS doubles risk-adjusted mortality. Recent estimates indicate ≈2% of patients with MI are inappropriately discharged home from the ED. Consistent with the vulnerable plaque hypothesis, the consequences of not detecting unstable angina could result in progression to MI after discharge from the ED. Although troponin-negative patients seem to be at low risk, many patients with unstable angina require urgent medical therapy or intervention.

Advanced imaging that can detect unstable angina in addition to MI could improve triage of patients. We hypothesized that cardiac MRI at rest can effectively assess possible or probable ACS with a combined examination of regional contractile function, perfusion, and viability.

Methods

Patients presenting to a community hospital ED with chest pain were prospectively evaluated after giving informed consent. MRI scans were performed as early as feasible after resolution of chest pain and stabilization in the ED. Inclusion criteria were ≥30 minutes of chest pain compatible with myocardial ischemia (chest pain score ≥4) within 12 hours before ED presentation, >21 years of age, and weight <270 pounds. Medical exclusions included STEMI, preg-

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nancy, and severe congestive heart failure (unable to lie flat). MRI contraindications included pacemakers, defibrillators, cerebral aneurysm clips, metal in eye, and implanted devices such as insulin pumps, ear implants, or neural stimulators. We did not exclude patients with stents, artificial valves, anuloplastic rings, sternal wires, atrial fibrillation, or other cardiac history.

**Definition of ACS and Ischemic Heart Disease**

ACS was prospectively defined to satisfy guidelines established by the American College of Cardiology and the American Heart Association (ACC/AHA)\(^1\) with the following modifications. Possible or probable ACS required resting chest pain compatible with myocardial ischemia of ≥30 minutes duration within 12 hours of ED presentation (an entry criterion that excludes chronic stable angina). NSTEMI ACS required abnormal serial troponin-I (≥1.96 μg/dL) with a temporal pattern consistent with acute MI and any clinical evidence of coronary artery disease. Confirmation of unstable angina required a 70% epicardial coronary stenosis or true positive abnormal stress test performed during the index hospitalization or subsequent 6- to 8-week follow-up period. For this study, ACS combines NSTEMI and unstable angina unless specifically noted.

Ischemic heart disease (IHD) was defined as any patient with ACS or prior MI. MRI was not used to define any end points. In the detection of ACS, an MRI was considered a true positive only if the findings were in the territory of a significant stenosis.

**Imaging Protocol**

Imaging was performed on a 1.5T scanner (CV/i, General Electric) using a cardiac phased-array receiver coil, raw image sequences, and noninvasive monitoring (Medrad). Shim and center frequency were determined using stimulated echo acquisition mode.\(^9\) The diagnostic scan began with resting first-pass perfusion imaging using 0.1 mmol/kg intravenous gadopentetate dimeglumine (Berlex Laboratories), a selective saturation pulse,\(^9\) and a segmented echoplanar gradient echo readout\(^10\) to image 7 to 9 locations every 2 heartbeats (acquisition window, 123 to 163 ms/image; spatial resolution, 3.0 to 3.3 mm in-plane). Left ventricular function was imaged with fast-gradient recalled echo\(^11\) or fast imaging with steady-state precession\(^12\) at a temporal resolution of 40 to 50 ms and a spatial resolution of 1.8 to 2.0 mm in-plane. To detect MI, an inversion-recovery method\(^13\)\(^\text{14}\) was performed ~20 minutes after a cumulative dose of 0.2 mmol/kg gadopentetate dimeglumine.

**Qualitative MRI Analysis**

Studies were read by the consensus of 3 cardiologists who were blinded to the study end point. An abnormal MRI required a regional wall motion abnormality\(^15\) or abnormal delayed gadolinium hyperenhancement. Abnormal delayed gadolinium hyperenhancement was defined as myocardium exhibiting higher signal intensity than the surrounding myocardium.\(^16\)

Abnormal perfusion was defined as an area that did not enhance as bright as surrounding myocardium on several consecutive images coinciding with early peak myocardial enhancement. To accept a perfusion defect as a definite abnormality, there had to be a regional wall motion abnormality or delayed hyperenhancement in a matching location.

**Quantitative MRI Analysis**

Regional left ventricular wall thickness was measured at end-systole and end-diastole using computer-assisted calipers in the anterior septum, lateral wall, inferior septum, and any suspected regional wall motion abnormality. Indices of regional left ventricular function were calculated (absolute wall thickening in millimeters, percent change of wall thickness from end-diastole, and ratio of systolic to diastolic wall thickness).

The signal intensities of gadolinium-enhanced myocardium and normal myocardium were measured with computer-assisted planimetry. The contrast difference index was the difference in signal intensity between 2 regions divided by the standard deviation of the signal intensity in the normal region.\(^16\) The signal intensity ratio between the 2 regions was also calculated (contrast ratio).

**Conventional Investigation**

Serum troponin-I was typically collected on presentation and 4 and 8 hours later. The most abnormal ECG before or immediately after the MRI scan was selected for interpretation. An abnormal ECG was defined by the presence of T-wave inversion, ST-depression, bundle-branch block, left ventricular hypertrophy, non-specific ST-T changes, or pathological Q waves. Strict ECG criteria for ischemia required an ST depression ≥1 mm or a T-wave inversion ≥3 mm. Attending physicians made decisions to perform coronary angiography on clinical grounds independent of the MRI team. Conventional tests were read-blinded to final MRI readings. MRI findings were not used to determine the validity of conventional tests.

**Statistical Methods**

Logistic regression was performed to assess whether an abnormal MRI added diagnostic information over clinical parameters used to predict ACS. Rather than considering individual cardiac risk factors, the total number of cardiac risk factors (TlCRF) was defined as the number of the following that were present: advanced age (>45 for men, >55 for women), hypercholesterolemia, hypertension, diabetes mellitus, family history of coronary artery disease, tobacco use, and history of previous MI. Thus, the TlCRF ranged from 0 to 7. The other variables entered into the logistic models were an abnormal ECG (AbECG; 1 if abnormal, 0 if normal) and initial troponin-I (AbTn; 1 if abnormal, 0 if not). For the ACS end point, a model was formed with and without abnormal MRI (AbMRI; 1 if abnormal, 0 if normal). Let p be the probability of ACS. The following logistic regression models were considered.

\[
\ln[p/(1-p)] = \text{Intercept} + A(\text{TlCRF}) + B(\text{AbECG}) + C(\text{AbTn})
\]

and

\[
\ln[p/(1-p)] = \text{Intercept} + A'(\text{TlCRF}) + B'(\text{AbECG}) + C'(\text{AbTn}) + D'(\text{AbMRI})
\]

The intercept and the coefficients represent the relative weighting of each variable and were estimated from the data using the logistic regression procedure of SAS (Version 6.12). Goodness of fit was assessed via the log likelihood. When the difference between “−2 log likelihood” for the model (which has a \( \chi^2 \) distribution with one degree of freedom) is statistically significant, AbMRI adds significantly to the diagnostic information over the clinical parameters alone. The analysis used SAS version 6.12 (Statistical Analysis System, SAS Institute Inc.).

Receiver-operating characteristic analysis was performed for each measurement to predict ACS. The sensitivity and specificity of an abnormal ECG, strict ECG criteria for ischemia, troponin, and the TIMI risk score were compared with MRI using a z-test that considered the correlation of diagnostic tests performed on the same subjects.

**Results**

The demographic characteristics of the 161 patients in the study were stratified by the ACS end point and summarized in Table 1. Of 193 consecutive patients who fulfilled inclusion criteria, 11 declined to participate and 21 were excluded for the following reasons: failure to stabilize in the ED \( (n=6) \), metallic implants \( (n=2) \), body size \( (n=3) \), and claustrophobia \( (n=10) \). One excluded patient was troponin-I-positive.

The MRI scan lasted 38±12 minutes and patients were away from the ED for 58±10 minutes. A total of 137 patients (85%) had MRI before availability of the second troponin results. All MRIs were performed within 12 hours of presen-
Diagnosis of ACS by Conventional Tests and Follow-Up

Follow-up was undertaken at 6 to 8 weeks to assure that no ACS was missed during the index hospitalization. Complete follow-up was achieved in 98% of patients. Troponin-I was available in 160 patients. The 3 patients with incomplete follow-up were all troponin-I–negative. A total of 22 of 25 patients had ACS diagnosed during the index hospitalization. Three patients were characterized as having ACS on the basis of the 6- to 8-week follow-up. One of these 3 patients returned with a STEMI, one returned 2 weeks later with unstable angina, and another had significant left anterior descending coronary artery disease on angiography after hospitalization.

Of the 161 patients studied, 16% had ACS \((n=25)\), including 10 with NSTEMI (troponin-I, 2.96 to 44.1 ng/mL) and 15 with unstable angina. Several clinical parameters were significantly different between ACS-positive and -negative patients (Table 1), including the TIMI risk score.\(^\text{17}\) Of the 44 patients who underwent coronary angiography, 25 met the end point of ACS and 19 did not. Ten of the 15 patients with unstable angina had severe or \(\geq 90\%\) coronary stenosis (Figure 1), and the remaining had 70% to 90% stenosis. Fourteen of the 25 patients with ACS underwent coronary revascularization, including 7 with unstable angina. The ECG met strict criteria for ischemia in only 2 patients with NSTEMI and 2 patients with unstable angina. There were 4 false-positive troponins: 1 was attributed to cardiac sarcoidosis and 3 patients had atypical temporal patterns of cardiac enzymes and no significant abnormalities on coronary angiography.

**Table 1. Demographic Summary**

<table>
<thead>
<tr>
<th></th>
<th>ACS ((n=25))</th>
<th>No ACS ((n=136))</th>
<th>(P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>68±13</td>
<td>57±14</td>
<td>0.0006</td>
</tr>
<tr>
<td>Men, %</td>
<td>60</td>
<td>57</td>
<td>NS</td>
</tr>
<tr>
<td>CAD risk factors</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Advanced age (men (&gt;45); women (&gt;55)), %</td>
<td>92</td>
<td>68</td>
<td>0.03</td>
</tr>
<tr>
<td>Hypertension, %</td>
<td>56</td>
<td>43</td>
<td>NS</td>
</tr>
<tr>
<td>Diabetes, %</td>
<td>28</td>
<td>10</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Hypercholesterolemia, %</td>
<td>64</td>
<td>47</td>
<td>NS</td>
</tr>
<tr>
<td>CAD in family, %</td>
<td>32</td>
<td>42</td>
<td>NS</td>
</tr>
<tr>
<td>History of smoking, %</td>
<td>48</td>
<td>39</td>
<td>NS</td>
</tr>
<tr>
<td>Total No. of CAD risk factors</td>
<td>4.2</td>
<td>3.2</td>
<td>0.003</td>
</tr>
<tr>
<td>Previous angina, %</td>
<td>40</td>
<td>12</td>
<td>0.001</td>
</tr>
<tr>
<td>Previous MI, %</td>
<td>28</td>
<td>13</td>
<td>NS</td>
</tr>
<tr>
<td>Previous PCI or CABG, %</td>
<td>44</td>
<td>16</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Location, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Substernal</td>
<td>32</td>
<td>31</td>
<td>NS</td>
</tr>
<tr>
<td>Precordial</td>
<td>24</td>
<td>18</td>
<td>NS</td>
</tr>
<tr>
<td>Quality, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Crushing, heaviness, pressure, or tightness</td>
<td>56</td>
<td>54</td>
<td>NS</td>
</tr>
<tr>
<td>Radiation to neck/arm</td>
<td>32</td>
<td>31</td>
<td>NS</td>
</tr>
<tr>
<td>Dyspnea, diaphoresis, or nausea</td>
<td>32</td>
<td>40</td>
<td>NS</td>
</tr>
<tr>
<td>Average chest pain score</td>
<td>8.4</td>
<td>7.5</td>
<td>NS (0.06)</td>
</tr>
<tr>
<td>BP on admission, mm Hg</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>136</td>
<td>141</td>
<td>NS</td>
</tr>
<tr>
<td>Diastolic</td>
<td>77</td>
<td>75</td>
<td>NS</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>69</td>
<td>70</td>
<td>NS</td>
</tr>
<tr>
<td>Cardiac medications, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antianginals or antihypertensives</td>
<td>52</td>
<td>34</td>
<td>NS</td>
</tr>
<tr>
<td>Aspirin</td>
<td>36</td>
<td>10</td>
<td>0.002</td>
</tr>
<tr>
<td>Digoxin</td>
<td>4</td>
<td>1</td>
<td>NS</td>
</tr>
<tr>
<td>Antilipid agent</td>
<td>28</td>
<td>12</td>
<td>NS (0.07)</td>
</tr>
<tr>
<td>TIMI risk score</td>
<td>2.5±1.2</td>
<td>1.6±0.8</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

Values are mean±SD or as indicated. CAD indicates coronary artery disease; PCI, percutaneous coronary intervention; CABG, coronary artery bypass grafting; BP, blood pressure; and NS, not significant.

**Figure 1.** Example of a troponin-I–negative patient with ACS secondary to a 95% stenosis of the left anterior descending coronary artery. The anteroseptum was akinetic and thus appears much thinner at end systole (arrows) than the other segments (left). There is a dark band indicating reduced subendocardial perfusion (middle) but no delayed hyperenhancement (right) in the same area, suggesting this is ischemic but not infarcted myocardium.

**Figure 2.** Sensitivity and specificity of various parameters for ACS. Probability values summarize the z-test, which was 2-tailed and adjusted for tests performed on same subjects. Sensitivity is shown by dark bars and specificity by white bars. ECG indicates abnormal ECG; ECG strict, strict electrocardiographic evidence of ischemia defined as ST depression >1 mm or T-wave inversion >3 mm; Trop, initial troponin-I; Trop peak, peak troponin-I; TIMI, TIMI risk score; and NS, not significant.
An abnormal ECG had a sensitivity of 80% (P=NS versus MRI) but a lower specificity of 61% (P<0.001 versus MRI). The sensitivity of strict ECG criteria for ischemia decreased to 16% (P<0.001 versus MRI), although the specificity (95%) was better than MRI (P=0.011). Troponin-I, either peak or initial, was not as sensitive to ACS as MRI (both P<0.001), but an abnormal troponin was more specific (P<0.001 versus MRI). A TIMI risk score ≥3 had 48% sensitivity and 85% specificity for ACS (P<0.001 and P=NS versus MRI, respectively). The attending physicians’ decision to admit a patient had a sensitivity of 88% and specificity of 55% for ACS.

Additional analysis was performed to understand better the diagnostic characteristics of MRI. For example, MRI detected all 10 NSTEMI, including the 3 patients with normal ECGs. However, because it was difficult to differentiate acute and chronic MI, it is important to analyze those patients with IHD, which encompasses both diagnoses. Thus, from the perspective of diagnosing ACS, all but 2 false-positive MRI scans can be explained by a prior MI.

**Quantitative MRI Analysis**

Quantitative MRI analysis is summarized in Table 2. Absolute wall thickening provided the best overall accuracy in detecting ACS (82%), NSTEMI (89%), and IHD (98%; Figure 3). A threshold of 3 mm of wall thickening detected 83% of ACS, all NSTEMI, and 91% of IHD.

Using a threshold of 5.0, the contrast difference index discriminated infarcted myocardium from normal myocardium and agreed in all cases with the qualitative reading. Compared with normal myocardium, the signal intensity of hyperenhanced myocardium averaged 14 standard deviations brighter (P<0.001). On the basis of the contrast ratio, infarcted myocardium was 4.1±0.8 times brighter than normal myocardium (P<0.001). Patients with unstable angina but no history of MI decreased the sensitivity of delayed gadolinium hyperenhancement images for ACS and IHD to 73% and 63%, respectively. For diagnosing ACS, the specificity of the contrast enhancement ratio was 91%.

**Multivariate Logistic Regression Analysis**

Abnormal MRI was the most significant test of those considered. In addition, abnormal MRI added significant independent diagnostic information to the other tests (Table 3, P<0.001 for model b compared with a).

The models had good overall fit, as demonstrated by their likelihood ratios (Table 3). For ACS, in the first model, total risk factors (TICRF), ECG, and initial troponin-I were highly significant. When MRI was added to that model, total risk factors and ECG were no longer significant, whereas MRI was significant (P<0.0001). There was a high correlation between total risk factors (TICRF), abnormal MRI, and ECG, which explained the lack of significance of these variables once MRI was added to the model. For prediction of ACS, the receiver-operating characteristic curves for the TIMI risk score and the current logistic regression models (with and without MRI) are displayed in Figure 4.

**Discussion**

This is the first study to demonstrate the feasibility of detecting ACS by MRI in the ED. The goals of chest pain imaging in the era of highly sensitive serum markers of myocyte necrosis are different than in prior years. Chest pain imaging needs added clinical value over infarct detection and should aim to confirm ACS rapidly. Qualitative interpretation of a resting MRI had 100% sensitivity for NSTEMI and 84% sensitivity for ACS (despite resolution of chest pain) and was performed with high specificity (85%). Quantitative analysis also achieved high diagnostic accuracy. Receiver-operator curve and multivariate logistic regression analyses showed that MRI was the strongest predictor of ACS and had independent diagnostic value over clinical parameters, including ECG, initial troponin-I, and the TIMI risk score.
Cardiac enzymes only detect the NSTEMI fraction of ACS. Perfusion may be normal between episodes of chest pain. However, regional wall motion may remain abnormal for hours after transient ischemia, a phenomenon known as myocardial stunning.18 With this background, it is not surprising that wall motion was the most powerful element of the MRI scan.

Radionuclide imaging for chest pain is the most developed modality for ED triage, with reports since 1979. 19 The results for infarct detection have been excellent, particularly for the Tc-99m–based tracers.19–25 However, there is room for improvement. In a cohort of 357 patients imaged acutely as part of an ED triage protocol, Heller et al24 reported normal images at rest in 12 of 35 patients (34%) who ultimately were diagnosed with ACS, including 2 with acute MI. Normal images were acquired in 7 of 32 patients with ACS (22%) in a cohort of 1000 patients with chest pain.22 In a multicenter trial of 102 ED patients with typical angina but nondiagnostic ECG, only 3 of 15 patients with unstable angina had abnormal sestamibi studies.21

Despite the relatively long period that acute imaging with radionuclides has been available, the technique has not been embraced on a large scale. Recently, an expert panel outlined the progress and difficulties with acute radionuclide imaging.26 These include isotope preparation, decay and licensing issues, a preponderance of small nuclear medicine laboratories with insufficient support to provide imaging 24 hours per day, and the difficulties associated with single-image interpretation without the customary second image for comparison. Other limitations include low spatial resolution and limited temporal resolution.

Echocardiography has had less impact in chest pain evaluation because of its limited sensitivity to acute MI, especially when the pain has resolved.27,28 Left ventricular systolic dysfunction detected by echocardiography has prognostic significance.29 Wall motion abnormalities may be subtle with subendocardial ischemia and difficult to visualize, with limited echocardiographic windows. In our experience, perfusion and delayed gadolinium hyperenhancement helped detect several cases with subtle wall motion abnormalities. However, reasonable judgment must be exercised to prevent overdiagnosis based on borderline abnormalities or the test specificity will degrade. Electron beam computer tomography had excellent negative predictive value but low specificity in patients with chest pain.30,31

MRI surmounts some of these barriers. Many hospitals have MRI capability for other emergencies such as stroke. Isotopes are not needed. Gadolinium is commercially available and well tolerated. Resolution, both spatially and temporally, is excellent. The multimodality characteristics of MRI provide 3 diagnostic opportunities. Wall motion can detect ischemia, postischemic stunning, and infarction. The

<table>
<thead>
<tr>
<th>Variable</th>
<th>Coefficient Estimate</th>
<th>Wald $\chi^2$</th>
<th>P</th>
<th>Odds Ratio</th>
</tr>
</thead>
</table>
| Model a for Probability of ACS Without MRI (25 events)*
| Intercept | -4.1026 | 30.18 | 0.0001 |
| TICRF | 0.3718 | 4.57 | 0.0325 | 1.5 |
| AbECG | 1.7606 | 9.70 | 0.0018 | 5.8 |
| AbTn | 2.2689 | 7.67 | 0.0056 | 9.7 |
| Model b for Probability of ACS With MRI (25 events)†
| Intercept | -3.8704 | 22.27 | 0.0001 |
| TICRF | -0.0196 | 0.0093 | 0.92 | 1.0 |
| AbECG | 0.9136 | 1.99 | 0.16 | 2.5 |
| AbTn | 2.0402 | 4.14 | 0.042 | 7.7 |
| AbMRI | 3.0754 | 19.12 | 0.0001 | 21.7 |

TICRF indicates total cardiac risk factors; AbECG, abnormal ECG; AbTn, abnormal initial troponin; AbMRI, abnormal MRI (see Methods for defining criteria for each variable); and DF, degrees of freedom.

*–2 Log Likelihood = 28.00 with 3 DF (P=0.0001).
†–2 Log Likelihood = 53.36 with 4 DF (p=0.0001).
gadolinium-enhanced methods help detect and characterize defects.

This study and others have validated the diagnostic utility of cardiac MRI for regional wall motion abnormality, perfusion, and viability. The MRI can be quantified with high diagnostic accuracy (Table 2), but the images are also suitable for rapid qualitative interpretations (Table 3). Logistically, the MRI protocol was feasible in the ED and did not interfere with patient management.

Certain considerations will need to be addressed before MRI will have widespread application in the ED. Many hospitals possess instrumentation that could perform these studies but may require expensive software or hardware upgrades to achieve similar performance. Approximately 10% of patients were excluded, most commonly for claustrophobia. Newer magnet designs may reduce claustrophobia. Cost-effectiveness will need to be analyzed at a suitable point in the future.

Limitations

The foremost limitation for this study is the inability to differentiate acute from chronic infarction, a problem common to all noninvasive methods studied to date. Because the MRI detects both acute and chronic MI, the accuracy of MRI is higher for IHD than ACS (Figure 3). ACS represents a diverse pathophysiology: acute NSTEMI, unstable angina, spontaneously reperfused acute MI, 2-week-old MI, extension of prior MI, and acute MI in one distribution but chronic MI in another distribution. We have not found criteria to differentiate acute from chronic MI in all cases. Nevertheless, either type of MI identifies a higher risk patient. Future advances may improve differentiation of acute and chronic MI. Despite these limitations, current MRI techniques had high sensitivity and specificity for ACS.

The definition of NSTEMI and unstable angina deviate somewhat from the ACC/AHA guidelines. Because the research protocol did not control decisions to perform coronary angiography, there is some potential for biasing the detection of disease. It is not clear if this would favor or go against the MRI. The skewed distribution favoring 90% to 99% stenosis rather than less severe stenosis suggests the patients identified as ACS were not incidental findings. Although our definition may have missed a few patients with ACS caused by less severe stenoses, it should be noted that 8% of our ACS group would have been categorized as having no significant coronary artery disease in the study by Roe et al (diagonal disease, Table 2). Limitations aside, chest pain evaluation with MRI seems promising because it can detect unstable angina and MI.

Acknowledgments

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