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IMAGING VIGNETTE

# Imaging Ischemic and Reperfusion Injury in Acute Myocardial Infarction

## Putting the Pieces Together With CMR

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**ACUTE MYOCARDIAL INFARCTION IS A COMPLEX CLINICAL CONDITION THAT AFFECTS THE MYOCARDIUM** in multiple ways. Cardiac magnetic resonance provides a range of noninvasive sequences that can optimally characterize jeopardized myocardium in acute coronary syndrome patients through both visual and quantitative (parametric) techniques, including nonenhanced ($T_2$-weighted imaging, native $T_1$-mapping, $T_2$ and $T_2$* mapping) and contrast-enhanced imaging (early and late gadolinium-enhanced imaging, post-gadolinium T1-mapping and calculation of extracellular volume) (Figures 1 to 4). These techniques identify myocardial edema and infarction, presence of microvascular obstruction and intramyocardial hemorrhage as well quantify myocardial and ventricular performance (Figures 1 to 4, Table 1). Correct use and interpretation of cardiac magnetic resonance images can provide a wealth of information for diagnosis and prognostication on in acute myocardial infarction (1,2). It should be noted relaxation times are dependent on the field strength and to some extent as well on the acquisition protocol used. All cases discussed were performed on a 1.5-T magnet.

**TABLE 1 Characterization of the Different Components of the Jeopardized Myocardium Using Comprehensive CMR**

| | $T_1$ | ECV | $T_2$ | $T_2$* | $T_2$-Weighted | EGE | LGE | Remarks |
|---|---|---|---|---|---|---|---|---|
| Myocardial edema | ↑ | ↑ | ↑↑ | – | **Bright** | ↑† | ↑† | Edema typically in CA perfusion territory |
| Myocardial necrosis | ↑ | ↑↑ | – | – | – | ↑ | ↑↑ | Enhancement typically in CA perfusion territory, subendocardial pattern with variable transmural spread of enhancement |
| MVO | – | ‡ | – | – | – | **Dark** | Dark§ | MVO in center of enhancement area |
| IMH‖ | ↓ | ¶ | – | ↓↓ | **Dark** | – | – | IMH in center of myocardial edema |

The key findings for differentiation of the different components in the jeopardized myocardium are shown in **bold**. †Faint enhancement. ‡Presence of MVO may affect accuracy of ECV calculation. §Fill-in of MVO between EGE and LGE. ‖IMH is nearly always associated with MVO (not vice versa). ¶Presence of IMH may affect accuracy of ECV calculation.

CA = coronary artery; CMR = cardiac magnetic resonance; ECV = extracellular volume; EGE = early gadolinium-enhanced imaging; IMH = intramyocardial hemorrhage; LGE = late gadolinium-enhanced imaging; MVO = microvascular obstruction.

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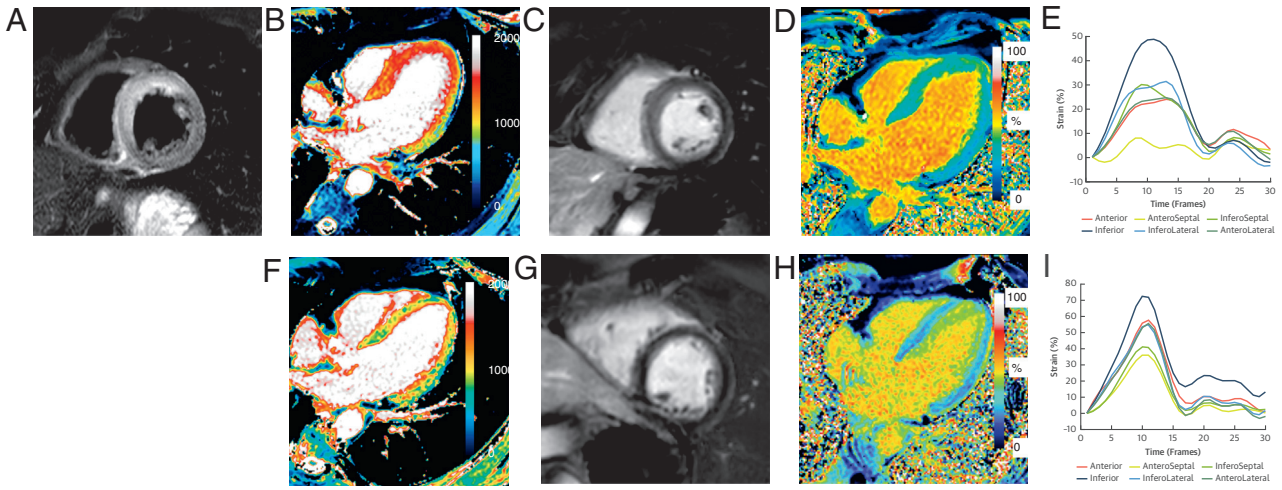
**FIGURE 1 Aborted STEMI**




Aborted ST-segment elevation myocardial infarction (STEMI) in a 42-year-old man with total left anterior descending (LAD) coronary artery occlusion (troponin T 0.5 μg/l). Cardiac magnetic resonance (CMR) (day 4 post percutaneous coronary intervention) shows diffuse myocardial edema and swelling ($T_2$-weighted imaging) **(A)** with increased $T_1$ values (1,202 ms vs. 954 ms remote myocardium) **(B)** and $T_2$ values (72 ms vs. 51 ms remote myocardium) in LAD territory. Following contrast administration, mild enhancement is shown in LAD territory (late gadolinium enhancement [LGE]) **(C)** with mild increase of extracellular volume (ECV) values (36% vs. 25% remote myocardium) **(D)**. CMR (4 months post MI) shows normalization of $T_1$ (988 ms) **(F)** and $T_2$ values (52 ms), resolution of LGE **(G)**, normalization of ECV values (28%) **(H)**. Normalization of end-diastolic wall thickness with improvement in radial strain (baseline CMR **[E]**, follow-up **[I]**) in LAD territory, and normalization of left ventricular ejection fraction (66% vs. 55% at baseline) (Online Videos 1 and 2 at baseline and follow-up, respectively). **Teaching point:** The presence of myocardial edema without typical infarct-like LGE pattern and only mild increases in ECV values, early post-PCI CMR is able to depict the reversible nature of ischemic injury.

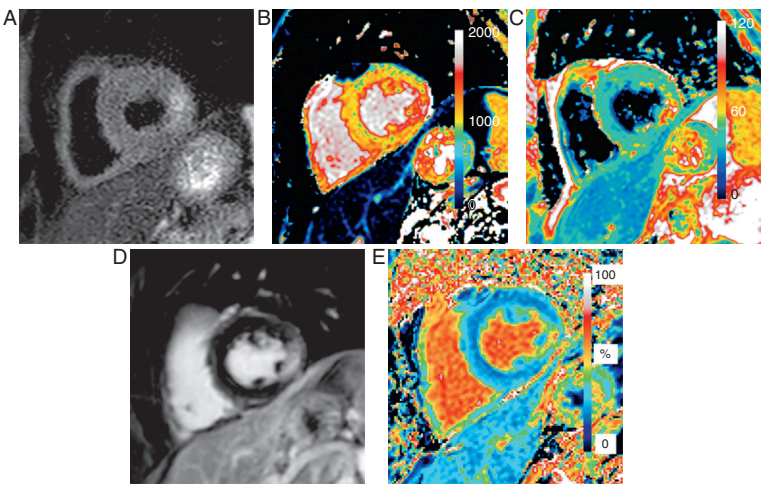



**FIGURE 2 Focal lateral MI**

Focal lateral MI in a 54-year-old woman presenting with STEMI but with nonobstructive coronary artery disease at cardiac catheterization. Mild increase in troponin T (1.02 μg/l). CMR at day 5 shows focal myocardial edema in mid-lateral left ventricular wall ($T_2$-weighted imaging) **(A)** with increased $T_1$ (1,253 ms vs. 1,021 ms remote myocardium) **(B)** and T2 values (88 ms vs. 50 ms remote myocardium) **(C)**. **(D)** LGE shows focal transmural enhancement with increased ECV values (62% vs. 26% remote myocardium) **(E)** and decreased contractility (radial strain 28% vs. 58% remote myocardium) in mid-lateral left ventricular wall, but no impact on left ventricular ejection fraction (71%). **Teaching point:** CMR is extremely helpful in acute coronary syndrome patients presenting non-obstructive coronary artery disease to locate and to characterize the jeopardized myocardium (1). Moreover, CMR allows for the differentiation of this condition from acute myocarditis because the patterns of enhancement differ; in other words, subepicardial and/or midwall pattern in myocarditis versus subendocardial pattern with variable transmural spread in acute MI. Abbreviations as in Figure 1.

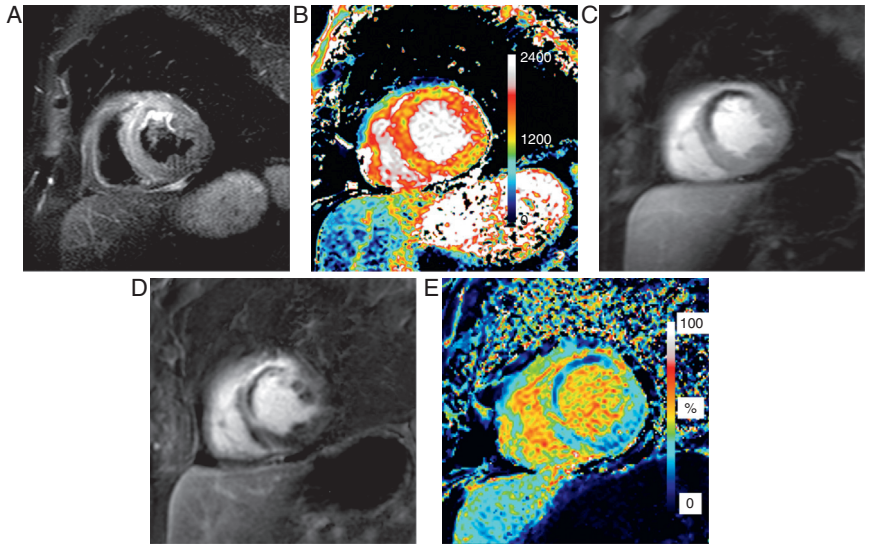
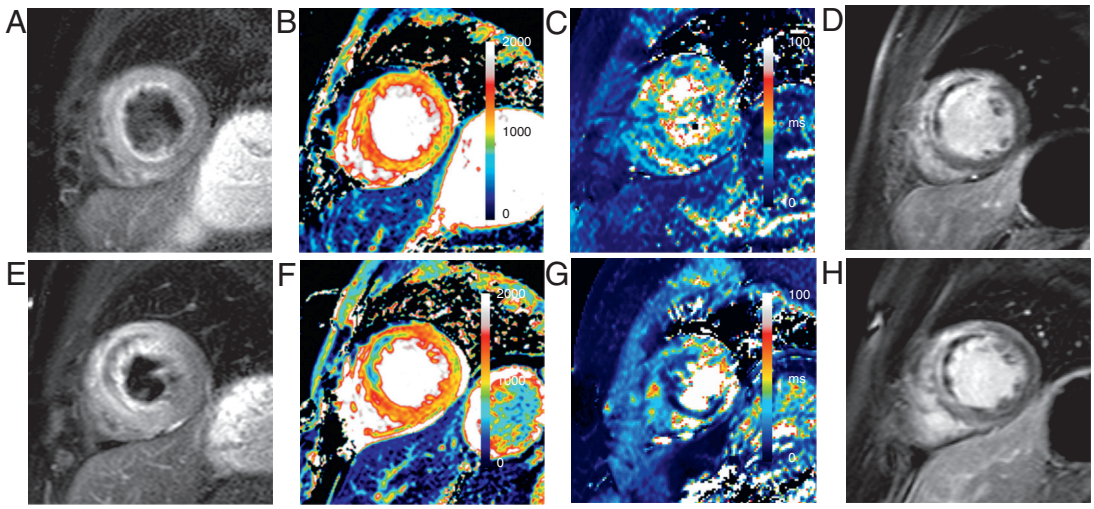



**FIGURE 3 MVO MI**

Microvascular obstruction (MVO) myocardial infarction (MI) in a 58-year-old man with total LAD coronary artery occlusion (troponin T 4.01 μg/l). CMR (day 3 post percutaneous coronary intervention) shows extensive myocardial edema ($T_2$-weighted imaging) **(A)** with increased $T_1$ (1,229 ms vs. 1,008 ms remote myocardium) **(B)** and $T_2$ values (68 ms vs. 49 ms) but normal $T_2$* values (32 ms vs. 29 ms remote myocardium) in LAD territory. Following contrast administration, a large area of MVO is visible at early gadolinium enhancement **(C)** with partial fill-in of MVO and transmural enhancement at LGE **(D)**. On the ECV map, MVO is visible as an area with very low ECV values **(E)**. Severe dysfunction in LAD territory (radial strain −10% vs. 22% remote myocardium) with moderate impact on left ventricular function (ejection fraction 39%) (Online Video 3). **Teaching point:** MVO reflects more severe myocardial damage and bears prognostic value in acute MI (2). Early gadolinium-enhanced imaging using a long inversion time (e.g., 500 ms) is the best approach to appreciate presence and extent of MVO. Abbreviations as in **Figure 1**.

**FIGURE 4 Post-Infarction IMH**




Post-infarction intramyocardial hemorrhage (IMH) in a 28-year-old man with total LAD coronary artery occlusion (troponin T 7.0 μg/l). CMR (day 1 post percutaneous coronary intervention) shows extensive myocardial edema ($T_2$-weighted images) **(A)** with increased $T_1$ values (1,157 ms vs. 988 ms remote myocardium) **(B)**, but normal $T_2$* values (28 ms) **(C)** in anteroseptal wall. Presence of transmural LGE with MVO **(D)**. Severe dysfunction in LAD territory with impaired left ventricular (LV) ejection fraction (32%) (Online Video 4). CMR (day 5 post percutaneous coronary intervention), shows appearance of a central hypo-intense ("dark") zone in jeopardized myocardium ($T_2$-weighed imaging) **(E)** exhibiting low $T_1$ values (850 ms centrally vs. 1,135 ms peripherally; 1,005 ms remote myocardium) **(F)** and low $T_2$* values (13 ms) **(G)**, whereas LGE findings **(H)** are comparable to day 1 **(D)**. Occurrence of apical LV thrombus at day 5 (Online Video 5). **Teaching point:** IMH is a marker of severe myocardial damage in acute MI, is associated with poor LV function, lack of functional recovery, and adverse LV remodeling (2). Abbreviations as in **Figures 1 and 3**.

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**KEY WORDS** acute myocardial infarction, magnetic resonance imaging, tissue characterization

**APPENDIX** For supplemental videos, please see the online version of this article.