Cardiac MR Imaging in Acute Coronary Syndrome : Application and Image Interpretation

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INTRODUCTION

- **ACS** → frequent cause of hospitalization and coronary intervention.

- **Cardiac MR Imaging** :

- initial work-up of chest pain
- early post reperfusion
- follow-up evaluation of ACS.

- MR Imaging can evaluate prognostic indicators of myocardial damage.
- MR Imaging is useful to rule out other causes of acute chest pain.

Acute Coronary Syndrome (ACS)

Spectrum of clinical symptoms compatible with acute myocardial ischemia.

- Unstable Angina
- STEMI
- N-STEMI

Underlying Mechanism of ACS

= partial / complete luminal thrombosis of an epicardial coronary artery, with or without vasospasm, due to coronary plaque enlargement, instability, rupture or erosion.

STEMI (thrombus is mostly *fibrin rich*) → Total Occlusion

N-STEMI (thrombus is predominantly *platelet rich and unstable*) → Partial or Intermittent Occlusion

DIAGNOSIS OF ACS

- Both **Unstable Angina and N-STEMI**, exhibit one or more of the following :
  - ✓ Rest angina (> 20 min)
  - ✓ New-onset angina (< 2 month previously)
  - ✓ Angina with increasing intensity and duration

  *Unstable angina → serum biomarkers are negative

  *N-STEMI → increase in cardiac marker and without ST-segment elevation.

- **STEMI**
  - ✓ prolonged chest discomfort (unrelieved by sublingual nitrogliserin)
  - ✓ ST-segment elevation on ECG
  - ✓ Increase in cardiac marker

CARDIAC MR IMAGING

Initial work-up for diagnosis of chest pain

Early after reperfusion

Late after reperfusion

A. Patient with symptoms suggestive of ACS, negative serum biomarkers and non-diagnostic ECG changes (Low Risk Group)

- MR Imaging → assess myocardial function, perfusion and viability.
- *Rule out* other causes of acute chest pain :
  - myocarditis
  - aortic dissection
  - aortic stenosis
  - Takotsubo cardiomyopathy

B. Early after Reperfusion Treatment (within first week)

Cardiac MR imaging can evaluate a variety of imaging parameters :

- regional myocardial dysfunction
- infarct distribution
- infarct size
- myocardium at risk (edema)
- Microvascular obstruction (MVO), and
- Intramyocardial hemorrhage

C. Late after Reperfusion Treatment (within 4-6 weeks)

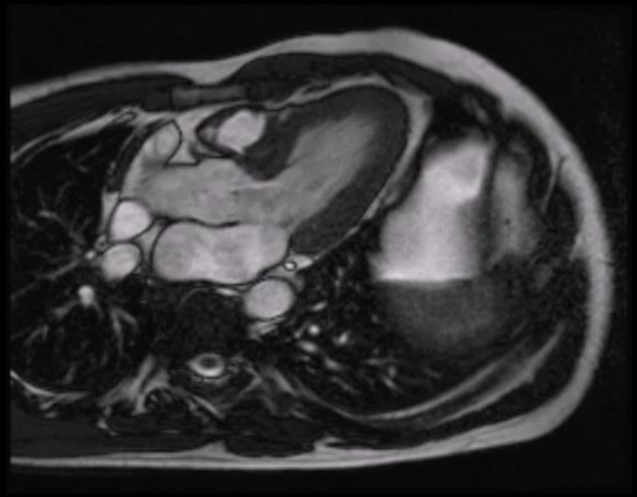
- MR Imaging is used to evaluate the final infarct size.

Cardiac MR Imaging Protocol for Early Post-Reperfusion ACS Patients Including Standard and Optional Sequences

Time (min)	Series	Technique
10	Function	Localization, short-axis function series Sequence: balanced steady-state free precession
20	Function	Long-axis series Outflow tracts: long axis and cross sectional Optional low-dose dobutamine stress (10 μg/kg/min in 5–10 minutes delay) in select cases
30	Edema detection	Short-axis series Triple IR fast spin-echo and/or diffusion-weighted imaging (b = 0, 50 sec/mm^2) (edema detection)
35	Hemorrhage detection	Optional series for detection of hemorrhage Precontrast T1 weighted or T2* weighted
40	Rest perfusion	4–5 short-axis sections (MVO detection) 0.05 mmol/kg gadolinium-based (first half dose) contrast material at 4 mL/sec injection rate The second half of contrast material will be injected
48	Early enhancement	Optional Early gadolinium enhancement (within 3 minutes) with inversion time of approximately 450–600 msec (MVO detection) Short- and long-axis series using IR balanced steady-state free precession or gradient echo
65	Late enhancement	Scout IR (inversion time ~ 250–350 msec) or phase-sensitive IR Short- and long-axis views at myocardial null point To show infarcted myocardium and MVO

Note.—IR = inversion recovery.

CMR can assess many different aspects of cardiac structure and function, all in a single imaging study



❖ 1. ASSESSMENT OF LV FUNCTION

- **Systolic LV function → primary prognostic factor after STEMI**

- **LV regional dysfunction** due to necrosis and stunning of viable myocardium.

* Dopamine can improve contractile function in stunned myocardium.

* Myocardial stunning → recover within 2 weeks

- Improvement of LV functional impairment depends on the infarct size and transmural extent of necrosis.

Extensive Acute MI often causes :

- Progressive LV enlargement
- Gradual deterioration of cardiac EF

Higher LV end systolic volume
Lower EF
High systolic wall stress

→ **Aggressive Care**

- **Assessment of contractile reserve of myocardium**

  (with low-dose dobutamine stress)

  → Important for predicting systolic recovery after STEMI.

  * >5 % increase in EF = positive response

  * Wall thickening ≤ 2 mm (end-systolic minus end-diastolic) → Abnormal

❖ 2. EVALUATION OF VIABILITY

- **Assessment of the size and extent of MI** with MR imaging, performed with Late Gadolinium Enhancement (LGE).

  Sequences : - ECG-gated IR sequence

  - phase-sensitive IR segmented 2D fast gradient-echo

- Inversion Time is set to null the myocardium.
- Images are acquired in **mid to late diastole** (to minimize cardiac motion).
- Following contrast agent administration → 10-30 minutes delay.

Infarcted Myocardium
→ Areas of subendocardial enhancement (high signal intensity)

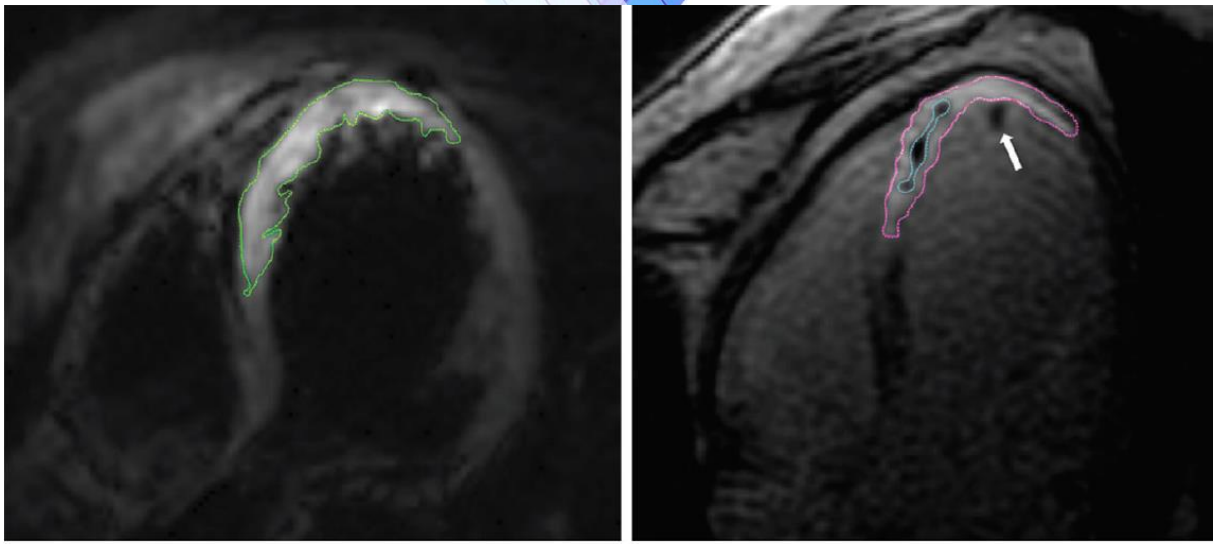
Infarct size – **best predictor for adverse outcome** in comparison late LV dysfunction.

- Persistent ST-segment elevation after anterior MI is largely related to the *larger extent of transmural necrosis* and *persistent microvascular damage*.

Transmural infarct = 63% in STEMI and 27% in N-STEMI.

Differentiate Acute from Chronic MI

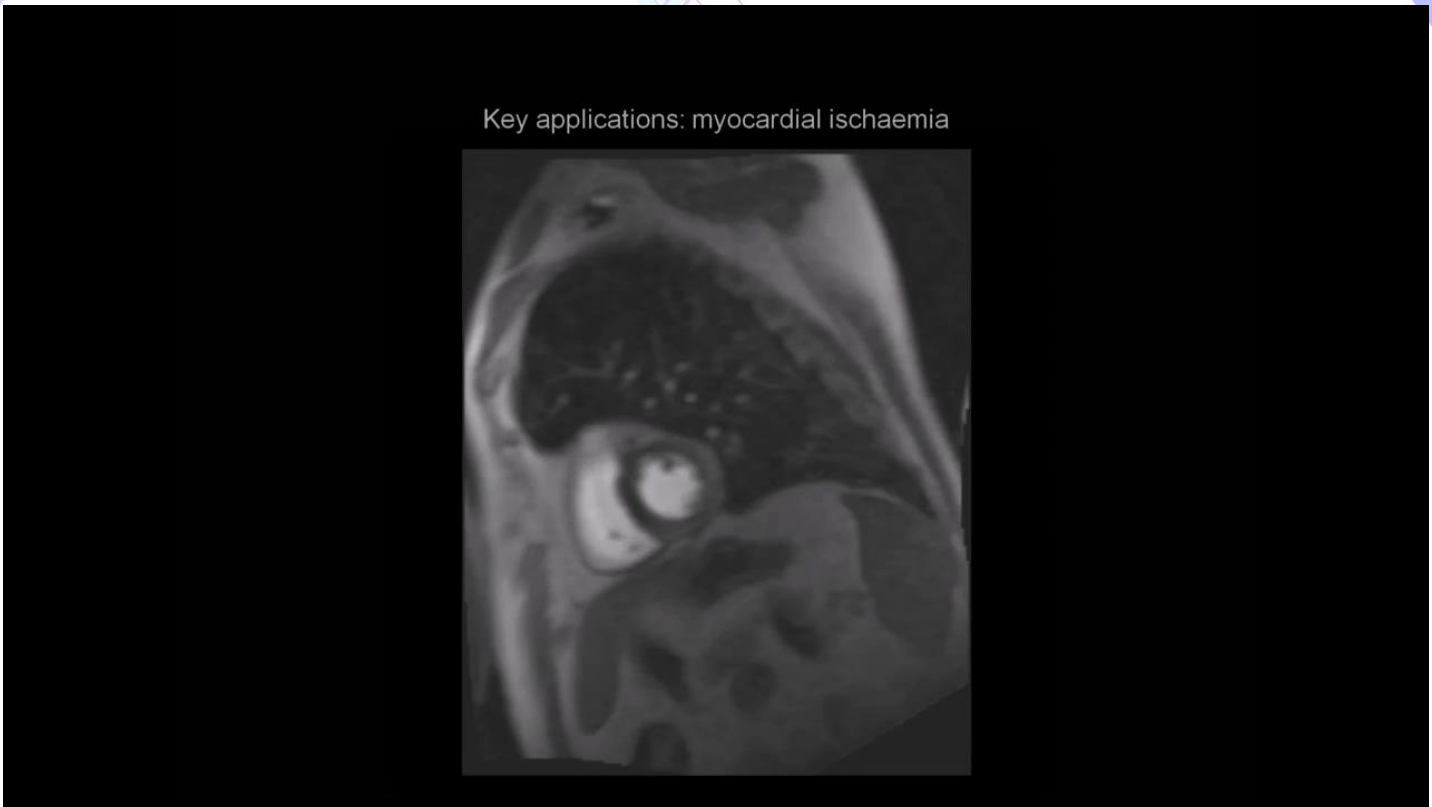
ACUTE	CHRONIC
• Abnormality wall motion • Will enhance after administration of contrast agent. • T2 hyperintensity in edema detection and MVO on LGE series	• Abnormality wall motion • Will enhance after administration of contrast agent. • Persistent thining of myocardial wall • Gray zone



a. b.

Figure 1: Images in 63-year-old man after STEMI. MR imaging depicted low ejection fraction around 20%, moderately dilated LV, and transmural MI involving the anteroseptal wall of the LV at mid and apical levels. **(a)** Four-chamber T2-weighted diffusion-weighted (b = 50 sec/mm^2) image shows high-signal-intensity myocardial edema involving the septum and apex (green area). **(b)** Corresponding LGE image shows large MI (pink area) in the same region and small foci of MVO (blue area) within it. A small apical thrombus is seen (arrow). In post-STEMI patients, the average size of MI is 17% and the average size of edema is 35% of the LV mass (20). Larger MIs, less salvageable myocardium, larger extent of MVO, and low ejection fraction are high-risk markers for adverse cardiovascular events.

Acute MI



Chronic MI

When we see incidental Q waves on an ECG, we ask the question:

"Has this patient had a silent myocardial infarction?"

Cardiac MRI can be a very effective way of finding out

❖ 3. EVALUATION OF MYOCARDIUM AT RISK

- **Most Important Goal of any infarct reperfusion strategy** :
  Salvage the Myocardium at Risk as soon as possible.

- As an infarct develops

Myocardial Edema → early marker of myocardial at risk in ACS

- Detection with T2-weighted sequences :
  - ✓ ECG-gated dark-blood fat-suppressed sequences (triple IR fast spin-echo) → applied before the injection of contrast material.
  - ✓ Short-tau inversion recovery (STIR) sequences

    --- to avoid artifact : bright blood T2-prepared steady-state free precession sequence.
  - ✓ T2 mapping

- Low-b-value diffusion-weighted imaging (provides better contrast)
- Native T1 mapping
- Early gadolinium enhanced (EGE) imaging

McAlindon et al…
- **Size of myocardial edema** is largest with **T2-weighted STIR**, with 5% reduction on T2 mapping and 23% reduction in bright blood steady-state free precession sequence.
- Size enhancement on EGE images was 22% smaller than T2-weighted STIR

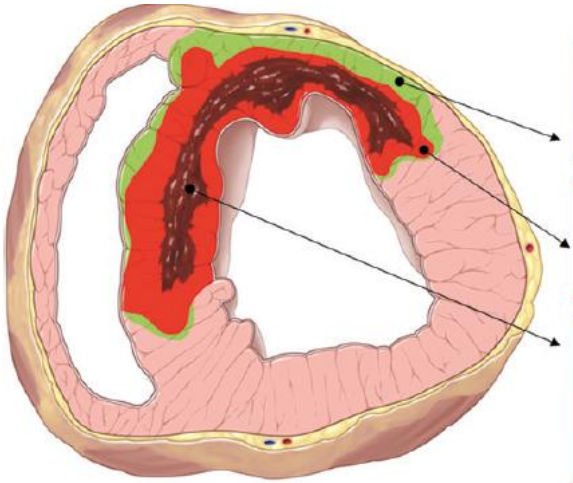
Potet et al…
- **Low-b-value DWI** may reflect lower microvascular perfusion and not just edema, signal-to-noise ratio is acceptable, improved suppression of blood pool signal.

Others…
- Size on **EGE images** is larger than on LGE images and correlates with the size of edema shown on T2-weighted STIR images.

Noted

Edema within the area at risk is a dynamic process, dependent on multiple variables :

- ❑ Infarct size
- ❑ Reperfusion status
- ❑ Time of MR study after reperfusion



	Pathophysiology	MR appearance
Area at risk	No cell destruction Edema Intact microvasculature	No LGE Bright on T2W Normal on rest MPI
Acute infarction	Cell destruction Edema Intact microvasculature	LGE Bright on T2W Normal on rest MPI
MVO	Occluded microvasculature Hemorrhage	MVO=dark on MPI/EGE/LGE may refill on very LGE Bright on T1W and hypointense on T2*

Figure 2: Schematic of the components of injured myocardium after reperfusion for STEMI. Area at risk = salvageable myocardium, *EGE* = early gadolinium enhancement, *MPI* = myocardial perfusion imaging. *T1W* = T1 weighted, *T2W* = T2 weighted.

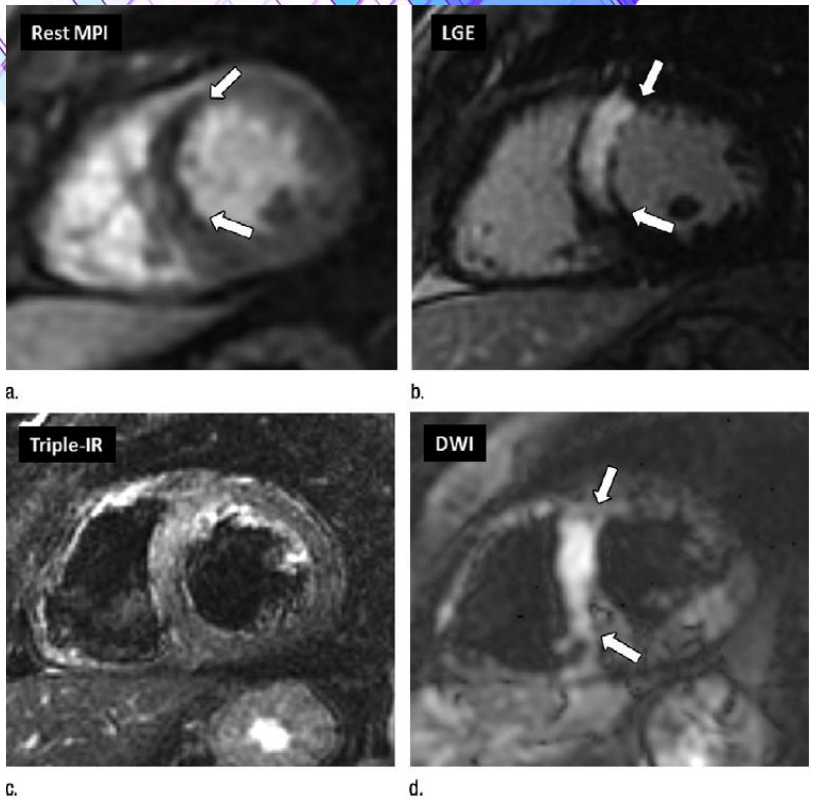


Figure 3: Detection of myocardium at risk. Short-axis MR images at midventricle level 48 hours after reperfusion therapy in a 56-year-old man with STEMI show a large transmural septal infarct. **(a)** Rest myocardial perfusion image *(MPI)* shows decreased signal intensity (arrows) in the anterior septum suggestive of MVO or infarct. **(b)** LGE image shows transmural infarct involving the entire thickness of the septum (between arrows). **(c)** Triple IR and **(d)** diffusion-weighted *(DWI)* (b = 50 sec/mm^2) images obtained to evaluate the extent of edema. Area of edema (between arrows on **d**) is better shown on diffusion-weighted image and matches with that on LGE image. Triple IR image shows bright-rim artifact along the subendocardial margin of the anterior wall. Blood-pool artifacts are less/lower on diffusion-weighted images compared with triple IR, whereas diffusion-weighted image suffers from image distortion and motion artifacts.

❖ 4. DETECTION OF MVO

Following reperfusion, **MVO** can occur due to :

- Swelling of endothelial cells
- Microembolization of plaque debris and fibrin clots
- Secondary inflammatory response (by release of vasogenic and thrombogenic factors)

⇒ Reperfusion Injury

Distribution MVO

* STEMI : left anterior descending artery or right coronary artery
* N-STEMI : left circumflex coronary artery

The extent of **MVO worsens** with longer durations of ischemia and is proportionally related to the size of MI.

MR Imaging Diagnosis of MVO

- Gadolinium-enhanced cardiac MR → diagnose and evaluate the extent of MVO.
- MVO increase in the first 48 hours, but it remains stable or become smaller between 2 and 9 days after reperfusions.

Sequences :

i. First-pass rest perfusion

- ✓ During bolus injection (4-5 mL/sec) of half dose of gadolinium (0.05-0.1 mmol/kg)

---- the 2nd contrast agent is injected after rest perfusion.

ii. LGE

- ✓ IR fast gradient-echo sequence (15-30 minutes after 2nd contrast agent dose and inversion time is set)

iii. EGE (< 3 minutes after 2nd contrast)

Infarcted Myocardium

Hyperenhanced or ‘bright’

(compared with normal myocardial)

MVO Zone

Central hypoenhanced region within the hyperenhanced region

(in EGE and LGE)

Size of MVO,

depends on :

- The sequence
- Duration of occlusion
- Infarct size
- Length of reperfusion
- Timing of imaging after MI

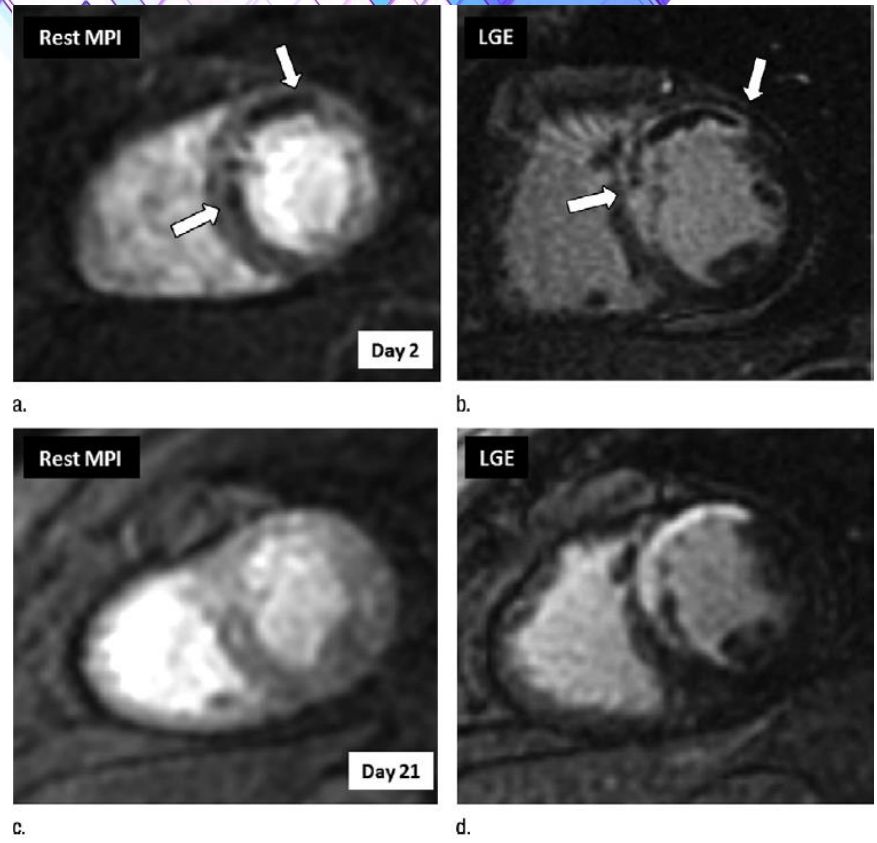


Figure 4: Images in 64-year-old man after reperfusion for STEMI. Two MR studies at **(a, b)** day 2 and **(c, d)** day 21 after reperfusion. (**a, c**) Short-axis rest myocardial perfusion images *(MPI)* and (**b, d**) 15-minute LGE images show temporal changes of MVO area in the anteroseptal myocardium. The MVO appears as a signal void usually on the endocardial side on myocardial perfusion images (arrows in **a**) surrounded by hyperenhanced tissue on early gadolinium enhanced and LGE images (arrows in **b**). The size of MVO depends on the sequence, duration of occlusion, infarct size, length of reperfusion, and timing of imaging after MI. MVO is best shown between day 2 and day 7 after reperfusion and may persist up to 4 weeks.

❖ 5. ASSESSMENT OF REPERFUSION HEMORRHAGE

Presence of intramyocardial hemorrhage after reperfusion

↓

Adverse LV Remodeling and poor prognosis

Hemorrhage usually occurs within the area of MVO, and correlates with the size of MVO.

1 weeks after reperfusion : No Reflow and Hemorrhage

Sequences :

- T2-weighted mapping
- Dual IR black-blood gradient multiecho T2* MR sequence
- T1-weighted IR sequence

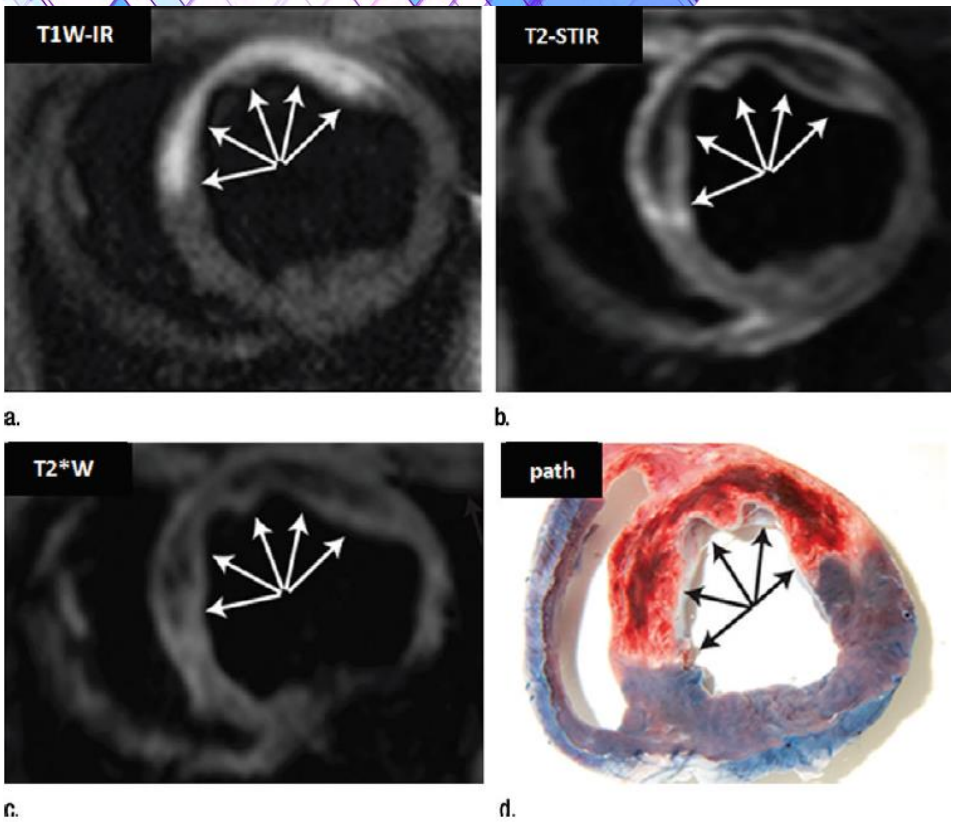


Figure 5: Short-axis cardiac MR images and corresponding pathologic slice obtained 4 days after ischemic reperfusion injury. In the anteroseptal myocardium, a distinct hyperintense core region (arrows) is present on the **(a)**T1-weighted IR image, while a hypointense core region (arrows) is seen on **(b)** T2-weighted STIR and **(c)**T2*-weighted images. **(d)** Each of the observed regions corresponds to intramyocardial hemorrhage (arrows) as confirmed at pathologic examination. (Reprinted, with permission, from reference 117.)

❖ 6. EVALUATION OF COMPLICATIONS

Important complications :

- ✓ Mitral regurgitation (subacute phase)
- ✓ Papillary necrosis
- ✓ LV thrombus
- ✓ Ruptured ventricular septum
- ✓ Aneurysm and pseudoaneurysm

CONCLUSION

MR Imaging

- ✓ initial work-up of chest pain
- ✓ early post reperfusion
- ✓ follow-up evaluation of ACS

Radiologist are encouraged to be familiar with MR characteristics of reperfusion injury

Knowing MR sequences and understanding the limitation of the sequences can improve image interpretation

thank you..