Ischemic and Nonischemic Cardiomyopathy: Delayed Enhancement Patterns on Cardiac MRI

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Learning Objectives

1. To discuss the physical principles and physiologic basis of myocardial delayed enhancement on cardiac MRI

2. To describe the differences in patterns between ischemic and nonischemic cardiomyopathy on cardiac MRI

Not covered

• Myocardial perfusion imaging
• Stress imaging
• Myocardial stunning and hibernation
• Many types of nonischemic cardiomyopathy

Physiology of myocardial delayed enhancement

Normal myocardium

- Rapid “wash-in” of extracellular Gd contrast
- Rapid “wash-out” of extracellular Gd contrast

Abnormal myocardium

- Delayed contrast “wash-in” from slow perfusion
- Delayed contrast “wash-out”, i.e. contrast retention in interstitium due to collagen matrix in fibrosis, amyloid glycoprotein deposition, etc.

Physical Principles of Delayed Enhancement with MRI

Vogel-Claussen et al. RadioGraphics 2006; 26:795-810
Inversion Time (TI)

Vogel-Claussen et al. Radiographics 2006; 26:795-810

Inversion Time Selection

Delayed Enhancement: Localization

- Myocardial Segments
- Myocardial Layers
- Coronary Territories

Myocardial Layers

- Subendocardium
- Mesocardium
- Subepicardium
- Transmural: all layers

Myocardial Segmentation

Long axis: LV divided into thirds:
- Apical
- Mid
- Basal

Short axis: 17 segments
- Apex: 1
- Radial division
  - Apical: 4
  - Mid: 6
  - Basal: 6


Left Ventricular Segmentation

1. basal anterior
2. basal anteroseptal
3. basal inferoseptal
4. basal inferior
5. basal inferoapical
6. basal anterolateral
7. mild anterior
8. mild anteroseptal
9. mild inferoseptal
10. mild inferior
11. mild inferoapical
12. mild anterolateral
13. apical anterior
14. apical anteroseptal
15. apical inferoseptal
16. apical inferior
17. apex
**Ischemic vs Non-ischemic**

**Ischemic**
- Focal subendocardial or transmural delayed enhancement
- Conforms to coronary vascular territory

**Non-ischemic**
- Typically spares subendocardium
- Does not conform to coronary territory
- Can be focal, multifocal, or diffuse

**Ischemic Patterns**

- Subendocardial
- Transmural
- Microvascular obstruction

**Examples**

- Subendocardial DE anterior wall: LAD territory
- Transmural DE apex & anterior wall: LAD territory

**Microvascular Obstruction**

Myocardial microcirculation (arterioles and capillaries)
- Obstructed: Plugged by microthrombi, neutrophils

Delayed enhancement imaging
- Infarct: Enhances slowly (“wash-in”); stays enhanced because of slow clearance (“wash-out”) of extracellular contrast
- MVO
  - Remains unenhanced
  - No contrast “wash-in”; nothing for “wash-out”
  - Looks “null” like normal myocardium
  - Subendocardial core of a transmural (or near-transmural) infarction

**Microvascular obstruction**

- Infarct: Delayed enhancement
- Area at risk: Reduced enhancement
- Normal myocardium: Enhanced
MVO versus thrombus

MVO versus thrombus

Delayed Enhancement Patterns


Nonischemic Cardiomyopathies: Selected Examples

- Myocarditis
- Amyloid
- Sarcoi
- Hypertrophic Cardiomyopathy
- Dilated Cardiomyopathy
- ARVD
- Noncompaction

Patterns

Layer:
- Subendocardial, subepicardial, mesocardial

Morphology:
- Linear, patchy, or nodular

Nonischemic:
- >1 vascular territory when subendocardial or spare subendocardium

Myocarditis

- Mesocardium
- Subepicardium
- Patchy

Myocarditis: 19 year old male with chest pressure and elevated troponin

T2-weighted IR FSE Basal inferolateral wall High signal = edema

T1-weighted IR GRE Basal inferolateral wall High signal = DE
**Amyloid**

- Diffuse subendocardial or transmural

**Amyloid**

Inversion time selection **BEFORE** contrast

- Inversion time selection **AFTER** contrast

- Delayed **REPEAT** inversion time selection

**Amyloid**

- Delayed enhancement pattern:
  - Global subendocardial or transmural
  - Occasionally more patchy and scattered
  - Guides endomyocardial biopsy

**Sarcoid**

- Patchy or nodular

- Subepicardial or mesocardial

- Negative if on steroids

**Hypertrophic Cardiomyopathy**

- Patchy or nodular

- Mesocardial (mid wall)

- Interventricular septum by RV insertion sites

**Differentiation of amyloid and “balanced” ischemic cardiomyopathy on DE:**

- Hypertrophic rather than dilated and thinned myocardium

- Pan-chamber hypertrophy and delayed enhancement

Hypertrophic Cardiomyopathy

Asymmetric septal hypertrophy

Still image from cine steady-state free precession

Delayed enhancement


Dilated Cardiomyopathy

• Mesocardium

Dilated cardiomyopathy

Most common form of nonischemic cardiomyopathy
• 50%=idiopathic
• Other 50% = sequelae of myocarditis, drug toxicity, alcohol abuse

Delayed enhancement
• ± Mesocardial stripe in interventricular septum
• Marker for increased risk of sudden cardiac death: arrhythmogenic focus

Idiopathic Dilated CM

Arrhythmogenic Right Ventricular Cardiomyopathy

• Variable pattern RV free wall and interventricular septum


ARVD

NOT major or minor 2010 Task Force criterion:
– Abnormal non-contrast myocardial signal suggestive of fibrofatty change
– Delayed enhancement

2010 ARVD Task Force criteria for MRI:
– RV dilatation and wall motion abnormalities ONLY

Abnormal myocardial signal on non-contrast MRI and/or DE: site(s) for potential myocardial biopsy
Noncompaction

• Variable foci in areas of noncompaction, i.e. hypertrabeculation

Noncompaction

Apical-to-mid chamber LV hypertrabeculation and thinning of underlying compacted myocardium (static images from cine steady-state free precession)

Tiny foci of DE: trabeculations and papillary muscles

White JA, Patel MR. Magn Reson Imaging Clin No Am 2007;541-564

Delayed Enhancement Patterns


Algorithm for Delayed Enhancement in Patients with Cardiomyopathy

References
