Delayed enhancement MRI imaging Patterns, pitfalls and pathology

Prabhakar Rajiah, MBBS, MD, FRCR

Cardiothoracic Imaging, Department of Radiology
University Hospitals Cleveland Case Medical Center, Ohio, United States

Learning Objectives

- To review the role of delayed enhancement MRI in evaluation of myocardial infarcts and non ischemic cardiomyopathies
- To learn the pattern of abnormalities in delayed enhancement cardiac MRI
- To recognize the pitfalls in delayed enhancement imaging

Delayed enhancement cardiac MRI

- Delayed enhancement (DE) imaging is an integral component of cardiac MRI
- Useful in ischemic and non-ischemic cardiomyopathies
- Characterization of cardiomyopathies is essential for proper treatment
- Presence of scar/fibrosis indicates bad prognosis
  - Arrhythmogenic focus
  - Higher incidence of adverse cardiovascular events
- DE imaging is also useful in the evaluation of cardiac masses and pericardium

Mechanism of Delayed Enhancement

- Normal myocardium
- Acute injury
  - Cellular injury and loss of cell membrane integrity
  - Expansion of extracellular space
- Chronic injury
  - Scar or fibrosis
- Delayed Contrast Enhancement

- Normal myocardium
- Contrast enhancement immediately after administration, but washes out quickly
- Abnormal myocardium
- Washes in slow

Mechanism of Delayed Enhancement

- Normal myocardium
- Normal extracellular space
- Scar tissue/fibrosis
- There's significant expansion of extracellular space with scar tissue, with retained contrast

Mechanism of Delayed Enhancement

- Normal myocardium
- Normal extracellular space
- Scar tissue/fibrosis
- There's significant expansion of extracellular space with scar tissue, with retained contrast

Delayed Contrast Enhancement
**Technique**

- Segmented k-space inversion recovery gradient echo pulse sequence.
- 10-15 minutes after intravenous injection of 0.1-0.2 mmol/kg of Gadolinium.
- Inversion time set to null signal from normal myocardium.
- Look and learn.

**Patterns of Enhancement**

**INFARCT**

1. **Subendocardial infarct** in LAD territory
2. **Full thickness transmural infarct** in RCA territory

**NON-ISCHEMIC**

- Mid myocardial linear enhancement may be seen in idiopathic type
- Basal and mid septum

**Myocardial Infarction**

- Delayed enhancement seen both in acute and chronic myocardial infarction.
- Sub-endocardial or transmural scarring in a vascular distribution is characteristic.
- Extent of scarred myocardium determines patients suitable for revascularization. Dysfunctional segments without scarring are probably hibernating. Hence benefit from bypass surgery.

<table>
<thead>
<tr>
<th>Delayed enhancement</th>
<th>Function</th>
<th>Viability</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;50 %</td>
<td>Dysfunctional</td>
<td>Viable</td>
</tr>
<tr>
<td>&gt;75 %</td>
<td>Dysfunctional</td>
<td>Non viable</td>
</tr>
<tr>
<td>51-75%</td>
<td>Dysfunctional</td>
<td>Uncertain</td>
</tr>
</tbody>
</table>

**Myocardial Infarction**

- Delayed enhancement seen both in acute and chronic myocardial infarction.
- Sub-endocardial or transmural scarring in a vascular distribution is characteristic.
- Extent of scarred myocardium determines patients suitable for revascularization. Dysfunctional segments without scarring are probably hibernating. Hence benefit from bypass surgery.

**Patterns of Enhancement**

**INFARCT**

1. **Subendocardial infarct** in LAD territory
2. **Full thickness transmural infarct** in RCA territory

**NON-ISCHEMIC**

- Mid myocardial linear enhancement may be seen in idiopathic type
- Basal and mid septum

<table>
<thead>
<tr>
<th>Delayed enhancement</th>
<th>Function</th>
<th>Viability</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;50 %</td>
<td>Dysfunctional</td>
<td>Viable</td>
</tr>
<tr>
<td>&gt;75 %</td>
<td>Dysfunctional</td>
<td>Non viable</td>
</tr>
<tr>
<td>51-75%</td>
<td>Dysfunctional</td>
<td>Uncertain</td>
</tr>
</tbody>
</table>

**Myocardial Infarction**

- Delayed enhancement seen both in acute and chronic myocardial infarction.
- Sub-endocardial or transmural scarring in a vascular distribution is characteristic.
- Extent of scarred myocardium determines patients suitable for revascularization. Dysfunctional segments without scarring are probably hibernating. Hence benefit from bypass surgery.

<table>
<thead>
<tr>
<th>Delayed enhancement</th>
<th>Function</th>
<th>Viability</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;50 %</td>
<td>Dysfunctional</td>
<td>Viable</td>
</tr>
<tr>
<td>&gt;75 %</td>
<td>Dysfunctional</td>
<td>Non viable</td>
</tr>
<tr>
<td>51-75%</td>
<td>Dysfunctional</td>
<td>Uncertain</td>
</tr>
</tbody>
</table>

**Myocardial Infarction**

- Delayed enhancement seen both in acute and chronic myocardial infarction.
- Sub-endocardial or transmural scarring in a vascular distribution is characteristic.
- Extent of scarred myocardium determines patients suitable for revascularization. Dysfunctional segments without scarring are probably hibernating. Hence benefit from bypass surgery.

<table>
<thead>
<tr>
<th>Delayed enhancement</th>
<th>Function</th>
<th>Viability</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;50 %</td>
<td>Dysfunctional</td>
<td>Viable</td>
</tr>
<tr>
<td>&gt;75 %</td>
<td>Dysfunctional</td>
<td>Non viable</td>
</tr>
<tr>
<td>51-75%</td>
<td>Dysfunctional</td>
<td>Uncertain</td>
</tr>
</tbody>
</table>

**Myocardial Infarction**

- Delayed enhancement seen both in acute and chronic myocardial infarction.
- Sub-endocardial or transmural scarring in a vascular distribution is characteristic.
- Extent of scarred myocardium determines patients suitable for revascularization. Dysfunctional segments without scarring are probably hibernating. Hence benefit from bypass surgery.

<table>
<thead>
<tr>
<th>Delayed enhancement</th>
<th>Function</th>
<th>Viability</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;50 %</td>
<td>Dysfunctional</td>
<td>Viable</td>
</tr>
<tr>
<td>&gt;75 %</td>
<td>Dysfunctional</td>
<td>Non viable</td>
</tr>
<tr>
<td>51-75%</td>
<td>Dysfunctional</td>
<td>Uncertain</td>
</tr>
</tbody>
</table>

**Myocardial Infarction**

- Delayed enhancement seen both in acute and chronic myocardial infarction.
- Sub-endocardial or transmural scarring in a vascular distribution is characteristic.
- Extent of scarred myocardium determines patients suitable for revascularization. Dysfunctional segments without scarring are probably hibernating. Hence benefit from bypass surgery.

<table>
<thead>
<tr>
<th>Delayed enhancement</th>
<th>Function</th>
<th>Viability</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;50 %</td>
<td>Dysfunctional</td>
<td>Viable</td>
</tr>
<tr>
<td>&gt;75 %</td>
<td>Dysfunctional</td>
<td>Non viable</td>
</tr>
<tr>
<td>51-75%</td>
<td>Dysfunctional</td>
<td>Uncertain</td>
</tr>
</tbody>
</table>
**Hypertrophic cardiomyopathy**
- Fibrosis due to increase in extracellular space, abnormal extracellular matrix due to myofibrillar disarray, with plexiform fibrosis and microscopic/ macroscopic replacement fibrosis seen in up to 80%.
- Acute, subacute, late hypertrophic.
- Basal mid anteroseptum/subepicardium, LV outflow points, all sites of maximal thickness.
- Associated with regional wall motion abnormalities.
- Burned out phase- thinned, full thickness.

**Myocarditis**
- Acute myocarditis: Commonly due to viral infection, can be idiopathic or immune mediated.
  - DE- Begins in epicardial zone, extends to variable degrees into myocardium.
  - Associated with regional wall motion abnormalities.
- Chronic myocarditis- Mid wall enhancement like dilated cardiomyopathy.
  - Parvovirus- inferolateral segment, subepicardial, good prognosis; HHV-6 virus- septal segment, midmyocardial, progression to cardiac failure.

**Sarcoidosis**
- Non-caseating granulomas.
- Cardiac involvement: 20-27%.
- Mid myocardial/subepicardial scarring.
- Subendocardial/transmural rare.
- Severe enhancement- associated with poor function.
- Acute phase- Myocardial edema.
- MRI guides endomyocardial biopsy sampling.

**Amyloidosis**
- Extracellular accumulation of β-pleated sheet fibrillary protein.
- AL,amilodial, senile systemic, reactive systemic.
- Concentric LV thickening with reduced systolic, diastolic function.
- Global, diffuse subendocardial enhancement in non-coronary distribution, which progresses to transmural.
- Enhancement in thickened atria, interatrial septum, valves.
- Altered T1 kinetics, earlier nulling of the myocardium than blood pool.
- Gd washes out faster than normal patients due to distribution into amyloid in body.

**LV Non compaction**
- Exaggerated trabeculation, non-compacted myocardium- 2-3 times compacted myocardium.
- Trabecular delayed enhancement in non-compacted areas.
- Inverse correlation with function.
- Presence in mid ventricle- bad prognosis.
**ARVD**

- Arrhythmogenic right ventricular dysplasia
- Progressive fatty or fibrofatty replacement of the RV myocardium.
- Major wall motion abnormality along with global systolic dysfunction/RV dilation
- Fat can be seen in RV free wall in "triangle of dysplasia"
- Diffuse or segmental scar, more common in RVOT and anterobasal wall.

**Fabry’s disease**

- X-linked glycosphingolipid disorder
- Alpha galactosidase A deficiency
- Globotriaosylceramide accumulation
- Symmetrical concentric thickening, reduced LV volume, increased ejection fraction
- RV: subepicardial to mid-myocardial
- Basal inferobasal segment is most commonly involved
- 4-15% of patients diagnosed as HOCM, bool Fabry’s disease

**Pulmonary hypertension**

- Septal and subepicardial enhancement at the right ventricular insertion points is common in pulmonary hypertension
- The level of systolic pulmonary pressure elevation appears to be the main determinant of the delayed enhancement

**Pericarditis**

- Pericarditis can be acute or chronic
- Diffuse enhancement of the pericardium correlates with pericardial inflammation
- Epicardial fat may enhance
- Constriction associated with pericardial inflammation may respond to medical management

**Thrombus**

- Thrombus is seen in areas of dyskinesis
- LA in mitral valve disease
- Delayed enhancement-Dark non-enhancement
- Delayed enhancement at high TI (600 ms): Myocardium bright, clot very dark
- Chronic organized thrombus: heterogeneous intermediate signal, patchy areas of enhancement due to neovascularity

**Tumors**

- Neoplasms show variable degree of delayed enhancement
- Diagnosis based on location, morphology and pattern of enhancement
- Malignancies: Irregular, infiltrative with heterogeneous enhancement
**Enhancement patterns**

<table>
<thead>
<tr>
<th>Disease</th>
<th>Pattern Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ischemic</td>
<td>Subendocardial/transmural in vascular distribution</td>
</tr>
<tr>
<td>Hypertrophic</td>
<td>Patchy, sandy, midmyocardial in hypertrophied areas, especially the septal insertion</td>
</tr>
<tr>
<td>Idiopathic dilated</td>
<td>Mid myocardial, septum, LV dilation, global LV dysfunction</td>
</tr>
<tr>
<td>Sarcoidosis</td>
<td>Subepicardial, progressing inwards</td>
</tr>
<tr>
<td>Amyloidosis</td>
<td>Diffuse, subendocardial to epicardial, altered Ti kinetics</td>
</tr>
<tr>
<td>Anderson Fabry</td>
<td>Mid myocardial, basal inferolateral wall, symmetrically thickened myocardium, normal or increased systolic function</td>
</tr>
<tr>
<td>Myocarditis</td>
<td>Subepicardial progressing inwards</td>
</tr>
<tr>
<td>Chronic myocarditis</td>
<td>Mid myocardial, linear</td>
</tr>
<tr>
<td>ARVD</td>
<td>RV free wall/septum, wall motion abnormalities, fatty replacement</td>
</tr>
<tr>
<td>LV Non compaction</td>
<td>Enhancement of non-compacted myocardium</td>
</tr>
<tr>
<td>Pericarditis</td>
<td>Diffuse or focal pericardial enhancement</td>
</tr>
<tr>
<td>Thrombus</td>
<td>No enhancement, in typical and longer (600 milliseconds) inversion time</td>
</tr>
<tr>
<td>Neoplastic masses</td>
<td>Variable enhancement</td>
</tr>
</tbody>
</table>

**Iatrogenic**

- Delayed enhancement is seen at sites of surprise, especially with placement of patches.
- Delayed enhancement is also seen following diastolic/myocardial infarction.

**Wrong inversion time**

- Selecting the optimal inversion time to null the myocardium is essential for good quality DE images.
- Please select inversion recovery sequence independent of inversion time.

**Subendocardial infarct vs blood pool**

- Differentiating subendocardial infarct from blood pool contrast between endocardium and papillary muscles may be challenging.
- Correlate with cine images to determine if enhancement is in myocardium or blood pool.

**Inversion time- amyloidosis**

- Identifying the optimal inversion time may be challenging in amyloidosis, due to diffuse infiltration.
- Ti values in amyloidosis are typically higher and at times lower than blood pool.

**Pitfalls**

- Iatrogenic enhancement
  - Improper imaging time
  - Improper inversion time
  - Differentiating infarct from blood pool
  - Correcting imaging sequence (Ti scout)
  - Correcting imaging sequence (Ti scout)
  - Improper suppression
  - Improper suppression
  - Proper sequence
  - Proper sequence
  - Proper sequence
  - Proper sequence
  - Proper sequence
  - Proper sequence

- Iatrogenic enhancement
  - Improper imaging time
  - Improper imaging time
  - Differentiating infarct from blood pool
  - Correcting imaging sequence (Ti scout)
  - Correcting imaging sequence (Ti scout)
  - Improper suppression
  - Improper suppression
  - Proper sequence
  - Proper sequence
  - Proper sequence
  - Proper sequence
  - Proper sequence
  - Proper sequence
  - Proper sequence
  - Proper sequence

**Iatrogenic**

- Delayed enhancement in the basal ventricular septum at site of VSD patch repair.

**Wrong inversion time**

- Selecting the optimal inversion time to null the myocardium is essential for good quality DE images.
- Please select inversion recovery sequence independent of inversion time.

**Subendocardial infarct vs blood pool**

- Differentiating subendocardial infarct from blood pool contrast between endocardium and papillary muscles may be challenging.
- Correlate with cine images to determine if enhancement is in myocardium or blood pool.

**Inversion time- amyloidosis**

- Identifying the optimal inversion time may be challenging in amyloidosis, due to diffuse infiltration.
- Ti values in amyloidosis are typically higher and at times lower than blood pool.

**Pitfalls**

- Iatrogenic enhancement
  - Improper imaging time
  - Improper inversion time
  - Differentiating infarct from blood pool
  - Correcting imaging sequence (Ti scout)
  - Correcting imaging sequence (Ti scout)
  - Improper suppression
  - Improper suppression
  - Proper sequence
  - Proper sequence
  - Proper sequence
  - Proper sequence
  - Proper sequence
  - Proper sequence
  - Proper sequence
  - Proper sequence

- Iatrogenic enhancement
  - Improper imaging time
  - Improper imaging time
  - Differentiating infarct from blood pool
  - Correcting imaging sequence (Ti scout)
  - Correcting imaging sequence (Ti scout)
  - Improper suppression
  - Improper suppression
  - Proper sequence
  - Proper sequence
  - Proper sequence
  - Proper sequence
  - Proper sequence
  - Proper sequence
  - Proper sequence
  - Proper sequence

**Subendocardial infarct vs blood pool**

- Differentiating subendocardial infarct from blood pool contrast between endocardium and papillary muscles may be challenging.
- Correlate with cine images to determine if enhancement is in myocardium or blood pool.
Epicardial fat

- Epicardial fat may be incompletely suppressed resulting in artifactual high signal, which may be confused with enhancement.
- Correlate with other imaging planes and sequences to confirm.

Artifacts

- Grading artifacts from cardiac, respiration or gastric motion can mimic lesion.
- Motion artifact is seen in phase encoding direction.

Summary

- Delayed enhancement is useful in characterizing cardiomyopathies.
- It is also useful in determining the presence of viable myocardium in myocardial infarction.
- Scar is an adverse prognostic indicator and is a substrate for ventricular arrhythmias.
- Delayed enhancement is also useful in the evaluation of masses and pericardium.
- Knowledge of pitfalls and artifacts is essential to avoid misdiagnosis.