Overview of complications of acute & chronic myocardial infarction- Revisiting pathogenesis and cross sectional imaging

A. J. Baxi, MD (UT Health Science Center, San Antonio)
R. Katre, MD (UT Health Science Center, San Antonio)
S. Saboo, MD (UT Southwestern, Dallas)
C. Restrepo, MD (UT Health Science Center, San Antonio)

Disclaimer: We do not have any conflict of interest or financial gain to disclose
Myocardial infarction (MI)

- MI is the leading cause of death and a main cause of morbidity in the United States.
- MI is defined as myocardial cell death due to prolonged ischemia resulting in an inadequate supply of oxygenated blood to an area of myocardium.
- Typically caused by luminal thrombosis superimposed on coronary atherosclerosis.
- Occasionally, it can be caused by coronary spasm, coronary embolism, or thrombosis in nonatherosclerotic vessels.
- Arteritis, dissection, congenital abnormalities, hypercoagulable states, and cocaine use are uncommon causes.

RadioGraphics 2013; 33:1383–1412
Introduction

• Various complications may develop after MI, particularly when treatment is delayed or inadequate

• Complications of MI include ischaemic, mechanical, arrhythmic, embolic and inflammatory disturbances

• Other complications: RV infarction & cardiogenic shock

• Most of these complication can be diagnosed on clinical, laboratory and echocardiography evaluation

• At times, cardiac MRI & MDCT is mandatory for evaluating them

• Accurate diagnosis of these complications is very important to optimize proper treatment and patient care

• It is imperative for the radiologist to be aware of and be confident in diagnosing the complications secondary to MI
Learning objectives

1. To discuss pathophysiology of acute and chronic myocardial infarction
2. To study the role of imaging in evaluating complications of myocardial infarction
3. To discuss imaging and differential diagnosis

In this exhibit, we discuss the characteristic multimodality imaging findings and differential diagnosis of common and uncommon complications of myocardial infarction. Increased awareness of such entities will contribute to optimized care of patients.
Arrhythmic complications of acute MI like supraventricular tachyarrhythmias, bradyarrhythmias, intraventricular blocks, etc, can be seen in about 90% of patients during or immediately after the event. The incidence of arrhythmia is higher with an ST-elevation MI and lower with a non-ST-elevation MI. Postgrad Med. 1991 Nov 1;90(6):85-8, 93-6.

Radiological imaging plays no role in accessing these complications.
Myocardial Infarction

- Acute MI is diagnosed when there is a characteristic increase and subsequent decrease in troponin or creatine kinase–MB (heart) fraction levels, along with Q waves or ischemic changes at ECG (ST segment elevation or depression) or coronary artery intervention.
- Acute & chronic infarcts are managed differently, establishing the infarct age is crucial.
- Both can have wall-motion abnormalities, perfusion defects, and scarring.

<table>
<thead>
<tr>
<th></th>
<th>Acute myocardial infarction</th>
<th>Chronic myocardial infarction</th>
</tr>
</thead>
<tbody>
<tr>
<td>High T2 signal (Myocardial edema)</td>
<td>Seen (most useful)</td>
<td>Not Seen</td>
</tr>
<tr>
<td>Delated enhancement</td>
<td>Seen</td>
<td>Seen</td>
</tr>
<tr>
<td>Wall Thickness</td>
<td>Preserved</td>
<td>Decreased</td>
</tr>
<tr>
<td>Regional wall motion abnormality</td>
<td>Seen</td>
<td></td>
</tr>
<tr>
<td>Microvascular obstruction</td>
<td>Seen (Poor prognosis)</td>
<td>Seen in 50% cases</td>
</tr>
</tbody>
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RadioGraphics 2013; 33:1383–1412
Patterns of Myocardial Infarction

Acute infarct with microvascular obstruction
(no-reflow phenomenon seen as Non-enhancing hypo-intensity- arrow)

Acute sub-endocardial MI

Multi-vessel transmural infarct with apical thrombus

Anterior wall

Transmural infarct- septum
True and false aneurysms

- Aneurysm of LV is seen in 12% of patients after MI & can be either true or false

**True:** Large, localized to the apical and anterolateral aspects of the LV wall, and made up of damaged myocardial wall

**False:** Small, usually occur along the posterolateral wall of the LV, and represents localized myocardial rupture covered by pericardial adhesions which shows delayed pericardial)

True Aneurysm with Calcification-Post MI (arrows)
Distinguishing between true vs. pseudo LV-aneurysm is important because the clinical management and complication risks are different.

<table>
<thead>
<tr>
<th></th>
<th>True aneurysm</th>
<th>False aneurysm</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Neck</strong></td>
<td>Wide neck &amp; has all 3 layers of the cardiac wall</td>
<td>Small neck and consists of ½ layers</td>
</tr>
<tr>
<td><strong>Pathology</strong></td>
<td>Progressive thinning of infarcted myocardium leads to aneurysmal dilatation</td>
<td>Free wall rupture of the LV that is contained by the pericardium</td>
</tr>
<tr>
<td><strong>Location</strong></td>
<td>Apex</td>
<td>Free lateral wall</td>
</tr>
<tr>
<td><strong>Delayed enhancement</strong></td>
<td>Usually not seen (Infarcted myocardium enhances)</td>
<td>Usually seen (Pericardium enhances)</td>
</tr>
<tr>
<td><strong>Rupture risk</strong></td>
<td>Low</td>
<td>High</td>
</tr>
</tbody>
</table>
| **Treatment**       | Medical management                                                         | Surgery 
*Br J Radiol. 2011 Feb; 84(998)*
*Radiology 2005;236:65–70* |

Courtesy: H Murillo, M.D. Sacramento, Ca.
LV aneurysm secondary to a large myocardial infarction (arrows). Cardiac gated CT shows abnormal thinning and bulging of the mid and distal left ventricle, as well as low density wall.
LV aneurysm secondary to a large myocardial infarction (arrows). Radiograph showing dilated LV with a curvilinear calcification. Cardiac gated CT shows abnormal thinning and bulging of the distal septum and apex of the left ventricle with calcification.

Teaching point: The infarcted myocardium may develop thinning, fatty replacement and calcification. The poorly contracting infarcted ventricle is also prone to the development of intraluminal thrombus.
Left Ventricular Apical True Aneurysm

65-year-old man with history of LAD territory MI. Four chamber SSFP MR image shows apical myocardial thinning and broad neck aneurysm (yellow arrow)

B. Late gadolinium enhancement inversion recovery vertical long axis image demonstrates transmural enhancement of the distal anterior wall and apex, represents a left anterior descending artery territory scar. (yellow arrow)
CCTA in patient with large, antero-septal and apical remote MI with chronic changes including, thinning, fatty infiltration, and aneurysmal LV apex with paradoxical dilatation during systole. Four-chamber views in systole (a) and diastole (b). Cardiac MRI of same patient demonstrating LV aneurysmal dilatation with delayed enhancement.
60 year old man with history of MI presented with chest pain.
(a) Axial non-contrast CT chest image shows hemopericardium (yellow arrows)
(b) Follow up MRI with Four-chamber (SSFP) image showing a large narrow-necked (arrowhead) pseudoaneurysm arising from the lateral wall of the left ventricle (LV) in LCX territory surrounded by pericardial fluid (orange arrow)
(c) Late gadolinium enhancement (LGE) short axis image shows full thickness LGE in its wall which is due to myocardial scarring, and bordering pericardial thickening (blue arrows)
Left Ventricular Pseudoaneurysm

Post-Acute MI Pseodoaneurysm

LV Apex Pseudoaneurysm-Post MI
84 year old female presenting with back pain and history of remote MI. CTA axial images, coronal and volume rendering reconstructions demonstrate a large, apical false aneurysm (*) with organized mural thrombus and wall calcification.
Pseudo-aneurysm: CT Imaging Findings

Cardiac gated CT shows LV apical pseudoaneurysm with a small neck (arrows)

50 year old female with history of chest pain, hyperlipidemia and cigarette smoking
Cardiac gated CT shows LV apical pseudoaneurysm with a small neck (arrows)
Acute and chronic thrombus

- Thrombus is seen in up to 20% of all infarcts
- 40% in anterior infarcts & 60% in apical infarcts
- Initial imaging by echocardiography
- MRI more sensitive for detection of small infarcts due to high contrast resolution
- MRI: Dark hypointense lesion surrounded by bright dead myocardium
- MDCT: hypodensity in the apical region
- Sometimes chronic thrombus can show patchy enhancement
RV infarction and thrombus

- RV infarction may occur as a complication of inferior-wall MI in up to 25% of patients. It may also be seen secondary to chronic lung disease or RV hypertrophy.
- Atrial infarction (most commonly in right atrium) is seen in 10% of inferior wall infarcts & is detected with delayed-enhancement MR imaging.

75 y/o woman with CAD causing right ventricular (yellow arrows) and basal LV inferolateral MI and chronic total occlusion of the right coronary artery with chronic systolic heart failure, LVEF of 28%, right ventricular failure with associated thrombus (red arrows) on chronic anticoagulation.

RadioGraphics 2013; 33:1383–1412
Laminar LV thrombus (red arrows) in a 55 years old Male with past medical history of CHF, hypertension and hyperlipidemia presented with shortness of breath. Severely reduced left ventricular systolic function (LVEF: 2.3%)

Acute Myocardial with Thrombus in LV
Chronic MI Changes with apical thinning (white arrows) and thrombus Formation (red arrows)

Thrombus can embolize leading to stroke, visceral infarction and death
Embolic Complications

- Usually occur within first 10 days after acute MI
- Incidence of clinically evident systemic embolism after MI is less than 2%
- Incidence increases in patients with anterior wall MI
- Most emboli arise from the left ventricle as a result of wall motion abnormalities or aneurysms
- Atrial fibrillation in the setting of ischemia can also contribute to systemic embolization
- Complications: stroke, although patients may have limb ischemia, renal infarction, or mesenteric ischemia

*Circulation* 2002; 106:2873–2876
Post MI Thrombus (red arrow) Embolizing in Bilateral Legs (white arrows)

35 y/o male
Mitral regurgitation (MR)

- Seen in 11%–59% of patients post-mi
- Major independent adverse prognostic determinant
- Can results from rupture of chordae tendineae & papillary muscle
- Due to restricted leaflet motion resulting in incomplete valve closure
- High rate of recurrence, even after surgical repair of the mitral valve

Papillary muscle rupture (PMR) is not a common finding after acute MI, however, is a life-threatening mechanical complication. PMR usually is seen as a bullet-shaped mass floating from the left ventricle to the left atrium.

*Arch Cardiovasc Imaging. 2015 August; 3(3): e30490*

RadioGraphics 2013; 33:1383–1412
Left Ventricular Free Wall Rupture

- FWR occurs only among patients with transmural MI
- Affects only 0.5% of MI patients with a substantial mortality rate of 20%
- Rupture is usually within 5 days of infarction in 50% of patients and within 2 weeks in 90% of patients
- Risk factors include advanced age, female gender, hypertension, first MI, and poor coronary collateralization
- The aim of treatment is accurate early diagnosis & perform emergent heart surgery to repair the rupture
MDCT images showing hemopericardium (yellow arrows) and mass effect on right cardiac chambers (red arrows) suggesting tamponade. Also seen is thinning and irregularity of apex and lateral wall of left ventricle probably suggesting acute myocardial infarction and rupture (blue arrows).
Ventricular septal defect

- Incidence has decreased dramatically with reperfusion therapy
- Defect usually occurs at the junction of preserved and infarcted myocardium in the apical septum with anterior MI, and in the basal posterior septum with inferior MI
- VSD almost always occurs in the setting of a transmural MI and is more often seen in anterolateral mis
- Early surgical closure is the treatment of choice, even if the patient's condition is stable

Cardiovasc Surg 2003; 11:149–154
J Am Coll Cardiol 2000; 36(3 suppl A):1110–1116
Am J Cardiol 2010; 105:59–63
49 years old man with history of RCA territory MI

A) Two chamber vertical long axis SSFP image shows basal inferior wall left ventricular pseudoaneurysm (red arrow)

B) Four chamber horizontal SSFP image shows large basal septal muscular VSD (blue arrow) and left to right shunt (jet) (Yellow arrow)

C) Inversion recovery Late gadolinium enhancement (LGE) short axis image shows basal inferior LV pseudoaneurysm with full wall thickness LGE consistent with myocardial scarring in basal inferior septum and inferior wall [RCA territory](green arrow)
Cardiac tamponade

- An infrequent but potentially catastrophic complication after acute MI
- Incidence & mortality has significantly decreased after fibrinolytic therapy
- Pathophysiology: Increase in intrapericardial pressure resulting in hemodynamic impairment, decreased blood pressure, cardiac inflow & stroke volume
- Imaging: Pericardial effusion, usually large, with distention of SVC/IVC; deformity & compression of the cardiac chambers and angulation/bowing of IVS

Post-acute myocardial infarction (yellow arrow) perforation leading to pericardial tamponade (red arrow)

Am Heart J. 2006;151(2):316-322
Inflammatory reaction in response to myocardial necrosis

Pathogenesis:

Acute pericarditis
- Seen in transmural MI
- Incidence approximately 10%
- Usually develops 24 and 96 hours after MI

Dressler's syndrome:
- Autoimmune
- Incidence between 1% and 3%
- Typically 2 to 8 weeks after MI

Pericardial effusion
- Seen in 25% of patients with MI
- It is seen more so with cardiac failure, anterior-wall MI, and large infarcts
- It can be simple or haemorrhagic
- Imaging:
  - Simple: Isodense/isointense
  - Haemorrhagic: Depends on age of blood

*Cardiology* 1994; 85:255–258
Transmural lateral wall infarction, pericarditis, and MR

Transmural lateral wall infarction related late gadolinium enhancement (red arrows) and akinesia of the basal to mid LV lateral wall, with associated pericarditis related pericardial enhancement (yellow arrows) in 60 year old man in the distribution of the LCX territory. Mild Mitral regurgitation on 3ch ssfp clip.
Chronic Congestive Heart Failure

- Inability to the heart to pump the volume of blood due to insufficient or defective cardiac filling and/or impaired contraction and emptying
- It can affect the left (common) or right cardiac chambers or both
- Can occur after MI from either a single large healed infarct or multiple small infarcts
- It is often worsened by associated mitral regurgitation
- Imaging: Cardiomegaly, pulmonary venous hypertension, and pleural effusions, decreased ejection fraction

Chronic CCF in a patient post-MI

Eur J Heart Fail. 2013 Jun 12.
RadioGraphics 2013; 33:1383–1412
Conclusion

- MI is the leading cause of death and a main cause of morbidity in the United States
- Various complications may develop after MI, particularly when treatment is delayed or inadequate
- Recognizing typical imaging manifestations of these manifestations with adequate clinical correlation is essential for timely and accurate diagnosis as well as for guiding treatment
- It is imperative for the radiologist to be aware of and be confident in diagnosing the complications secondary to MI
## Summary

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<tr>
<th>Complication</th>
<th>Manifestations</th>
<th>Role of Imaging</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ischemic</strong></td>
<td>Angina, re-infarction, infarct extension</td>
<td>Cardiac gated MDCT and Cardiac MRI useful</td>
</tr>
<tr>
<td><strong>Mechanical</strong></td>
<td>Heart failure, cardiogenic shock, mitral valve dysfunction, aneurysms, cardiac rupture</td>
<td>Cardiac gated MDCT and Cardiac MRI useful</td>
</tr>
<tr>
<td><strong>Arrhythmic</strong></td>
<td>tach/brady-arrhythmias, intraventricular blocks</td>
<td>No role of imaging</td>
</tr>
<tr>
<td><strong>Embolic</strong></td>
<td>Central nervous system or peripheral embolization</td>
<td>CT /MRI useful to diagnose thromboembolism and associated complications</td>
</tr>
<tr>
<td><strong>Inflammatory</strong></td>
<td>Acute pericarditis or Dressler's syndrome</td>
<td>Imaging manifestations not so common</td>
</tr>
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Presenting author contact information

• Ameya J Baxi, MD
  Email id: baxi@uthscsa.edu