Non-traumatic acute cardiac emergencies: Complete radiological spectrum

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

Presenting author contact information
Ameya J Baxi, MD
Email id: baxi@uthscsa.edu

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Introduction

• Chest pain is always cause of concern especially if cardiac in origin
• The diagnosis of cardiac origin is initially established on clinical, laboratory and echocardiography evaluation
• Many a times, these may not be sufficient and evaluation using cardiac MRI and MDCT imaging is mandatory for reaching accurate diagnosis as many of the pathologies presenting with non-traumatic cardiac pain/emergency have distinct imaging appearance
• Accurate diagnosis is very important to optimizing proper treatment
• Categorizing these pathologies serves as a road map in patient care
Learning objectives

1. To discuss non-traumatic cardiac emergencies
2. To study the role of imaging in the diagnosis and evaluation of these conditions
3. To discuss imaging based differential diagnosis

- Cardiac arrest
- Myocardial infarction (MI)
- MI complications
- Cardiac and paracardiac aneurysms
- Pericardial tamponade
- Aortic root dissection
- Cardiac infections
- Arrhythmogenic Right Ventricle Dysplasia (ARVD)
- Hypertrophic Cardiomyopathy

In this exhibit, we discuss the characteristic multimodality imaging findings and differential diagnosis of common and uncommon non-traumatic cardiac emergencies. Increased awareness of such entities will contribute to optimized care of patients.
Cardiac arrest

• Cardiac arrest is not rare in clinical practice and is often reversible in most cases if treated at the earliest
• Imaging a patient having a cardiac arrest on the CT examination table is not common and limited radiology literature is available
• Altered hemodynamics resulting from cardiac failure causes stasis of blood in dependent organs of the body and injected intravenous contrast material, being heavier than blood, tends to accumulate in the dependent portions of the venous system seen as dependent contrast pooling and layering
• This in appropriate clinical scenario is a marker of the worsening clinical condition
• It is therefore very important to identify pertinent findings & act quickly
54-year-old male, found down. CT chest was done

MDCT showing dense contrast pooling (yellow arrows) in the SVC, IVC, hepatic veins, as well as in the right heart chambers with the formation of a blood-contrast level (red arrow). Also seen is hemopericardium (blue arrows) and tamponade. Thinning and irregularity of apex and lateral wall of left ventricle probably suggesting acute myocardial infarction and rupture (white arrows)

Autopsy: Rupture Acute Myocardial Infarction
38-year-old male came for dialysis catheter exchange

Chest radiograph showing right dialysis catheter

Follow up radiograph showing altered course of right dialysis catheter and patient developed chest pain and hypotension

CECT showing hemopericardium (yellow arrows) & mass effect on right cardiac chambers (red arrows) suggesting tamponade. Also seen is high density contrast in the pericardium (blue arrows) suggesting extrapericardial SVC perforation during a catheter exchange.
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
• Acute & chronic infarcts are managed differently, establishing the infarct age is crucial

RadioGraphics 2013; 33:1383–1412
Patterns of Myocardial Infarction

- Acute sub-endocardial MI
- Multi-vessel transmural infarct with apical thrombus
- Acute infarct with microvascular obstruction (no-reflow phenomenon seen as Non-enhancing hypo-intensity - arrow)
- Anterior wall
- Transmural infarct - septum
Complications of MI

- Various complications may develop after MI, particularly when treatment is delayed.
- 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.

- True aneurysm
- Pseudoaneurysm
- Acute and chronic thrombus
- Systemic thromboembolism
- Pericardial tamponade
- Pericarditis
- Myocardial and septal perforation
- Papillary muscle rupture
- Mitral regurgitation
- Cardiac failure

Radiological imaging plays no role in accessing arrhythmic complications like supraventricular tachyarrhythmias. 

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>
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<tbody>
<tr>
<td>Neck</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>Pathology</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>Location</td>
<td>Apex</td>
<td>Free lateral wall</td>
</tr>
<tr>
<td>Delayed enhancement</td>
<td>Usually not seen (Infarcted myocardium enhances)</td>
<td>Usually seen (Pericardium enhances)</td>
</tr>
<tr>
<td>Rupture risk</td>
<td>Low</td>
<td>High</td>
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</table>
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). Cardiac MRI shows abnormal thinning and bulging of the left ventricle apex and delayed transmural enhancement.
Left Ventricular Pseudoaneurysm

Post-Acute MI Pseodoaneurysm

LV Apex Pseudoaneurysm-Post MI
Acute and chronic thrombus

- Thrombus 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

RadioGraphics 2013; 33:1383–1412
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.
Post MI Thrombus (red arrow) Embolizing in Bilateral Legs (white arrows)

35 y/o male
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

Large basal septal muscular VSD (blue arrow) and left to right shunt (jet) (Yellow arrow) and basal inferior wall left ventricular pseudoaneurysm (red 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

Am Heart J. 2006; 151(2):316-322
Coronary artery aneurysm (CAA)

- Incidence of CAA during catheter angiography is <1%
- CAA is diagnosed when the vessel diameter is >1.5 times normal vessel
- Right CAA account for 50% of CAA
- In the adult, atherosclerosis is the most common etiology, followed by collagen vascular diseases
- In children, Kawasaki disease is the most common cause

Teaching Point: First described by Morgagni in 1761 in a patient with coronary artery dilatation and syphilitic aortitis. Munkner et al reported the first case of antemortem diagnosis of a coronary artery in 1958. CAA are classified as true aneurysms (composed of three layers) & pseudoaneurysms (composed of single/double layer due to disruption of external elastic membrane)
Coronary artery aneurysms in a 35 y/o male. Sequela of Kawasaki’s disease

Atherosclerotic coronary Artery Aneurysm

Teaching point: The aim of imaging is to evaluate (a) the distribution, (b) maximal diameter, (c) presence or absence of intraluminal thrombi, (d) number, (e) extension, and (f) associated complications such as myocardial infarction RadioGraphics 2009; 29:1939–1954
Saphenous Vein Graft Aneurysm (SVGA)

- SVG >1.5 times the expected diameter of the vessel
- True aneurysm: all layers of the vessel wall are involved
- False pseudoaneurysm: Disruption of the vessel wall
- False aneurysms are more common and develop earlier, usually at a suture line
- More common location is at an SVG graft to the LAD, followed to an SVG graft to the RCA
- Complications include rupture, thrombosis, embolization and infarction

50 y/o M, status post CABG presents with chest pain. New MI. Pseudoaneurysm SVG graft to RCA (red arrows)

SVG-RCA graft pseudoaneurysm

Teaching Point: True aneurysms typically arise > 5 years after bypass, occur in the body of the graft and are related to accelerated atherosclerosis. Pseudoaneurysms more commonly occur within 6 months after surgery & arise at either proximal or distal anastomotic sites.
Sinus of Valsalva (SoV) Aneurysm

- Can be congenital or acquired
- Congenital most often arise from right SoV
- Acquired, is usually secondary to AV endocarditis or Marfan disease.
- When large, may bulge, protruding out from the cardiac contour
- Congenital SoV aneurysms most commonly arise from right coronary SoV (R-SoV) and the non-coronary SoV (N-SoV) because of incomplete fusion (or weakness) of 2 halves of distal bulbar septum
- Congenital left SoV are rare

Previously healthy, 40 y.o. athletic male with one week of severe chest pain, shortness of breath, and orthopnea.
Mycotic Aneurysm

- True primary bacterial infection of the ascending aortic wall resulting in aneurysm formation is rare
- Believed to occur either after an episode of bacterial endocarditis or from an aortic jet lesion causing endothelial trauma
- Most common organisms include Staphylococcus aureus, Staphylococcus epidermidis, Salmonella & Streptococcus

Bacterial endocarditis and acquired L-SoV aneurysm axial (a) and sagittal (b) views at the level of SoV (arrows)

Aortic Pseudoaneurysm

- A false aneurysm/pseudoaneurysm involves enlargement of the aorta due to dilatation of only the outer layers of the vessel wall.
- They generally involve focal penetration of the intima and media, usually manifesting as a complication of trauma, surgery, infection, or atherosclerosis.
- The adventitia and perivascular connective tissue remain intact.

26-years-old male status post aortic valve and ascending aortic dacron graft 10 years back, now septic and presents with an perigraft fluid/abscess (red arrows) and root pseudoaneurysm (blue arrows)

Moktassi A, et al. JACR 2006;57:238
86-years-old male status post aortic valve replacement, now septic. MDCT shows perivalvular leak (red arrows) and aortic root pseudoaneurysm (blue arrows).

49-years-old male status post aortic valve replacement. MDCT shows aortic root pseudoaneurysm (blue arrows).
Post-operative collection and Pseudoaneurysm

29-years-old IV drug abuser male. Aortic valve and ascending aortic dacron graft, 10 years before. New onset chest pain and fever. MDCT shows pseudoaneurysm of the aortic root (red arrows) and collection likely abscess around the ascending aorta graft (blue arrows).

Potential imaging pitfalls in the postoperative period include mimickers of pathologic processes such as felt pledgets, graft folds, and nonabsorbable hemostatic agents.

Aortic Root Dissection

- Most common acute emergent condition of the aorta
- Spontaneous longitudinal separation of aortic intima & adventitia by circulating blood
- Classic history: acute chest pain radiating to the back
- Proximal dissections can be seen with AR in 40–50% of cases
- Type A dissections account for 60%–70% and require urgent surgical intervention to avoid extension into aortic root, pericardium, or coronary arteries
- Early diagnosis & treatment are essential for improving prognosis

The overall outcome is determined by the type and extent of dissection and presence of associated complications; therefore, evaluation of the entire aorta, branch vessels, and iliac and proximal femoral arteries is recommended to aid in treatment planning. Untreated, type A dissections are associated with a mortality rate of over 50% within 48 hours.
CT angiogram thoracic aorta axial, coronal, and sagittal images show Stanford type A dissection. The dissection flap (yellow arrow) involves aortic root (green arrow).

CCTA demonstrating Stanford type A dissection of aortic root (red arrows) extending into the ascending aorta (blue arrow). The patient also has hemopericardium (green arrows).
Cardiac Infection

- Infections can involve any of the three layers of the heart (endocardium, myocardium, pericardium)
- Virtually all classes of infectious organisms (virus, bacteria, fungi, parasites) can affect the heart.
- Since circulating blood passes through the heart continuously, the propensity of bloodborne infections to be carried to and from the heart is substantial
- Imaging plays a key role in the diagnosis and follow-up

*Feuchtner GM et al. JACC 2009;53:436*
Aortic Valve Infection

- Aortic Valve infection can be seen in non-drug abusers and in IV drug abusers
- Degenerative valvular disease now more common than rheumatic heart disease as a major risk factor
- 50% of all cases in the elderly (USA)
- Complications include CNS (stroke, abscess), visceral and MSK infarction/infection

CCTA demonstrate Aortic valve vegetations (arrow), in two different patient with infective endocarditis

Gahide G et al. AJR 2010;194:574
Infectious Myocarditis

• A wide variety of infectious and noninfectious causes are associated with myocarditis
• Viruses are the most common cause of myocarditis in the western world
• Enteroviruses and adenoviruses are the most common etiologic agents
• Viral myocarditis is a significant cause of dilated cardiomyopathy
• Pericardial effusion is common in patients with myocarditis (32%-57%)

Friedrich M et al. JACC 2009;53:1475

Asymmetrical areas of enhancement not following any vascular territory

Viral myocarditis seen as epicardial late enhancement on the lateral wall of LV
Arrhythmogenic right ventricular dysplasia

- Rare genetic disorder characterized by progressive loss of myocytes with fibrofatty replacement of RV and, less commonly, LV
- Patients may have ventricular arrhythmias and LBBB
- RV dilatation (> 42 mm) with dyskinetic bulges & sacculations (yellow arrow)
- Transmural fatty deposition
- Decreased RV free wall systolic thickness (red arrow)
- Regional or global RV contractile dysfunction
- Major and minor criteria

Circulation. 2010 April 6; 121(13): 1533–1541

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ARVD in a 38-year-old-male
Hypertrophic Cardiomyopathy

- Most common genetic CV disease
- Prevalence - 1:500; Annual mortality rate 1%
- Most common cause of sudden death in the young, including trained athletes
- Heterogeneous clinical, morphologic and genetic expression

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<tr>
<th>Risk Stratification Factor</th>
<th>Negative Prognostic Indicator</th>
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<tbody>
<tr>
<td>LV wall thickness</td>
<td>$\geq 30$ mm</td>
</tr>
<tr>
<td>Gradient across LVOT</td>
<td>$\geq 30$ mm Hg</td>
</tr>
<tr>
<td>Delayed enhancement</td>
<td>Represents fibrosis; presence and extent can be determined with MR imaging</td>
</tr>
<tr>
<td>LV ejection fraction</td>
<td>Decreased to $&lt; 50%$ (burned-out phase)</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>LV apical aneurysms</td>
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</tbody>
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Hypertrophic Cardiomyopathy

3 chamber views in systole (a) and diastole (b) in a 10-year-young boy showing “dumbbell” shaped LV cavity due to mid-ventricular and asymmetric mid-septal hypertrophy (red arrows). Also seen is apical thinning (yellow arrows) and significantly narrowed LV cavity size in systole and diastole. Patient also had systolic anterior motion of mitral valve leaflet and left ventricular outflow tract obstruction (not shown). The septal thickness was 2.6 cm and LV ejection fraction was 53%.

Courtesy Dr Dhanashree Rajderkar