Cardiac Aneurysms, Pseudoaneurysms and Diverticula
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Aneurysms and Pseudoaneurysms of the LV

- Progressive thinning of infarcted myocardium may lead to aneurysmal dilatation - true aneurysm.
- True aneurysms have a wide neck and contain all 3 layers of the cardiac wall.
- False aneurysms (Pseudo-aneurysms) represent a free wall rupture of the LV that is contained by the pericardium.

Chronic MI Changes: Pathology

Gross pathologic specimen demonstrating typical chronic MI changes and complications including myocardial thinning, aneurysm formation, scarring, and thrombus formation in the akinetic aneurysmal chamber.

Aneurysmal Changes in Chronic MI

- Distinguishing between true vs. pseudo LV-aneurysm is important because the clinical management and complication risks are different.
- True aneurysm:
  - The risk of rupture is low.
  - Medical management is usually adequate.
- Pseudo-aneurysms:
  - The risk of rupture is high and unpredictable.
  - Surgical resection and repair are required.
- LV free wall rupture is usually not contained and fatal.
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.

Aneurysmal Changes in Chronic MI

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.

Pseudo-aneurysm CT Imaging Findings

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.

LV Pseudoaneurysm

- 50 year old female with history of chest pain, hyperlipidemia and cigarette smoking
### Chronic MI Changes: Thrombus Formation

- Multiple factors lead to thrombus formation:
  - Stasis
  - Abnormal wall motion
  - Endothelial injury.
- Thrombus is readily seen in contrast-enhanced CT or MRI images as filling defects in MI-affected areas.
- Thrombus formation in an apical aneurysm dilatation with associated thinning and scarring.
- Other sequelae of chronic MI associated with thrombus formation include:
  - CHF
  - Dilated cardiomyopathies.
- Embolization of thrombus: <5%.

#### Large infarct >LV thrombus
- Thrombus can embolize leading to stroke, visceral infarction and death.

#### Traumatic pseudoaneurysm
- GSW to the chest with RV injury

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**Diagram and CCTA demonstrating:**

- Calcified LV aneurysm with thrombus formation (arrow).
- The infarcted myocardium may develop thinning, fatty replacement and calcification.
- The poorly contracting infarcted ventricle is also prone to the development of intraluminal thrombus.

**Post myocardial infarction aneurysm:**

- Cardiac MR shows an abnormal area of thinning and bulging on the posterior wall of the left ventricle (black arrows).
- Post-contrast injection delayed images confirm delayed enhancement of the infarcted myocardium (white arrow).
**True vs. False LV Aneurysm Differentiation with MR**

- n=22 patients
- Delayed enhancement of the pericardium in all patients with false aneurysm and in 17% of true aneurysms
- Sensitivity 100%
- Specificity 83%
- Accuracy 86%
- PPV 57%    NPV 100%

Koren E et al. Radiology 2005;236:65-70

**Congenital vs Traumatic pseudoaneurysm**

Three week old boy post surgical repair of congenital heart disease presents a posttraumatic (iatrogenic) pseudoaneurysm of the left ventricle (arrows).

**Ventricular Diverticulum**

- Congenital cardiac diverticula more commonly occur in the LV.
- It is a pouch or saclike projection from the cardiac lumen, and its wall contains all layers of normal ventricular myocardium.
- Prevalence: 0.04%-2.2%
- When large, may predispose to thrombus formation and peripheral embolization.
- Most are small (<1.5 cm).

**Left ventricular diverticula in two different patients. CCTA images illustrate the different shapes and locations of these diverticular formations my have (arrows).**

**Apical diverticulum of the LV. Contrast enhanced CT (A,B) and cardiac MR (C), demonstrate a focal saccular formation at the apex of the left ventricle (arrows).**

**Two diverticular formations within the septum in the same patient. Contrast enhanced CT shows projection of contrast at the basal (white arrows) and mid ventricular septum (black arrows).**

**Contrast enhanced CT shows projection of contrast at the basal (white arrows) and mid ventricular septum (black arrows).**
Interatrial septal aneurysm (IASA)

- Abnormal protrusion of the interatrial septum toward the right or left atrium.
- Prevalence: 2% - 5% in TEE series
- Probably congenital
- Mobile, thin wall (<2 mm), commonly involves the fossa ovalis.
- 90% protrude into the right atrium.
- Association with interatrial shunting is common (PFO or ASD)
- Other associations: MV prolapse (23%) and aneurysm of the sinus of Valsalva (5%).

Interventricular Septum Aneurysm

- Rare
- Congenital
- Membranous IV septum
- VSD (20%)
- Arrhythmia
- Complications
  - Rupture (intracardiac shunt)
  - Endocarditis
  - Thrombus, embolism
Coronary artery aneurysm (CAA)

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

Atherosclerotic aneurysms of the LAD (arrow) in a 45 y/o male patient with long history of cigarette smoking and cocaine abuse.

Kawasaki disease. Catheter angiography in two different infants demonstrates multiple coronary artery aneurysms.

Coronary artery aneurysms of the RCA and LAD in a 25 y/o female with past medical history of Kawasaki disease in her infancy.
Atherosclerotic Coronary Artery Aneurysm

63 y/o M, with PMH of Diabetes and CAD. Stent placed in RCA. Sepsis and fever. Cultures (+) for Staph Aureus. Autopsy: Infectious aneurysm RCA and purulent pericarditis.

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.
Sinus of Valsalva Aneurysm (SoVA)

- Localized weakness of the wall of the sinus of Valsalva, that leads to a focal bulging of the coronary sinus, that may rupture into an adjacent cardiac chamber, creating an aortocardiac fistula (34%).
- Originates from the right sinus in 70%, the noncoronary sinus (30%), and rarely from the left sinus.


Sinus of Valsalva Aneurysm

- Can be congenital, or acquired.
- Congenital, most often arise from the right SoV.
- Acquired, is usually secondary to AV endocarditis or Marfan disease.
- Since the aortic root is intracardiac, the aneurysm may not be visible.
- When large, may bulge, protruding out from the cardiac contour.

Pathophysiology of Congenital SoV Aneurysms

Typical SoV aneurysm progression

- Congenital left coronary SoV aneurysms (L-SoV) are uncommon because the left aortic valve cusp does not arise from the bulbar septum.

- Congenital SoV aneurysms most commonly arise from the right coronary SoV (R-SoV) and the non-coronary SoV (N-SoV) because of incomplete fusion (or weakness) of the 2 halves of the distal bulbar septum.

Congenital SoV Aneurysms Associations

- 50% of cases (R-SoV or N-SoV) associated with VSD
- Congenital SoVA with VSD are more commonly seen in Asians (30-60%)
- Aortic insufficiency in 20-30%
- Bicuspid aortic valve in 10%
- Congenital SoVA may also be associated with pulmonary stenosis, coarctation, and ASD

Ruptured SoV Aneurysm

- Rupture can occur spontaneously, following trauma, strenuous exertion or bacterial endocarditis.
- Rupture SOVA produces an intracardiac fistual with a left to right shunt.
- L-SoV aneurysm rupture may bleed into the pericardium.

56 yo female with fatal rupture of R-SoV into the RV.

13 yo female with fatal rupture of R-SoV aneurysm into RA.

56 yo female with fatal ruptured R-SoV aneurysm into RA followed by RA rupture.
Clinical Presentation of SoV Aneurysms

- Unruptured SoV aneurysms are asymptomatic unless large enough to cause:
  - obstruction of the RVOT
  - tricuspid stenosis and insufficiency due to prolapse of aneurysm through tricuspid valve
  - conduction abnormalities due to mass-effect on the bundle of His or its fascicles.
  - compression of the RCA (R-SoVA)

- Ruptured SoV aneurysms present with sudden onset of severe chest pain and dyspnea.

Ruptured SoVA

- When ruptured, produces an intracardiac fistula (L → R shunt).
- When a SoVA from the left coronary sinus ruptures, may bleed into the pericardium.
- Association with ventricular septal defect (VSD) (59%) and aortic regurgitation (25%) is common.

J Thorac Cardiovasc Surg 1990;99:288

Acute Presentation

- Usually due to sudden, large rupture/perforation.
- Symptoms include sudden, severe chest pain and dyspnea.
- Chest X-ray: enlarged cardiac silhouette, pulmonary edema, and CHF.
- Since the aortic root is intracardiac, the aneurysm may not be visible in plain radiographs.

Diagnostic Imaging

- Echocardiography with color Doppler is the current modality to confirm diagnosis.
- Diagnostic Criteria:
  - Root of aneurysm must be above the aortic annulus.
  - Aneurysm is saccular.
  - The size of the aorta above is normal.

Cardiac CTA, aortography, and magnetic resonance imaging may also be used.

CCTA volume rendered 3D reconstruction (a) of an unruptured SoV aneurysm (white arrow). Catheter angiography (b) demonstrating an unruptured N-SoV aneurysm. Magnetic resonance imaging (c) demonstrating an unruptured N-SoV aneurysm.
Rupture SoVA into the RV
Previously healthy, 40 y.o. athletic male with one week of severe chest pain, shortness of breath, and orthopnea.

Acquired Pathology Affecting the SoV
Bacterial endocarditis and acquired L-SoV aneurysm axial (a) and sagittal (b) views at the level of SoV.

Right Atrial Aneurysm
- Rare, probably congenital
- Intrapericardial
- Asymptomatic
- Arrhythmia (SupraVent Tach)
- ASD
- Complications
  - Thrombus formation
  - Stroke (paradoxical embolism)
  - Pulmonary embolism

Pre and Post-Surgical Repair
R-SoV ruptured aneurysm into RV
Aorta
**Left Atrial Diverticula and Accessory Appendages**

- Left atrial diverticula and left atrial accessory appendage are common anatomic variants.
- Prevalence: left atrial diverticula: 20%
  accessory appendage: 8%
- Most common location of left atrial diverticula is on the anterior and superior aspect of the left atrium.
- Most accessory appendages have a left lateral inferior location.


**Coronary Sinus Atresia with Aneurysm / Diverticula**

- 21 y/o F, super-obese (BMI 52.4) with Wolff-Parkinson-White syndrome
- Failed attempt to canalize the coronary sinus (twice)
- Two aneurysmal dilations of the coronary sinus
- Dx: Coronary sinus atresia
- In pts with CS atresia blood from the coronary sinus drain into the atria by way of enlarged thebesian veins

Mantini E et al. Circulation 1966;33:317
Summary

- Several types of aneurysms and pseudoaneurysms may arise from the different anatomic structures within the heart.
- Pathophysiology, morphology and clinical significance of these different anomalies vary greatly.
- Imaging plays a crucial role in the identification and appropriate management of these conditions.
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