Cardiac MRI: Application to Disease

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Outline
- Imaging planes
- Disease findings
  - Pulse sequences used for each indication
  - Pathophysiology being evaluated

Imaging planes

3 Plane localizer

Axial Haste

2 Chamber
Planned from axial images---usually HASTE
2---chamber view

Pseudo short axis
Planned from 2 chamber cine

Pseudo short axis

4 chamber
Planned from Pseudo SA and 2 Chamber

4 chamber

True short axis
LVOT
Left ventricular outflow tract
Planned off of pseudo SA

LVOT₁ and LVOT₂

Trans-aortic valve view
Planned off LVOT₁ and LVOT₂

Trans-aortic valve view

Disease

CMR for Myocardial Disease
- Coronary artery disease
  - Is there viable myocardium? Function and viability study
- Infiltrative myocardial disease
  - Sarcoidosis and Amyloidosis
- Myocarditis
  - Most called idiopathic, some cases with viral etiology
- Cardiomyopathy
  - Dilated CMQ, Hypertrophic CMQ, and ARVC
CMR for Myocardial Disease

- **CORONARY ARTERY DISEASE**
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Artery Disease Viability Study

- Usual clinical question: Would this patient benefit from CABG?
- Goal is to distinguish myocardium that has the ability to contract versus myocardium that is replaced by fibrosis or scar.
- Fibrosis and scar will not regain function after CABG

Coronary Artery Disease
The “Function and Viability Study”

- The basic protocol
  - 3 plane loc, axial haste
  - Cine bright blood (SSFP) - 2chamber, short axis (stack), and 4 chamber
  - *Delayed enhancement* - SA, 2C, 4C
    - Key sequence, looking for SCAR

Function and viability

- Delayed enhancement = scar!
- Scar!!

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Infiltrative myocardial disease
Sarcoidosis

- Cardiac sarcoidosis = presence of non-caseating granulomas in the pericardium, myocardium, or endocardium → leading to clinical sequelae
- Myocardial granulomas have been associated with cardiac arrhythmia and even sudden death
Infiltrative disease
Sarcoidosis

THE PROTOCOL
- 3-plane loc, axial haste
- Cine bright blood (SSFP), 2-chamber, SA stack, and 4-chamber
- Looking for focal areas of myocardial thinning, wall motion abnormality, aneurysm formation
- T2-weighted images
- Looking for myocardial edema and inflammation secondary to this granulomatous process
- Delayed enhancement in SA, 1C, and 4C
- Focal myocardial delayed enhancement = inflammation and/or granulomatous involvement
- Often in non-ischemic pattern (not confined to coronary artery distribution)
- Sometimes characterized as “patchy” or “mid-myocardial” in distribution

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Infiltrative disease
Amyloidosis

- Cardiac amyloidosis = amyloid proteins are abnormally deposited within the heart, especially in the myocardium
- Nobody famous 😎
- Imaging findings: left ventricular thickening, enlarged atria, pericardial effusion, diffuse subendocardial enhancement involving the RV and LV

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THE PROTOCOL: Essentially a "function and viability study"
- 3-plane loc, axial haste
- Cine bright blood (SSFP) 2-chamber, SA stack, and 4-chamber
  - Evaluate cardiac function - often mildly reduced
  - Evaluate myocardial wall thickness and mass
- Delayed enhancement - SA, 2-chamber, 4-chamber
  - Classic pattern = diffuse subendocardial enhancement
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Myocarditis

- Myocarditis = inflammation of the myocardium with necrosis of the adjacent myocytes; usually infectious
- Best diagnostic clue: Suddenly decreased systolic function + dilated heart + otherwise healthy person shortly after viral illness
- CMR = cardiomegally + pulmonary edema

Myocarditis

The protocol — long and exhausting for patient, tech, and radiologist:
- 2-plane tsk, axial base
- Cine bright blood (CEBP) 2–chamber, SA stack, and 4–chamber
- Chambers usually analyzed
- Evaluate cardiac function - usually reduced (evaluate extent of systolic dysfunction)
- T2–weighted images - usually in SA and 4–chamber
  - Looking for myocardial edema and inflammation
  - This is relative to skeletal muscle
  - Be sure to remind your radiologist to get plenty of skeletal muscle in the field of view - for both pre and post–contrast images, get the arm in there!
- Pre and post contrast T1–weighted images - usually SA and 4–chamber
  - Compare myocardial enhancement to skeletal muscle enhancement
  - Necrosis if possible
  - Evaluating amount of hypoxia and inflammation
- Delayed enhancement images - SA, 4C, 2C
  - Classic pattern in acute disease is subepicardial region (to distinguish from ischemic disease)
  - Looking for inflammation and scar
Myocarditis

Classic pattern could be described as patchy mid-myocardial DE. Usually regional involvement rather than global. Often involving inferolateral segments.

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CARDIOMYOPATHY

- DILATED CMO, Hypertrophic CMO, and ARVC

Cardiomyopathy

Dilated CMO

- Disease of heart muscle, LV becomes enlarged (dilated) and cannot pump blood as effectively to the body (heart failure)
- Can present as no symptoms → Terrible heart failure
- Usually idiopathic → need to test other causes
- Viral infections, genetic, diabetes, CAD, etc.

THE PROTOCOL

- 3–plane fast, axial haste
- Cine bright blood
- LA, RV, a chamber, and a chamber
- To evaluate function and chamber sizes
- Delayed enhancement
- To evaluate for infarct

Cardiomyopathy

Dilated CMO

- LV too big
- Function poor

Cardiomyopathy; Hypertrophic CMO

- Many types. Most popular is the genetic disorder, which can lead to sudden death, especially in young people
- hallmark: hypertrophic cardiomyopathy

THE PROTOCOL

- Cine bright blood
- LA, RV, a chamber, and a chamber
- To evaluate function and chamber sizes
- Delayed enhancement
- To evaluate for size or obstruction
**Cardiomyopathy**

**Hypertrophic CMO**

Areas of delayed enhancement = scar
Can be source of arrhythmia

**Cardiomyopathy**

**ARVD**

- Arrhythmogenic right ventricular dysplasia
- Cardiomyopathy characterized by fibrofatty infiltration of the RV leading to arrhythmia and possibly sudden cardiac death
- Another "right" study

**THE PROTOCOL**

1. Three-plane bic, axial haste
2. Cine bright blood (SSFP/SPGR)
   - 4-chamber and SA cine
   - Evaluate for areas of dyskinesis of the RV wall
3. RV and arterial MR — evaluate for fibrofatty infiltration of RV
4. Delayed enhancement — fibrofatty tissue

**ARVD**

Axial T1-weighted black blood spin-echo images show extensive transmural fatty replacement of the right ventricular myocardium

**Valve disease**

*Looking for stenosis or regurgitation*

- Aortic
- Mitral
- Tricuspid
- Pulmonic

**Aortic valve**

- Stenosis or regurgitation
- Morphology—cusp?
- Helpful sequences to obtain:
  - Cine bright blood—LVOT and Trans aortic valve
    - Look for the "jet" of aortic regurgitation
  - Phase contrast

**Aortic valve--- regurgitation**
Aortic valve--- regurgitation

Aortic valve--- stenosis

Often seen with bicuspid aortic valve

Look for post-stenotic aortic dilatation
Double IR axial image is nice

Mitral valve

- Stenosis or regurgitation
- Cine bright blood (SSFP/SPGR)
  - SA stack, 2 chamber, 4 chamber, and 3 chamber
- Phase contrast

You get the point! Same for tricuspid and pulmonic valves.

Mitral valve--- regurgitation

Pericardial disease

- Pericarditis
- Pericardial constriction
- Pericardial fluid
  - Simple, blood, or malignant
Pericarditis

- Inflammation usually due to infection
- Double IR
  - Nice in 4-chamber and/or SA views
  - Evaluate for pericardial thickening
- Delayed enhancement
  - Look for inflammation

Pericardial constriction

- Usually a thickened, fibrotic pericardium that forms a non-compliant shell around the heart, which prevents the heart from expanding when blood enters
- In US, #1 and #2 causes are radiation therapy and surgery
- Worldwide, #1 cause is infection = TB
- Key sequences:
  - Double IR--- to evaluate for pericardial thickening
  - High temporal resolution SA cine--- to look for septal bounce
  - Delayed enhancement--- to look for pericardial inflammation
  - THOSE TAG LINES!! - to evaluate the motion of layers of pericardium
Pericardial fluid

Take home points

- Key imaging planes and pulse sequences for specific disease pathology
- Cine bright blood is a workhorse
- Delayed enhancement for inflammation, infiltration, or infarct
- Phase contrast for flow quantification
- T1, T2, and contrast images for characterization

You

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