Newly Diagnosed Heart Failure patient:
When to Order an MRI and Why

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Disclosures

- None
Prevalence of Heart Failure by Age/Gender

NHANES: 2003-2006)
Potential Causes Of Heart Failure

- Ischemic heart disease: 1082
- Hypertension: 463
- Unknown: 382
- VHD: 340
- Alcohol: 297
- Diabetes: 275
- System disease: 270
- Dilated CMP: 201
- Infection: 93
- Hypertrophic CMP: 93
- Cor pulmonale: 40
- Cytotoxic treatment: 36
- Pericardial disease: 21
- Myocarditis: 17
- Mediastinal irradiation: 15
- Other CMP: 7

What do we do as clinicians?

Listen
Examine

“Precision in diagnosis precedes rational therapy.”

C. F. Wooley, MD

Establish diagnosis, prognosis
Deliver optimal prevention, treatment
Develop new therapies based on better understanding of disease mechanisms
What Can MRI Offer

- Assessment of biventricular function
- Assessment of myocardial scar
  - Ischemic
  - Non-ischemic
  - Infiltrative
- Assistance with prognostication
  - Response to revascularization
  - Response to cardiac resynchronization therapy
  - Risk for SCD
DME Forms the Cornerstone

Differentiation of Heart Failure Related to Dilated Cardiomyopathy and Coronary Artery Disease Using Gadolinium-Enhanced Cardiovascular Magnetic Resonance

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Two Phases of Myocardial Enhancement

- **Normal Myocardium**
- **Infarcted Myocardium**
- **Ischemic Myocardium**

- **First-Pass** (within 1 min)
- **Delayed Enhancement** (> 5 min)

Injection timing and temporal analysis is illustrated with images and curves showing myocardial enhancement phases.
Delayed HE Mechanism: Higher [Gd] = shorter T1

Normal Myocardium
- Intact cell membranes
- [Gd] = Low

Acute MI
- Ruptured cell membranes
- [Gd] = High

Scar
- Collagen matrix
- [Gd] = High
Delayed Enhancement Patterns

- Classic Coronary Distribution

A  Subendocardial Infarct

B  Transmural Infarct

Karamitsos JACC 2009
Delayed Enhancement Patterns: Non-Coronary Distribution

A Mid-wall HE
- Idiopathic Dilated Cardiomyopathy
- Myocarditis
- Hypertrophic Cardiomyopathy
- Right ventricular pressure overload (e.g., congenital heart disease, pulmonary HTN)
- Sarcoidosis
- Myocarditis
- Anderson-Fabry
- Chagas Disease

B Epicardial HE
- Sarcoidosis, Myocarditis, Anderson-Fabry, Chagas Disease

C Global Endocardial HE
- Amyloidosis, Systemic Sclerosis, Post cardiac transplantation
DME: LAD-territory infarct scar
Mid-Myocardial Hyperenhancement
Infiltrative Cardiomyopathies

- EMB subject to sampling error
- CMR ‘samples’ the entire myocardium
- Sarcoidosis
- Amyloidosis
- Hemochromatosis
- Chagas disease
- Gaucher’s disease, Anderson-Fabry disease, etc.
Clinical Patient

- 85 y/o male with shortness of breath, falls
- PMH: “enlarged heart”
- ECG: atrial fibrillation
- 68”, 124 lb; SCr 2.0
- Questions:
  - Ischemia?
  - Structural heart disease?
  - Cardiomyopathy?

Stress CMR with real-time cine and perfusion
Cardiac Amyloid

- Congo red
- Polarized light with congo red

Images:
- DME TI Scout
- DME TI 70msec
- DME TI 200msec
Myocardial Amyloidosis

- Cardiac involvement is cause of death in ½ of patients with AL amyloidosis
- 30 patients with tissue diagnosis of cardiac amyloid (EMB in 29, autopsy in 1) underwent CMR
- 97% concordance in diagnosis of cardiac amyloidosis

- DME distribution:
  - Subendocardial in 42%
  - Midwall in 28%
  - Subepicardial in 18%
- Characteristic T1 properties of myocardium, blood
Myocarditis

- Clinical presentation ranges from nonspecific systemic symptoms to fulminant hemodynamic collapse
- Autopsy prevalence of 8.6 – 12% of SCD in young adults
- While EMB typically involves 4-6 samples, postmortem study suggests >17 samples needed to make diagnosis >80% of the time\(^1\)

CMR in Myocarditis

- 32 patients with clinically-suspected myocarditis based on:
  - Flu-like symptoms w/i prior 8 weeks
  - Fatigue/malaise, CP, SOB, or tachycardia
  - AV block, ST depression, VT
- Baseline CMR, LV EMB in regions of enhancement
- 3 month follow-up

Contrast enhancement in 28 (88%), corresponding foci of myocyte damage and macrophage infiltration

Predominantly lateral wall, epicardial quartile of myocardium

Only 14 had elevated troponin

Two patients diagnosed with HCM

Further investigations of utility of CMR in predicting myocarditis outcome
Myocarditis: Giant Cell

DME with extensive epicardial hyperenhancement
Myocarditis

CMR Cine Images  Markedly abnormal DME-CMR

Small focus of mononuclear cells
Clinical Case

- 16 year-old asymptomatic basketball player
- ROS: no syncope, palpitations, DOE, etc.
- PMH: negative
- FH: unremarkable
Physical Examination

- Height 182 cm, weight 71 kg
- BP 118/54, HR 45-60
- Symmetric pulses
- II/VI SEM at LUSB, no positional change
- Rest of PE unremarkable
- Normal echocardiogram (‘1cm LV walls’)
- Because of abnormal ECG, patient referred for cardiac magnetic resonance
- CMR exam included:
  - 3D cine
  - Post-gad DME for scar/infiltrate
  - Non-contrast MRA for coronary artery origins/ prox course and aorta
Validation of Histopathology of the DME regions

- HCM heart explanted at the time of cardiac transplantation.
- LVH, Macroscopic scaring
- Myofibril disarray /collagen scar
- DME extent of enhancement correlated with extent of collagen.

Moon et al. JACC 2004; 43:2260-4
- Certain patterns of DME are associated with higher risk of sudden cardiac death.
  - Diffuse vs. confluent
  - Location

- Association of DME with unstable electrical substrate

Maron Circulation 2010 vol 74: 2271-2282
**Table 2.** Associations Between Pattern of Hyperenhancement and Clinical Phenotype

<table>
<thead>
<tr>
<th>Type of Hyperenhancement</th>
<th>n (%)</th>
<th>Clinical Phenotype</th>
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<tbody>
<tr>
<td>Trans-septal</td>
<td>4 (7%)</td>
<td>Young, gross asymmetric LVH; extensive diffuse hyperenhancement; high risk of sudden death</td>
</tr>
<tr>
<td>RV septal</td>
<td>4 (7%)</td>
<td>Extensive RV surface of septal hyperenhancement; strong family history of sudden death</td>
</tr>
<tr>
<td>Ventricular junction</td>
<td>12 (23%)</td>
<td>Moderate symmetrical LVH; limited hyperenhancement at RV insertion points; lower risk of sudden death</td>
</tr>
<tr>
<td>Multi-focal</td>
<td>9 (17%)</td>
<td>Large focal areas of hyperenhancement; LBBB if basal septum; associated with progressive disease</td>
</tr>
<tr>
<td>Subendocardial</td>
<td>2 (4%)</td>
<td>Like infarcts, but not IHD, in these patients</td>
</tr>
<tr>
<td>Other</td>
<td>11 (21%)</td>
<td>Other patterns or trivial hyperenhancement</td>
</tr>
<tr>
<td>None</td>
<td>11 (21%)</td>
<td>Typically young and at low risk</td>
</tr>
</tbody>
</table>

IHD = ischemic heart disease; LBBB = left bundle branch block; LVH = left ventricular hypertrophy; RV = right ventricular.
Clinical Case

- 36 y/o African-American male with palpitations, near-syncope
- PMH: sarcoidosis
- PE, echocardiogram unremarkable

![ECG Image]

- CMR
42 y/o male with atrial fibrillation refractory to drug therapy
FH: no known cardiovascular disease
PE: unremarkable
Echocardiogram: low-normal EF
CMR exam to delineate pulmonary veins pre-ablation
Dx: arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC/D)

Change in management:
- RFA plus ICD placement
- Screening of family members
Predicting Response to Therapies
Predicting Resynchronization Therapy Response

- 40 ICMP patients referred for CRT
  - EF ≤35%, LBBB, QRS >120ms
- Baseline CMR: scar location, transmurality
- Baseline, post-implant TDI
  - Y/N dyssynchrony: septal-to-lateral wall delay ≥65ms
- Baseline, 6-month clinical assessment incl. NYHA class, 6-minute walk, QOL score

Bleeker GB et al. Circ 2006; 113:969-76.
Responder:
≥1 NYHA Class improvement or ≥25% increase in 6-minute walk

95% response rate if severe dyssynchrony without PL scar

Transmural posterolateral scar in 14:
Response rate 14% compared to 81% in patients without posterolateral scar regardless of dyssynchrony
Summary

- CMR readily distinguishes ischemic from non-ischemic cardiomyopathy
- CMR provides diagnostic as well as prognostic information; tissue diagnosis in some disorders
- CMR can predict response to revascularization, medical, resynchronization therapies