CT for Myocardial Characterization of Cardiomyopathy

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Etiology, treatment, prognosis, & imaging

- Etiology
  - Unknown
  - Genetic & Familial
  - Secondary

- Treatment
  - Supportive care
  - Transplantation

- Prognosis
  - Progressive heart failure
  - Sudden cardiac death

- Imaging role:
  - Structural and functional characterization
  - Identifying etiology which is treatable or controllable.
  - Predicting prognosis

- Imaging modality
  - Echocardiography
  - MRI
  - CT
Imaging methods & protocol

- Structural change
  - Dilatation of ventricle
  - Hypertrophy of myocardium
  - Regional abnormality
  - Enlargement of atria or pulmonary veins

- Functional change
  - Systolic dysfunction
  - Diastolic dysfunction

- Tissue characterization
  - Enhancement
  - Perfusion
  - T1/T2/T2* value
  - ECV
  - Calcification

- MRI
  - Morphology and function
  - Perfusion
  - Late gadolinium enhancement
  - T1 mapping & ECV fraction
  - Coronary angiography (optional)

- CT
  - Coronary angiography
  - Delayed enhancement

- CT & MRI
Delayed enhancement

- Necrosis
- Infiltration
- Fibrosis

Kim RJ
Cardiovascular MRI and MRA
Etiology by enhancement pattern

**Mesocardial**
- HCMP
- DCMP
- Pulmonary hypertension

**Subendocardial**
- Infarction
- Amyloid
- Hypereosinophilic syndrome
- Histiocytoid cardiomyopathy
- Cardiac transplant

**Subepicardial**
- Myocarditis (most common)
- Sarcoid

**Transmural**
- Infarction (most common)
- Myocarditis, severe
- Sarcoid, chronic

Areas of abnormal contrast enhancement can also be used as guide for endomyocardial biopsy to increase diagnostic yield

RadioGraphics 2009;29:89-103
Prognostic implication of LGE

- A certain amount of irreversible myocardial damage may cause adverse **ventricular remodeling**.
  → ventricular dilatation and dysfunction
- Myocardial areas affected by irreversible damage will **not respond to heart failure drug therapy**
- Irreversible myocardial damage may trigger **arrhythmic events** causing sudden cardiac death.
- The extent of total scar (cutoff >5% LV mass) might be the most important parameter in patients with ICM and NICM
- The number of separate scarred areas – independent predictor of outcomes with hazard ratio of 1.7
A. All-cause mortality

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Log-rank P < .001

B. Cardiovascular mortality or transportation

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Log-rank P = .998

C. Sudden cardiac death or aborted sudden cardiac death

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D. Heart failure death, hospitalization, or transplantation

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Diffuse fibrosis?

All or None

Diffuse fibrosis

Mild fibrosis

Quantification
T1 mapping & ECV

- Absolute value (time)
- Quantification of tissue value
- T1 mapping
  - pre-contrast enhancement (native)
  - post-contrast enhancement
- Extracellular volume fraction (ECV)
  - $\Delta R1_{myo} = \frac{1}{T1_{myo\text{–post}}} - \frac{1}{T1_{myo\text{–pre}}}$
  - $\Delta R1_{blood} = \frac{1}{T1_{blood\text{–post}}} - \frac{1}{T1_{blood\text{–pre}}}$
  - $ECV = \Delta R1_{myo} / \Delta R1_{blood} \times (100 - HCT)$
Standardization and consideration of protocol

- Native T1 mapping
  - Heart rate
  - 1.5T vs. 3.0T
    - 3.0T: T1 1315 ± 39 ms
    - T1 value: 1.5T < 3T

- Post-contrast T1 mapping
  - Contrast agent, dose
  - Infusion vs. bolus: similar
  - Waiting time: 8.5 - 23.5 min
  - Cardiac phase: systolic < diastolic
  - Region: nonseptal ≠ septum

*JCMR 2011;13:75*
*JCMR 2012;14:17*
ECV predicts outcome

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mortality Outcome (n=39)</th>
<th>Death, Cardiac Transplant, or Left Ventricular Assist Device Outcome (n=43)</th>
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</thead>
<tbody>
<tr>
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<td>Hazard Ratio (95% CI)</td>
<td>Hazard Ratio (95% CI)</td>
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<tr>
<td>Extracellular volume fraction (ECV) (for every 3% increase)</td>
<td>1.55 (1.27-1.88)</td>
<td>1.48 (1.23-1.78)</td>
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<td>Left ventricular ejection fraction (for every 5% decrease)</td>
<td>1.23 (1.11-1.37)</td>
<td>1.29 (1.16-1.43)</td>
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<td>Myocardial infarction size tertile</td>
<td>1.13 (0.81-1.57)</td>
<td>1.15 (0.86-1.55)</td>
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<td>Age (for every 10 year increase)</td>
<td>1.29 (1.02-1.64)</td>
<td>1.17 (0.94-1.46)</td>
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ECV in myocardium without LGE

**Figure 3** Extracellular volume fraction of infarcted myocardium (Infarct LGE) and regions of Atypical LGE were significantly higher than ‘normal appearing’ myocardium.

**Figure 4** Extracellular volume fraction of myocardium increases as a function of age. Directionally, these findings are consistent with an age-related increase in diffuse fibrosis.

**Figure 5** Extracellular volume fraction of ‘normal appearing’ myocardium is inversely related to ejection fraction in patients with myocardial infarction. These findings are consistent with adverse post-infarct remodelling in myocardium remote from infarction.

Eur Heart J 2012;33:1268
Why not CT?

Circulation 1979;60:284

Circulation 2006;113:394-404
Advantage of CT delayed enhancement

- Delayed imaging in conjunction with coronary artery angiography
- Isotropic 3D volume data
- No need an adjustment like TI in MRI
Application to cardiomyopathy

Myocarditis

HCMP

J Am Coll Cardiol 2012;60

Eur Radiol 2013;23:1034-1043 (ASCI square)
Clinical application of delayed CT

- Nonischemic cardiomyopathy
- Differentiation from ischemic cardiomyopathy
- Pattern diagnosis for specific etiology
- Volume fraction of hyper-enhanced myocardium for risk stratification
Dual-energy delayed CT for quantifying DIFFUSE fibrosis

- Rabbit model with myocardial fibrosis induced by Doxorubicin
  - Doxorubicin induced cardiomyopathy (1.0mg/kg twice a week for 6 weeks)

- Imaging
  - Dual-energy CT (2\textsuperscript{nd} generation dual-source)
  - MRI
    - T1-mapping MRI (MOLLI, 3T-unit)
    - LGE MRI

Baseline EF= 53.5%
16 weeks EF= 36.7%

JACC CVI: in revision
Extracellular volume fraction vs. Collagen volume fraction
ECV measurement with dual energy CT: Advantages

- Might be more accurate
  - measurement error ↓
- Pre-contrast CT is not required.
- No misregistration between two sets of images (pre- & post- in MRI)
- Isotropic whole heart volume data
F/75 HCMP, Apical LV

MR ECV (Hct 33.8)
Septal: 33.8%
Inferior: 42.6%

CT ECV (Hct 32.5)
Septal: 31.5%
Inferior: 42.8%

Radiology: in revision
Per-patient analysis

MRI ECV = 35.51 ± 8.61%
CT ECV = 35.98 ± 8.30%
P = 0.098
ICC = 0.969

Per-segment analysis

23 patients with cardiomyopathy

MRI ECV = 35.51 ± 10.50%
CT ECV = 35.98 ± 10.62%
P = 0.105
ICC = 0.940
ECV measurement with dual energy CT: Clinical application

- In addition to differential diagnosis of ischemic and nonischemic cardiomyopathies in combination with CTA and CT MDE
  - Detecting diffuse fibrosis
  - Quantifying diffuse fibrosis
Assessment of myocardial delayed enhancement is feasible and diagnostic with dual-energy cardiac CT in CMP and shows a good correlation with MRI in volume measurement.

Myocardial ECV with dual-energy cardiac CT in cardiomyopathy is feasible to detect and quantify diffuse myocardial fibrosis.

ECV calculated with dual-energy cardiac CT showed excellent agreement with that with cardiac MRI, suggesting the potential of myocardial tissue characterization with cardiac CT.
THANK YOU