EAE Teaching Course

Magnetic Resonance Imaging

Competitive or Complementary?

Sofia, Bulgaria, 5-7 April 2012

F.E. Rademakers
Complementary?

Of Course
A Tale of Coronary Artery Disease and Myocardial Infarction

Elizabeth G. Nabel, M.D., and Eugene Braunwald, M.D.

Figure 1. Decline in Deaths from Cardiovascular Disease in Relation to Scientific Advances.

The timeline shows the steady decline in cardiovascular deaths over the late 20th and early 21st centuries, along with major advances in cardiovascular science and medicine. ALLHAT denotes Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial, CASS Coronary Artery Surgery Study, GISSI Italian Group for the Study of Streptokinase in Myocardial Infarction, HMG-CoA 1-hydroxy-3-methylglutaryl coenzyme A, ISIS-2 Second International Study of Infarct Survival, MI myocardial infarction, NCEP National Cholesterol Education Program, NHBPEP National High Blood Pressure Education Program, PCI percutaneous coronary intervention, SAVE Survival and Ventricular Enlargement, and TIMI 1 Thrombolysis in Myocardial Infarction 1.
Clinical relevance

• Treatment is warranted
  – To alleviate symptoms
  – To improve prognosis

  – The balance between these is governed by
    • Circumstances
    • Age
    • Expectations
    • ...

• Balance between risk and benefit
• Cost – benefit / effectiveness / social acceptance
Determinants of vulnerability

- Inflammation and repair
  - Cap thickness
  - Core size

Vulnerable plaque
- Fibrous cap
- Lipid-rich core
- Cap disruption
  - Vulnerability
  - Triggers

Unstable coronary artery disease
- Determinants of thrombosis
  - Thrombogenic substrates
  - Degree of stenosis
  - Thrombotic/lytic equilibrium

Lysis/repair
- Thrombolysis
- Lysis/remodelling
- Recanalization

Downstream embolization
A Tale of Coronary Artery Disease and Myocardial Infarction

Elizabeth G. Nabel, M.D., and Eugene Braunwald, M.D.
Acute Myocardial Infarctions Evolve Most Frequently From Plaques With Mild to Moderate Obstruction

Coronary Lesions on Pathology

Prevalence

- **Korean war** 77%

- **Vietnam** 45%

- **Velican** 33%

- **Arbustini** 42%
  – **Thrombosis** 8%

- **Baroldi (40yrs)** 74%

- **Berenson**
  – **Children** 8%
  – **Middle-age** 69%

- **Angelini** 20%

- **McGill** 20%
Figure 1. Prevalence of AHA grades in LAD by sex and 5-year age group. Women, □; men, ■; error bar = SE.
Figure 2. Prevalence of atherosclerotic stenosis ≥40% in LAD by sex and 5-year age group. Women, □; men, ■; error bar=SE.
Main Causes of Coronary Thrombosis

Rupture (65%)

Erosion (30%)

Pathologic Intimal Thickening (50%)

Fibroatheroma (50%)

Frequency of Coronary Thrombi in Culprit Lesions by Decade in Men Dying Sudden Coronary Death

Percent

Age Range in Years

30-39
40-49
50-59
60-69

Acute Thrombus
Plaque Rupture
Plaque Erosion
Stable Plaque

p=0.05

R. Virmani
Rupture, Stenosis, Cap Thickness and Necrotic Core

Arterioscler Thromb Vasc Biol. 2010;30:1282-1292
**Figure 1** Ruptured fibrous cap-acute coronary syndrome plaque. Optical coherence tomography (A1 and A2), coronary angiography (B), intravascular ultrasound (C), angiography (D), volume rendering (E), curved multiplanar reformation (F1), and the cross-sectional images (F2) were obtained in a culprit lesion with plaque rupture in a 67-year-old male presenting with acute coronary syndrome. Optical coherence tomography revealed fibrous cap disruption (white arrow in A1) and thrombus formation in adjacent slices (white arrow in A2). Coronary angiography (B) showed yellow plaque and red thrombus formation through the blue coronary angiography guide catheter. Intravascular ultrasound (C) indicates two focal calcium deposits <90°. Angiography (D) and volume-rendered computed tomographic images (E) disclose a significant stenosis in the middle segment of the left anterior descending coronary artery (yellow arrows in D and E). Curved multiplanar reformation computed tomographic images (F1) reveal positive remodelling associated with focal calcium deposits (yellow arrows in F1). Curved multiplanar reformation and the cross-sectional images (F2) display the presence of soft plaque with an attenuation of <30 HU (red arrow in F1 and F2).
### Table 1  Baseline clinical and angiographic characteristics in 57 lesions for 57 patients with ruptured fibrous cap-, intact fibrous cap-acute coronary syndromes, and stable angina

<table>
<thead>
<tr>
<th></th>
<th>RFC-ACS</th>
<th>IFC-ACS</th>
<th>Stable angina</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Patients (n)</strong></td>
<td>25</td>
<td>10</td>
<td>22</td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>63.7 ± 10.1</td>
<td>61.5 ± 8.2</td>
<td>62.6 ± 8.2</td>
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<tr>
<td>Male (n, %)</td>
<td>25 (100)</td>
<td>8 (80)</td>
<td>22 (100)</td>
<td>0.007</td>
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<tr>
<td>Clinical presentation (n, %)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NSTEMI</td>
<td>15 (60)</td>
<td>4 (40)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>UAP</td>
<td>10 (40)</td>
<td>6 (60)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Stable angina pectoris</td>
<td>—</td>
<td>—</td>
<td>22 (100)</td>
<td></td>
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<tr>
<td>Diabetes (n, %)</td>
<td>8 (32)</td>
<td>3 (30)</td>
<td>8 (36)</td>
<td>0.922</td>
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<tr>
<td>Hypertension (%)</td>
<td>12 (48)</td>
<td>6 (60)</td>
<td>13 (59)</td>
<td>0.692</td>
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<tr>
<td>Hypercholesterolaemia (%)</td>
<td>12 (48)</td>
<td>6 (60)</td>
<td>10 (45)</td>
<td>0.739</td>
</tr>
</tbody>
</table>

**Smokers (n, %)**

<table>
<thead>
<tr>
<th></th>
<th>RFC-ACS</th>
<th>IFC-ACS</th>
<th>Stable angina</th>
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</thead>
<tbody>
<tr>
<td>Non-smoker</td>
<td>10 (38)</td>
<td>1 (9)</td>
<td>7 (35)</td>
</tr>
<tr>
<td>Smoker</td>
<td>15 (62)</td>
<td>9 (90)</td>
<td>15 (65)</td>
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</table>

<table>
<thead>
<tr>
<th></th>
<th>RFC-ACS</th>
<th>IFC-ACS</th>
<th>Stable angina</th>
</tr>
</thead>
<tbody>
<tr>
<td>AVG/LBDL</td>
<td>2.55 ± 0.52</td>
<td>2.46 ± 0.46</td>
<td>2.64 ± 0.58</td>
</tr>
<tr>
<td></td>
<td>0.94 ± 0.20</td>
<td>1.09 ± 0.22</td>
<td>0.96 ± 0.25</td>
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</table>

**QCA**

<table>
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<th>RFC-ACS</th>
<th>IFC-ACS</th>
<th>Stable angina</th>
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</thead>
<tbody>
<tr>
<td>RD pre (mm)</td>
<td>2.55 ± 0.52</td>
<td>2.46 ± 0.46</td>
<td>2.64 ± 0.58</td>
</tr>
<tr>
<td>MLD pre (mm)</td>
<td>0.94 ± 0.20</td>
<td>1.09 ± 0.22</td>
<td>0.96 ± 0.25</td>
</tr>
<tr>
<td>Lesion length (mm)</td>
<td>20.7 ± 7.0</td>
<td>22.5 ± 7.2</td>
<td>22.9 ± 9.4</td>
</tr>
</tbody>
</table>

**Non-culprit lesions in non-culprit vessel assessed by CT angiography**

<table>
<thead>
<tr>
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<th>RFC-ACS</th>
<th>IFC-ACS</th>
<th>Stable angina</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-culprit lesions (n)</td>
<td>19</td>
<td>8</td>
<td>12</td>
</tr>
<tr>
<td>Average lesion severity (%)</td>
<td>42 ± 19</td>
<td>32 ± 12</td>
<td>46 ± 20</td>
</tr>
<tr>
<td>NCP ≤ 30 HU (%)</td>
<td>7 (37)</td>
<td>2 (25)</td>
<td>1 (8)</td>
</tr>
<tr>
<td>Spotty calcification (n, %)</td>
<td>5 (26)</td>
<td>2 (25)</td>
<td>2 (16)</td>
</tr>
<tr>
<td>Positive remodelling (n, %)</td>
<td>10 (53)</td>
<td>1 (13)</td>
<td>2 (16)</td>
</tr>
</tbody>
</table>
Index Lesions for Patients with MI / ACS / PCI

A. Percentage of patients with each index lesion type

Total number of patients = 362

- Lesions originally < 50% DS: 29%
- Lesions originally ≥ 50% DS: 52%
- Restenosis lesions: 19%

B. Percentage of index lesions of each lesion type

Total number of lesions = 4282

- Lesions originally < 50% DS: 3%
- Lesions originally ≥ 50% DS: 21%
- Restenosis lesions: 68.193

Figure 3. Categories of index lesions in patients with myocardial infarction, acute coronary syndrome, and percutaneous coronary intervention. A, Percentage of patients presenting with a given type of index lesion. B, Type as a percentage of all lesions of that type. DS indicates diameter stenosis.

Index Lesions for Patients with Symptom Progression Without MI / ACS / PCI

A. Percentage of patients with each index lesion type

Total number of patients = 137

- Lesions originally < 50% DS: 36%
- Lesions originally ≥ 50% DS: 39%
- Restenosis lesions: 25%

B. Percentage of index lesions of each lesion type

Total number of lesions = 1670

- Lesions originally < 50% DS: 4%
- Lesions originally ≥ 50% DS: 20%
- Restenosis lesions: 34/138

Figure 4. Types of index lesions in patients with symptom progression without myocardial infarction, acute coronary syndrome, and percutaneous coronary intervention during follow-up. A, Percentage of patients presenting with a given type of index lesion. B, Type as a percentage of all lesions of that type. DS indicates diameter stenosis.
<table>
<thead>
<tr>
<th>Baseline Features</th>
<th>MI or ACS (n=130)</th>
<th>PCI-Only (n=222)</th>
<th>Symptom Progression Only (n=137)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>OMT + PCI</td>
<td>47 (36)</td>
<td>70 (32)</td>
<td>88 (64)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>OMT only</td>
<td>83 (64)</td>
<td>152 (68)</td>
<td>49 (36)</td>
<td></td>
</tr>
<tr>
<td>CABG</td>
<td>26 (20)</td>
<td>34 (15)</td>
<td>11 (8)</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td>No. nonrevascularized lesions originally ≥50% DS per</td>
<td>2.7±0.2</td>
<td>2.4±0.1</td>
<td>1.9±0.1</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>patient</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average %DS of nonrevascularized lesions originally</td>
<td>67±1</td>
<td>70±1</td>
<td>66±1</td>
<td>&lt;0.04</td>
</tr>
<tr>
<td>≥50% DS per patient</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average original %DS per patient</td>
<td>36±1</td>
<td>35±1</td>
<td>33±1</td>
<td>&lt;0.02</td>
</tr>
</tbody>
</table>

Data are presented as mean±SE or n (%). MI indicates myocardial infarction; ACS, acute coronary syndrome; PCI, percutaneous coronary intervention; OMT, optimal medical therapy; CABG, coronary artery bypass graft; DS, diameter stenosis.

**WHAT IS KNOWN**

- Acute coronary syndromes are believed to arise commonly from lesions <50% diameter stenosis.
- Prognosis in patients with coronary disease is related to overall atherosclerosis burden.

**WHAT THE STUDY ADDS**

- In patients treated with optimal medical therapy, symptoms that developed during follow-up were attributed to progression of lesions originally <50% diameter stenosis in one third of patients.
- Lesions <50% that progressed represented <4% of all such lesions.
- In patients treated with optimal medical therapy and percutaneous coronary intervention, lesions originally ≥50% diameter stenosis were responsible for new symptoms in 20% of patients and 68% in optimal medical therapy-only patients.

- The number of nonrevascularized lesions originally ≥50% diameter stenosis was the only angiographic predictor of myocardial infarction or acute coronary syndrome.
Prognosis

• Ischemia
  – Presence
  – Extent
  – + knowledge of viability

• Prevention of ACS
  – Unstable plaque
  – Evolution towards Heart Failure

• Arrhythmia risk
  – Borderzone of necrosis with mixture of normal cells, fibrosis, persistent ischemia
What can be of help?

- Risk factors
- Symptoms
- Calcium score
- Stenosis severity
- Ischemia
  - Can guide therapy
Stenosis classification

Box-and-whisker plot showing the fractional flow reserve (FFR) values of all lesions in the categories of 50% to 70%, 71% to 90%, and 91% to 99% diameter stenosis. The red horizontal line corresponds to the FFR cut-off value for myocardial ischemia (FFR ≤0.80 corresponds with myocardial ischemia).

Figure 1

Angiographic Severity Versus Functional Severity of Coronary Artery Stenoses

Figure 3

Proportions of Functionally Discussed Coronary Arteries in Patients With Angiographic 3- or 2-Vessel Disease

All 509 patients had 3-vessel (A) or 2-vessel (B) disease by the angiographic coronary artery disease definition. Angiographic coronary artery disease (CAD) is defined as the number of major epicardial coronary arteries (left anterior descending artery, right coronary artery, or left circumflex artery) with at least 1 stenosis of ≥50%; the minimum is 2 and the maximum is 3. In all of these patients, the FFR was measured in all angiographically diseased coronary arteries. The respective numbers of diseased coronary arteries by the definition of functional CAD are displayed in the respective segments. Functional CAD is defined as the number of major epicardial coronary arteries with at least 1 stenosis with an FFR ≤0.80; the minimum is 0 and the maximum is 3. 3-VD = vessel disease; other abbreviation as in Figure 1.
FFR in Left Main Disease

A

\[
\text{\% Survival} \quad \text{\% Diameter Stenosis} = r = -0.38, p < 0.001
\]

No at risk 136 103 72 52 38 26
FFR ≥ 0.80 71 56 41 30 14 10
FFR < 0.80

B

\[
\text{\% MACE free} \quad \text{\% MACE free} = p = 0.5
\]

No at risk 136 106 77 57 42 30
FFR ≥ 0.80 71 56 40 29 15 10
FFR < 0.80

Circulation. 2009;120:1505-1512
Choice of treatment
Figure 1  Sequence of Events Following Occlusion of a Coronary Artery

Following coronary artery occlusion diastolic, systolic, and electrocardiographic (ECG) changes precede the development of angina.

Figure 2  Prognosis of Patients With Exercise-Induced Ischemia With or Without Symptoms

Probability of remaining free of myocardial infarction and sudden death in control subjects who had no known history of coronary artery disease (CAD), and in patients with medically treated CAD and ST-segment depression during exercise with (Group #2) or without (Group #1) angina. Reprinted, with permission, from Weiner et al. (35).

Figure 3  Survival After PCI in Patients With Ischemia With or Without Symptoms

Probability of event-free survival by symptomatic and ischemic status. Reprinted, with permission, from Zeiweg et al. (32). PCI = percutaneous coronary intervention.
<table>
<thead>
<tr>
<th>Test</th>
<th>No. of studies</th>
<th>Sensitivity % (95% CI)</th>
<th>Specificity % (95% CI)</th>
<th>InDOR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exercise echo</td>
<td>55</td>
<td>82.7 (80.2-85.2)</td>
<td>84.0 (80.4-87.6)*</td>
<td>3.0 (2.7-3.3)</td>
</tr>
<tr>
<td>Adenosine echo</td>
<td>11</td>
<td>79.2 (72.1-86.3)</td>
<td>91.5 (87.3-95.7)</td>
<td>3.0 (2.5-3.5)</td>
</tr>
<tr>
<td>Dipyridamole echo</td>
<td>58</td>
<td>71.9 (68.6-75.2)</td>
<td>94.6 (92.9-96.3)*</td>
<td>3.0 (2.8-3.2)</td>
</tr>
<tr>
<td>Dobutamine echo</td>
<td>102</td>
<td>81.0 (79.1-82.9)</td>
<td>84.1 (82.0-86.1)*</td>
<td>2.9 (2.7-3.0)</td>
</tr>
<tr>
<td>Combined echo</td>
<td>226</td>
<td>79.1 (77.6-80.5)</td>
<td>87.1 (85.7-88.5)*</td>
<td>2.9 (2.8-3.0)</td>
</tr>
<tr>
<td>Exercise SPECT</td>
<td>48</td>
<td>88.1 (85.8-90.3)†</td>
<td>68.8 (62.8-74.8)</td>
<td>2.7 (2.6-3.0)</td>
</tr>
<tr>
<td>Adenosine SPECT</td>
<td>14</td>
<td>90.5 (89.0-91.9)†</td>
<td>81.0 (73.5-88.6)</td>
<td>3.4 (3.0-3.8)**</td>
</tr>
<tr>
<td>Dipyridamole SPECT</td>
<td>23</td>
<td>90.4 (87.3-93.5)†</td>
<td>75.4 (66.2-84.6)</td>
<td>2.7 (2.3-3.1)</td>
</tr>
<tr>
<td>Dobutamine SPECT</td>
<td>16</td>
<td>83.6 (78.4-88.8)</td>
<td>75.1 (71.1-79.0)</td>
<td>2.5 (2.1-2.9)</td>
</tr>
<tr>
<td>Combined SPECT</td>
<td>103</td>
<td>88.1 (86.6-89.6)†</td>
<td>73.0 (69.1-76.9)</td>
<td>2.8 (2.6-3.0)</td>
</tr>
<tr>
<td>EBCT</td>
<td>21</td>
<td>93.1 (90.7-95.6)†</td>
<td>54.5 (45.3-63.8)†</td>
<td>2.6 (2.2-3.0)</td>
</tr>
</tbody>
</table>

CI: Confidence interval, InDOR, natural logarithm of the diagnostic odds ratio.

*Nonoverlapping confidence intervals indicating a statistically higher specificity than the corresponding SPECT test.

**Nonoverlapping confidence intervals indicating a statistically higher InDOR than exercise and dobutamine SPECT and EBCT.

†Nonoverlapping confidence intervals indicating a statistically higher sensitivity than the corresponding echocardiography test.

‡Nonoverlapping confidence intervals indicating a statistically higher sensitivity than all other tests, except for adenosine and dipyridamole SPECT and a statistically lower specificity than all other tests except for exercise SPECT.

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Summary diagnostic log odds ratios (boxes) and 95% confidence intervals (horizontal lines) for each type of test. The area of each box is inversely proportional to the variance in the test group, hence giving more visual prominence to test groups where the effect is more precisely estimated.
FIG 1. Comparison of sensitivities and specificities with 95% confidence intervals of the various techniques for the prediction of recovery of regional function after revascularization. (Color version of figure is available online.)
FIG 3. Comparison of sensitivities and specificities with 95% confidence intervals of the various techniques for the prediction of recovery of global left ventricular function after revascularization. (Color version of figure is available online.)
Figure 3: Kaplan-Meier Survival Curves Comparing Overall and Cardiovascular 3-Year Survival According to Treatment and Presence of Myocardial Viability in Dysfunctional Myocardium

Overall (A) and cardiovascular (B) 3-year survival were significantly worse in patients with viable myocardium who remained under medical treatment or undergoing incomplete revascularization not including dysfunctional regions. Abbreviations as in Figure 2.

Figure 5: Kaplan-Meier Survival Curves Comparing Overall and Cardiovascular 3-Year Survival in 43 Pairs of Propensity Score-Matched Patients

In these propensity score-matched patients, overall (A) and cardiovascular (B) 3-year survival remained significantly worse in patients with viable myocardium who remained under medical treatment or undergoing incomplete revascularization not including dysfunctional regions. Abbreviations as in Figure 3.
Figure 4  Hazard Ratio of Risk of 3-Year Death According to Presence or Absence of Viability and Treatment

Patients with dysfunctional viable myocardium had a 4.56 higher risk of death when remaining under medical treatment, or when revascularization was incomplete, not including the dysfunctional myocardium. CI = confidence interval; R/ = remaining under medical treatment.
Teaching Cases

JAN BOGAERT

CLINICAL CARDIAC MRI
SECOND EDITION
Acute MI (Non-STEMI)

- 36-year-old man admitted with retrosternal pain, nicotine abuse.
- ECG: non-STEMI / troponin I : 16 µg/l.
- Occlusion 1st lateral branch.
- Cardiac MRI: LV EDV 219 ml - EF 57%, mild hypokinesia in lateral wall, T2w-imaging: edema in anterolateral wall (arrows, still frames upper row), with strong transmural enhancement on late Gd MRI (arrows, lower panels).
Idiopathic DCM

- 11-year-old girl presenting with DCM.
- Cardiac MRI shows severely dilated and dysfunctional ventricles (LV EDV 324 ml - EF 9% / RV EDV 520 ml – EF 7%) with left-sided cardiac rotation / dilated atria and dilated IVC / mild pericardial effusion.
- Late Gd imaging shows diffuse strong enhancement of epicardial LV borders and diffuse RV enhancement. Myocardial biopsy shows myocardial replacement fibrosis.

See Fig. 27 Heart Muscle Diseases
RV Myocardial Infarction (2)

- T2w-imaging shows focal edema of LV inferoseptal wall (arrow, c) and diffuse edema in RV wall (arrowheads, c).
- Late Gd imaging shows focal transmural enhancement in LV inferoseptal wall (arrow, d) and diffuse enhancement of RV wall (arrowheads, d,e,f) except the anterior part of RV free wall.
- Findings of an acute MI involving both ventricles (limited LV, extensive RV) with a severe impact on RV function.

See Fig. 31 Ischemic Heart Disease
Stress Cardiomyopathy (Apical Form)

- 72-year-old woman admitted with chest pain irradiating to left arm during walking. 1-2 mm Elevation in V3-V5. Mild increase of cardiac enzymes (Troponin I: 3.8 μg/L). No significant CAD on coronary angiography but akinesia of apical half of LV, resembling a Tako-Tsubo.
- Cardiac MRI (performed two days after the acute event) shows decreased LV function (EF 41%), with residual akinesia of the entire LV apex and diffuse myocardial edema on T2w-imaging in LV apex (right panel). No abnormal myocardial enhancement on late Gd imaging. Small amount of pericardial fluid and bilateral pleural fluid.

Fig. 47 Heart Muscle Diseases
Stress Perfusion Defect in LAD Territory

- STRESS MPI
- REST MPI

See Fig. 8 Myocardial Perfusion
43-year-old woman with recent history of CAD, with LAD stent for ACS complicated with LM dissection, urgent CABG (GSV end-to-side LAD, GSV side-to-side ramus angularis, GSD end-to-side LCx). Recurrent exercise-related interscapular chest pain.

Cardiac MRI shows normal LV volumes (EDV 166 ml) and function (EF 56%) at rest (left panel) but extensive and long-lasting perfusion defect during persantine stress (segments 1,2,6,7,8,11,12,13,14). Real-time cine MRI (right panel) immediately after perfusion MRI shows severe dysfunction in non-enhanced myocardial regions. Late Gd imaging shows smal(ler) transmural enhancement in anterolateral wall (segments 1,7,12).

Cardiac catheterization shows occlusion of LAD and venous graft to LAD with filling of distal LAD by RCA collaterals, slow flow in venous graft to LCx.
Stress perfusion imaging at 2 short-axis levels (a,b) shows extensive anterior (including anteroseptum and lateral wall, arrows, a,b).

Cine imaging performed immediately following stress perfusion imaging shows severely impaired myocardial contractility (due to myocardial ischemia) in the hypoperfused myocardium (arrows, c,d).
46-year-old patient with multi-vessel CAD and recent history of NSTEMI. Cardiac catheterization shows occlusion of mid RCA and 2nd lateral branch LCx. Non-stenotic CAD in LAD. RCA filling by LAD collaterals. PCI 2nd lateral branch LCx.

LV EDV 215 ml – SV 111 ml – EF 52%. Mild thinning of the LV mid anterolateral wall showing mild to moderate hypokinesia.
Comprehensive MRI in IHD (2)

- STRESS MPI
- REST MPI
Rest MPI (previous slide) shows a perfusion ‘defect’ in the LV anterolateral wall, corresponding to the area of enhancement on late Gd imaging.

Stress MPI shows extensive and long-lasting perfusion defect in LV inferior wall (segments 3, 4, 9, 10, 15).

Late Gd imaging (suboptimal image quality) shows almost complete transmural enhancement of the LV anterolateral wall (segments 6, 12, 16).

Findings of anterolateral transmural MI, and extensive stress-induced perfusion defect in RCA territory. Rest cardiac volumes/function within normal limits.
Relative frequency of stress test and/or imaging studies performed within 90 days before enrollment in the SPARK (Study of Myocardial Perfusion and Coronary Anatomy) Imaging Trials in Coronary Artery Disease) by imaging arm. Comparisons of previous studies by modality are performed using the Cochran-Mantel-Haenszel chi-square test. The p value indicates the frequency of previous studies differed by index study modality. CTA = coronary computed tomography angiography; ETT = exercise treadmill test; PET = positron emission tomography; SPECT = single-photon emission computed tomography.

Figure 2
Post-Test Referral for Cardiac Catheterization

(A) Relative frequency of referral for cardiac catheterization within 90 days after SPECT, PET, and CTA as a function of study result. Differences in catheterization rates across study results and modalities were significant (Cochran-Mantel-Haenszel chi-square test: p < 0.001). Results of statistical testing of differences in catheterization rates between modalities within study result categories are indicated by asterisks. Differences in catheterization rates among modalities in normal/non-obstructive and mildly abnormal are significant after Bonferroni adjustment for multiple comparisons. (B) Risk-adjusted rate of referral for cardiac catheterization within 90 days after SPECT, PET, and CTA as a function of study result. Risk adjustment based on the results of general linear modeling using regions and sites as random effects (Table 4). Adjustment includes consideration of patients’ age, sex, race, anginal symptoms, diabetes, hospital status, and previous imaging study. The p values shown represent contrasts between modalities. Contrasts assume mean values of numeric predictions and modality values of categorical predictors. Differences in catheterization rates between modalities in normal/non-obstructive and mildly abnormal are significant after Bonferroni adjustment for 3 multiple comparisons. Abbreviations as in Figure 1.
Table 3  Results of Multivariable Modeling of Referral for Cardiac Catheterization at 90 Days

<table>
<thead>
<tr>
<th>Variable</th>
<th>Wall Chi-square Statistic (p Value)</th>
<th>Adjusted Odds Ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Imaging modality (overall)</td>
<td>17.4 (0.0001)</td>
<td>18.92 (1.63-20.27)</td>
</tr>
<tr>
<td>CTA**</td>
<td>13.4 (0.0001)</td>
<td>18.92 (1.63-20.27)</td>
</tr>
<tr>
<td>PET**</td>
<td>4.0 (0.046)</td>
<td>5.93 (0.84-39.43)</td>
</tr>
<tr>
<td>Imaging results (overall)</td>
<td>18.6 (0.0001)</td>
<td>26.45 (6.06-135.62)</td>
</tr>
<tr>
<td>Mildly abnormal</td>
<td>18.6 (0.0001)</td>
<td>26.45 (6.06-135.62)</td>
</tr>
<tr>
<td>Moderately to severely abnormal</td>
<td>40.1 (0.0001)</td>
<td>199.22 (16.38-2,108.21)</td>
</tr>
<tr>
<td>Imaging modality = imaging results</td>
<td>18.6 (0.0001)</td>
<td>26.45 (6.06-135.62)</td>
</tr>
<tr>
<td>PET, mild abnormality</td>
<td>3.6 (0.14)</td>
<td>0.34 (0.04-4.42)</td>
</tr>
<tr>
<td>CTA, mild abnormality</td>
<td>6.9 (0.09)</td>
<td>0.44 (0.09-9.0)</td>
</tr>
<tr>
<td>PET: moderate to severe abnormality</td>
<td>2.1 (0.24)</td>
<td>0.25 (0.04-1.09)</td>
</tr>
<tr>
<td>CTA: moderate to severe abnormality</td>
<td>0.3 (0.99)</td>
<td>0.08 (0.03-2.51)</td>
</tr>
<tr>
<td>Age &gt;= 75 years, yes</td>
<td>13.4 (0.02)</td>
<td>1.7 (0.49-6.49)</td>
</tr>
<tr>
<td>40-44 yrs</td>
<td>4.0 (0.13)</td>
<td>3.96 (0.13-12.23)</td>
</tr>
<tr>
<td>45-54 yrs</td>
<td>3.4 (0.09)</td>
<td>3.03 (0.87-10.51)</td>
</tr>
<tr>
<td>55-64 yrs</td>
<td>3.8 (0.08)</td>
<td>3.79 (0.95-15.07)</td>
</tr>
<tr>
<td>65-74 yrs</td>
<td>3.0 (0.35)</td>
<td>5.11 (0.54-48.39)</td>
</tr>
<tr>
<td>Male</td>
<td>8.2 (0.004)</td>
<td>2.8 (1.05-7.28)</td>
</tr>
<tr>
<td>White race</td>
<td>8.4 (0.003)</td>
<td>3.46 (1.25-9.40)</td>
</tr>
<tr>
<td>Regional</td>
<td>16.1 (0.001)</td>
<td>3.16 (1.78-5.62)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>2.2 (0.49)</td>
<td>1.15 (0.49-2.79)</td>
</tr>
<tr>
<td>Hospital status/center/external</td>
<td>2.1 (0.38)</td>
<td>1.52 (0.86-2.68)</td>
</tr>
<tr>
<td>Previous imaging study</td>
<td>0.3 (0.90)</td>
<td>0.99 (0.35-2.73)</td>
</tr>
</tbody>
</table>

*These odds ratios are for imaging modality (CTA, CTA) compared with no abnormal or normal computed tomography. **These odds ratios are for imaging results (overall, mild abnormality) compared with normal findings or angiographic coronary artery computed tomography. The odds ratios are for the age group range 40-59 years. The odds ratios are for the presence of the characteristics compared with its absence.

Figure 3  Post-Test Referral for Revascularization

Relative frequency of referral for coronary artery bypass graft (CABG) and percutaneous coronary intervention (PCI) within 90 days after normal or nonobstructive versus abnormal cardiac imaging study results. In patients referred for cardiac catheterization in each of the 3 imaging arms, Revascularization rates were higher in patients with abnormal versus normal or nonobstructive CTA results (Cochran-Mantel-Haenszel, p < 0.05). Abbreviations as in Figure 1.

Figure 4  Medical Therapy Before and After Noninvasive Testing

Absolute frequency of medication use at baseline and 90 days in the subgroup of patients with moderately or severely abnormal imaging study results. Comparisons between frequencies of medication use are made using a chi-square test of association. The p values indicate differences in baseline versus 90-day medication use. LLA = lipid-lowering agent.
Figure 5  Post-Test Changes in Patient Management

Frequency of referral for catheterization (Cath), medication (Med) change, both, or neither at 90 days as a function of cardiac imaging study results. Comparisons made with chi-square tests of association within each test result.
Conclusion

• Look for ischemia and viability
  – Presence
  – Extent

• Use the technique you know best

• ACT UPON YOUR FINDINGS