Acute Coronary Syndromes

2016

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68 yo Man, Chest pain after lunch on the way to car. 
Bad sushi?
CAD is a diffuse process with focal atherosclerotic material (plaque).

Some plaques are obstructive but not thrombotic. Others are potentially thrombotic but not obstructive.

Myocardial Infarction = Death of myocardial cells.

Clinical MI = symptoms, ECG and Biomarkers
CAD as a cause of Myocardial Ischemia and Infarction

Normal

Atherosclerotic Plaque

IE
E
IP
A
M
Angiography vs. Pathology
Fibrous plaque
Soft plaque
Positive remodeling

ACS
LAD

Angiography vs CTA for CAD

Motoyama et al. JACC 2007
Natural History of CAD: A story of remodeling

- Normal Artery
- Minimal CAD
- Moderate CAD
- Severe CAD

Compensatory expansion limits luminal encroachment
Luminal encroachment overcomes expansion
• Acute Coronary Syndrome

• Plaque crater, erosion

• Thrombus

• Calcific nodule

• 72 year-old Man
What are the Big 5 medications for CAD?

1. BB
2. ASA/antiplatelet agents
3. Statins
4. Nitrates
5. Antihypertensive and other risk factor medications
Beta blockers
CA blockers
ACEI
NTG

Ranolazine

Statins

Heparin

ASA

NTG

Braunwald’s Heart Disease, 7th Edition
Ischemic Cascade

- Perfusion Abnormalities
- Systolic Dysfunction
- Diastolic Dysfunction
- Angina
- Δ ECG
- Stress ECG
- Stress Echo/MRI
- Nuclear Imaging

Duration and severity of ischemia
Who needs Stress Testing?

Indications for diagnostic testing in patients with suspected CAD and stable symptoms.

<table>
<thead>
<tr>
<th></th>
<th>Asymptomatic</th>
<th>Symptomatic</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Low (&lt;15%)</td>
</tr>
<tr>
<td>Class*</td>
<td>Level(^d)</td>
<td>Class*</td>
</tr>
<tr>
<td>--------------------------------</td>
<td>--------------</td>
<td>--------------</td>
</tr>
<tr>
<td><strong>Anatomical detection of CAD</strong></td>
<td></td>
<td>--------------</td>
</tr>
<tr>
<td>Invasive angiography</td>
<td>III</td>
<td>III</td>
</tr>
<tr>
<td>CT angiography(^{18})</td>
<td>III</td>
<td>B</td>
</tr>
<tr>
<td><strong>Functional test</strong></td>
<td></td>
<td>--------------</td>
</tr>
<tr>
<td>Stress echo</td>
<td>III</td>
<td>III</td>
</tr>
<tr>
<td>Nuclear imaging</td>
<td>III</td>
<td>III</td>
</tr>
<tr>
<td>Stress MRI</td>
<td>III</td>
<td>B</td>
</tr>
<tr>
<td>PET perfusion</td>
<td>III</td>
<td>B</td>
</tr>
<tr>
<td>Combined or hybrid imaging test</td>
<td>III</td>
<td>C</td>
</tr>
</tbody>
</table>

EuroIntervention 2015;10:1024-1094 published online ahead of print September 2014
2014 ESC/EACTS Guidelines on myocardial revascularization
## Spectrum of ACS Presentations

<table>
<thead>
<tr>
<th></th>
<th>UA</th>
<th>NSTEMI</th>
<th>STEMI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Definition</strong></td>
<td>Ischemia without necrosis</td>
<td>Necrosis (nontransmural)</td>
<td>Transmural necrosis</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>Negative Biomarkers</td>
<td>Positive biomarkers</td>
<td>Positive biomarkers</td>
</tr>
<tr>
<td></td>
<td>No ECG ST-segment elevation</td>
<td>ECG ST-segment elevation</td>
<td></td>
</tr>
<tr>
<td><strong>Treatment</strong></td>
<td>Invasive or conservative depending on risk</td>
<td>Immediate reperfusion</td>
<td></td>
</tr>
</tbody>
</table>
Heart Attack Warning Signs

- Chest discomfort
  - Pressure
  - Squeezing
  - Fullness
  - Pain
- Discomfort in other areas of the upper body
  - Arms
  - Jaw
  - Neck
  - Back
  - Stomach
- Shortness of Breath
- Cold sweat, nausea or lightheadedness
- **Women have atypical presentations!! Be more wary**
### Who Presents With Atypical Symptoms?

<table>
<thead>
<tr>
<th></th>
<th>Only Atypical Symptoms (no chest pain)</th>
<th>Any Typical Symptoms (chest pain present)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Older patients (mean age, years)</td>
<td>74.2</td>
<td>66.9</td>
</tr>
<tr>
<td>Women</td>
<td>49.0%</td>
<td>38.0%</td>
</tr>
<tr>
<td>Diabetes</td>
<td>32.6%</td>
<td>25.4%</td>
</tr>
<tr>
<td>Prior heart failure</td>
<td>26.4%</td>
<td>12.3%</td>
</tr>
<tr>
<td>Mean time between first symptoms and presentation (hours)</td>
<td>7.9</td>
<td>5.3</td>
</tr>
<tr>
<td>% diagnosed with MI on admission</td>
<td>22.2%</td>
<td>50.3%</td>
</tr>
<tr>
<td>% receiving reperfusion treatment</td>
<td>25.3%</td>
<td>74.0%</td>
</tr>
<tr>
<td>In-hospital mortality</td>
<td>23.3%</td>
<td>9.3%</td>
</tr>
</tbody>
</table>

Key Features of an ECG

- **P-R Interval**: (continued from next heartbeat)
- **T-P Interval**: (continued from next heartbeat)
- **QRS complex**
- **Ventricular depolarization**
- **Atrial depolarization**
- **Ventricular repolarization**

Time (s)

Example of ST-segment Elevation (STEMI)
Example of ST-segment Depression (UA/STEMI)

- SPF
- J point

STD

NSTEMI

ST Depression
Example of T-wave Inversion (UA/STEMI)

T wave changes
Normal 12-lead ECG

INFERIOR

LATERAL

ANTERIOR

LATERAL

Early-Stage Acute MI (STEMI)
3-Day-Old MI (STEMI)

- ST-segment elevation
- T-wave inversion
UA - NSTEMI

T-wave inversion
Treatment of Acute Coronary Syndrome

Initial Treatment of ACS

STEMI*
- antiplatelet, anti-ischemic, or anticoagulant therapy
  - Thrombolitics
  - PCI or CABG

UA/NSTEMI†
- antiplatelet, anti-ischemic, or anticoagulant therapy
  - Early Invasive
    - PCI or CABG
  - Initial Conservative

Long-term medical management

* Also known as Q-wave MI
† Also known as non-Q-wave MI
# New clinical classification of MI

<table>
<thead>
<tr>
<th>Classification</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Spontaneous MI due to coronary event, i.e. plaque erosion and/or rupture, fissuring, or dissection</td>
</tr>
<tr>
<td>2</td>
<td>MI secondary to ischemia due to an imbalance of O\textsubscript{2} supply and demand, as from coronary spasm or embolism, anemia, arrhythmias, hypertension, or hypotension</td>
</tr>
<tr>
<td>3</td>
<td>Sudden unexpected cardiac death, including cardiac arrest, with new ST-segment elevation; new LBBB; or pathologic or angiographic evidence of fresh coronary thrombus—in the absence of reliable biomarker findings</td>
</tr>
<tr>
<td>4a</td>
<td>MI associated with PCI</td>
</tr>
<tr>
<td>4b</td>
<td>MI associated with documented in-stent thrombosis</td>
</tr>
<tr>
<td>5</td>
<td>MI associated with CABG surgery</td>
</tr>
</tbody>
</table>
**Table 5** Common ECG pitfalls in diagnosing myocardial infarction

<table>
<thead>
<tr>
<th>False positives</th>
<th>False negatives</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benign early repolarization</td>
<td>Prior myocardial infarction with Q-waves and/or persistent ST elevation</td>
</tr>
<tr>
<td>LBBB</td>
<td>Paced rhythm</td>
</tr>
<tr>
<td>Pre-excitation</td>
<td>LBBB</td>
</tr>
<tr>
<td>Brugada syndrome</td>
<td></td>
</tr>
<tr>
<td>Peri-/myocarditis</td>
<td></td>
</tr>
<tr>
<td>Pulmonary embolism</td>
<td></td>
</tr>
<tr>
<td>Subarachnoid haemorrhage</td>
<td></td>
</tr>
<tr>
<td>Metabolic disturbances such as hyperkalaemia</td>
<td></td>
</tr>
<tr>
<td>Failure to recognize normal limits for J-point displacement</td>
<td></td>
</tr>
<tr>
<td>Lead transposition or use of modified Mason–Likar configuration</td>
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</tbody>
</table>

Biomarkers of Myocardial Damage

- Troponin T (~15%)
- Tropomyosin (<5%)
- Troponin C
- Troponin I
- Myosin-binding protein C (~15%)
- Actin
- Myosin light chain (<1%)
- β-Myosin heavy chain (~35%)
- Myosin rod
- Myosin head
Cardiac-specific troponins are optimum biomarkers (Level IC)

For STEMI, reperfusion therapy should be initiated as soon as possible and is not contingent on a biomarker assay (Level IC)


Anderson JL, et al. J Am Coll Cardiol 2007;50:e1–e157, Figure 5.
Troponin Elevation and Likelihood for Mortality


% mortality at 42 days

Troponin levels

- <0.4
- <1.0
- <2.0
- <5.0
- <9.0
- ≥9.0

0 2 4 6 8
Non-MI Causes of Troponin Elevation

Not a Myocardial Infarction

Cardiac

- Congestive heart failure
- Infection
  - Viral cardiomyopathy
- Inflammation
  - Myocarditis
  - Pericarditis
- Trauma
  - Surgery
  - Electrical shock
- Ablation procedures
- Malignancy
- Stress cardiomyopathy
- Infiltrative diseases

Systemic

- Pulmonary embolism
- Toxicity
  - Anthracyclines
- Trauma
  - Blunt chest wall injury
  - Congestive heart failure (due to volume overload)
- Renal failure
- Sepsis
- Stroke
- Subarachnoid hemorrhage

J Am Coll Cardiol. 2014;63(3):201-214
54 yo M w 2h severe substernal CP - ECG
Left Coronary System has mild CAD. RCA is 100%
Final post Stent
PCI vs Fibrinolysis in STEMI

Systematic Overview

(23 RCTs, n=7,739)

Short term (4-6 weeks)

Medical Therapy for STEMI Managed by Primary PCI

Presentation

ASA

Anticoagulant

UFH
(Clopidogrel 600
Prasugrel 60, or Ticagrelor 180)

P2Y12 inhibitor

Eptifibatide
(ABCX)

GP IIb/IIIa

IV prn

Beta Blocker

Oral within 24h

Statin

Access—Wire—Balloon

ED

CCL
Importance of Rapid Reperfusion in STEMI

30-minute delay = 8% increase in 1-year mortality


48 yo Man, Chest pain after lunch while walking to car.
48 yo M, HBP with Chest pain while walking
## TIMI Risk Score (n=7)

<table>
<thead>
<tr>
<th>TIMI Risk Score Calculator</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Age ≥65 years?</td>
<td>Yes (+1)</td>
</tr>
<tr>
<td>≥3 Risk Factors for CAD?</td>
<td>Yes (+1)</td>
</tr>
<tr>
<td>Known CAD (stenosis ≥50%)?</td>
<td>Yes (+1)</td>
</tr>
<tr>
<td>ASA Use in Past 7d</td>
<td>Yes (+1)</td>
</tr>
<tr>
<td>Severe angina (≥2 episodes w/in 24 hrs)?</td>
<td>Yes (+1)</td>
</tr>
<tr>
<td>ST changes ≥0.5 mm?</td>
<td>Yes (+1)</td>
</tr>
<tr>
<td>+ Cardiac Marker?</td>
<td>Yes (+1)</td>
</tr>
<tr>
<td>Total Score</td>
<td>pts</td>
</tr>
</tbody>
</table>


What does TIMI RISK mean?

Increasing TIMI RISK 0/1 to 5/7 increases risk of death, MI, urgent revascularization within 14 days 5% to 41%.

Antman EM et al. TIMI 11B, JAMA 2000;284:835-842
STEMI?
Differential Dx for ACS Chest Pain Syndromes (beyond STEMI, NSTEMI, UA)

- Aortic dissection
- Pulmonary embolus
- Perforating ulcer
- Pericarditis
- GERD (Gastroesophageal reflux disease)
- Heart failure, Pneumonia, Pneumothorax