Chest Pain Patient Evaluation in the ED:

Detecting AMI/ACS Patients & Excluding Those Without Disease

The Rapid (Two Hour) Rule Out in the Emergency Department
Global Objectives

- Diagnose AMI & ACS patients
- Decide which chest pain patients do not have AMI or ACS
- Know which patients can be discharged safely from the ED
- Have no misses

Session Objectives

- Provide AMI, ACS patient overview
- Discuss the early and recent literature that supports the rapid evaluation of ED chest pain patients
- Decide how to rapidly assess ED chest pain patients
- Understand to whom the rapid rule out may apply

Specific Learning Points

- AMI & ACS patients are unstable or are sick in some discernible way
- If the ED patient has not AMI or ACS, then admission is not necessary
- The rapid ED evaluation excludes acute cardiac instability
- A protocol must logically exclude Dx
- Provocative testing is not mandatory
Clinical Cases

Chest Pain Case #1: Hx

- 58 year old male
- Chest pain, sub-sternal, severe
- Onset less than one hour prior
- Nausea, diaphoresis
- No known cardiac history
- Smoker, ?cholesterol

Chest Pain Case #1: Px

- 98.8 100/60 110 24
- Gen: Screaming in pain, diaphoretic
- Chest: BS equal
- CV: Reg rhythm without
Chest Pain Case #2: Hx

- 48 year old male
- Sudden onset of chest pain
- SOB, nausea
- ? Cardiac hx, on ASA
Chest Pain Case #2: Px

- 98.6 160/90 116 24
- Gen: Diaphoretic, pale, anxious
- Chest: Clear BSBE
- CV: Reg without
- Exam otherwise normal
Chest Pain Case #3: Hx
- 43 year old female
- Sharp chest pain, intermittent
- “Chest blowing up like a balloon”
- SOB and tired with exertion
- Dad died at age 45 due to AMI
- Her 3rd visit to EDs in one week

Chest Pain Case #3: Px
- 98.6 154/88 92 20
- Gen: Warm, dry, mildly anxious
- Chest: Clear BSBE
- CV: Reg without
- Exam otherwise normal
How common is acute myocardial infarction and/or acute coronary syndrome?
**AMI/ACS Epidemiology**
- CAD: 7 million Americans
- AMI: 1.5 million per year
- 1996: 750,000 admissions
  - 50% NSTEMI
  - 50% STEMI

**AMI/ACS Incidence**
- STEMI US Incidence
  - 121 per 100,000 in 1997
  - 77 per 100,000 in 2005
  - At least one-third reduction
- Related to enhanced prevention
- Risk factor mitigation

**Acute Coronary Syndrome Definitions**
ACS Disease Spectrum

- Stable angina
- Variant angina
- Unstable angina
- AMI/ACS
- Cardiogenic shock
- Cardiac arrest

Definitions

Stable Angina

- Stable Angina
  - CP < 10min (Brief)
  - Improved with rest or NTG
  - No change in characteristics
  - Same exacerbating factors
  - Same frequency

Definitions

Variant Angina

- Variant Angina
  - Arterial spasm is cause
  - At rest, frequently at night
  - Pain longer than stable angina
  - \( \uparrow \) ST segments
  - 2/3 have atherosclerosis
  - Hard to sort out clinically
**Definitions**

**Unstable Angina**
- Recent onset
- Changing clinical features
- More easily induced
- \( \uparrow \) frequency, duration, severity
- Relieved by NTG
- May occur at rest

**Unstable Angina Outcomes**
- Unstable angina
  - 40% AMI incidence without Rx
  - 17% mortality without Rx

**Acute Myocardial Infarction**
- Acute myocardial infarction
  - Chest pain > 15-20 min
  - Less relief with NTG or rest
  - Either EKG \( \Delta \)'s or + enzymes
  - Mortality 35% if untreated
AMI/ACS Pathophysiology

- What is the mechanism of an acute coronary syndrome?
- How does it occur?
- How does it manifest itself in ED chest pain patients?

AMI/ACS Mechanism

- Cell death and tissue necrosis results from the imbalance between myocardial oxygen supply and demand
- Limited cardiac muscle blood supply due to an obstructing coronary artery lesion
Coronary Artery Plaque

Coronary Artery Plaque Formation

Chronic CAD

- Chronic coronary artery occlusion:
  - Rings of tree: a chronic process
  - Decreasing circumference over time
  - Collaterals may exist
  - Diffuse coronary artery disease
  - Patients present with cardiomegaly, CHF, worsening exertional dyspnea
**Acute Coronary Syndrome**

- Acute coronary artery occlusion:
  - Acute volcanic eruption
  - Plaque rupture, thrombus formation
  - Dynamic loss of function
  - Present with ACS/AMI
  - New onset pain, dyspnea, syncope, dysrhythmias

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**Coronary Artery Plaque Rupture**

- Sudden changes in intraluminal pressure
- Bending, twisting during heart contraction
- Lipid content of plaque
- Thickness of fibrous cap
- Plaque shape
- Mechanical injury
- Infection?
“At-Risk” Coronary Arteries

- Vessels with what type of occlusion are most likely to cause an acute coronary syndrome?
- Is it the coronary artery of 40, 60, or 80% occlusion?

“At-risk” Coronary Arteries

- Chronic Rings: 80%+ lesions
- UA/NSTEMI because collaterals exist
- Acute rupture: 30-80% vessel lesions
- STEMI occurs, fewer collaterals exist
- Large, chronic obstructing lesions not necessarily at the greatest risk for rupture, ACS

Coronary Artery Plaque
**AMI/ACS Pathophysiology**

- What is the difference in pathology between ST elevation AMI (STEMI) and non-ST elevation AMI (non-STEMI)?
- Does this difference impact outcome?

**AMI/ACS: Acute Thrombosis**

- **UA/NSTEMI:**
  - Partial occlusion.
  - Primarily Platelets
  - Intra-plaque thrombus (platelet dominated)

- **STEMI:**
  - Full occlusion.
  - Platelets, red blood cells, fibrin.
  - Intra-plaque thrombus (platelet dominated)

**SUDDEN DEATH**

- 81% occlusive
- 100% fibrin-rich
- 100% non-occlusive
- 71% platelet-rich
- 19% non-occlusive
- 29% fibrin-rich

*AMI/ACS Thrombus Features*
Acute Coronary Syndrome
- Partial plaque disruption
- White clot: platelet aggregation
  - UA/NSTEMI
- Large, complete plaque disruption
- Red clot: clotting factors involved
  - STEMI
AMI/ACS Pathophysiology

Open Artery Imperative

- Open artery quickly
- Minimize myocardium loss
- Avoid fatal dysrhythmias
- Prevent long-term dysfunction

AMI/ACS Pathophysiology

Open Artery Theory

- Spontaneous reperfusion
- 33% reperfusion by 1 day
- 50% reperfusion by 1 week
- Physiologic homeostasis
- Too late to avoid complications

AMI/ACS Outcome

- What factors impact AMI/ACS?
- Does it differ based on the EKG changes and AMI type (NSTEMI vs. STEMI?)
**EKG & AMI/ACS Outcome**

<table>
<thead>
<tr>
<th>Pt Risk</th>
<th>EKG Characteristics</th>
<th>Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>High Risk</td>
<td>Pathologic Q-waves</td>
<td>42% incidence of MI</td>
</tr>
<tr>
<td></td>
<td>Ischemic ST or T Δ’s</td>
<td>14% serious complications</td>
</tr>
<tr>
<td></td>
<td></td>
<td>10% mortality</td>
</tr>
<tr>
<td>Low Risk</td>
<td>LVH, LBBB, paced,</td>
<td>14% incidence of MI</td>
</tr>
<tr>
<td></td>
<td>Non-specific ST or T Δ’s,</td>
<td>0.6% serious complications</td>
</tr>
<tr>
<td></td>
<td>No Δ from previous non-AMI EKG</td>
<td>0% mortality</td>
</tr>
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</table>

**AMI/ACS Complications**

- Dysrhythmias
- Cardiac pump failure

<table>
<thead>
<tr>
<th>Class</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>No CHF</td>
<td>5%</td>
</tr>
<tr>
<td>Mild CHF</td>
<td>15-20%</td>
</tr>
<tr>
<td>Pulmonary edema</td>
<td>40%</td>
</tr>
<tr>
<td>Cardiogenic shock</td>
<td>80%</td>
</tr>
</tbody>
</table>

**AMI/ACS Diagnosis:**

*History and Physical*
How do we assess risk in chest pain patients?

AMI/ACS Diagnosis: History

Pain characteristics
Palliative/provocative features
Associated cardiac symptoms
Cardiac history
Cardiac risk factors
Family Hx: early AMI, sudden death

AMI/ACS: Risk Assessment

Associated cardiac symptoms
- Dyspnea
- Diaphoresis
- Nausea
- Palpitations
**AMI/ACS: Other Diagnoses?**
- Increased exercise or use?
- Recent or remote trauma?
- History of costochondritis?
- PE symptoms, history or RF?
- Pericarditis sx, history or RF?
- Asthma, bronchitis, pneumonia sx?

**AMI/ACS: Other Diagnoses?**
- Gastrointestinal history
  - GERD
  - Gastritis
  - Duodinitis
  - Pancreatitis
  - Gallbladder disease

**AMI/ACS: Risk Assessment**
- Low, medium, high risk
- Does the patient have an acute coronary syndrome (ACS)?
- Is there a ruptured plaque that is occluded by a white or red thrombus?
**AMI/ACS History**

- Important clinical tool
- Chest pain may not be “classic”
  - Crushing, heaviness 24%
  - Burning pain 23%
  - Pleuritic pain 19%
  - Reproducible pain 8-15%
  - Sharp/stabbing pain 5%

**Anginal Equivalents**

- 25-30% of MI’s are clinically silent
- Women, diabetics and elderly at risk
- All three may present atypically
  - Less likely to present with CP
  - Less likely to have diaphoresis, N/V
- Dizziness, syncope may be only sx

**Atypical Presentations**

- Back, neck, jaw, abdominal pain
- Dyspnea
- Diaphoresis
- N/V
- Syncope
- Confusion
- Generalized weakness
- Dizziness, TIA
AMI/ACS

**History**

- Associated symptoms
  - Diaphoresis 80%
  - Nausea 60%
  - Belching 47%
  - Vomiting 39%

Coronary Atherosclerosis

**Risk Factors**

- Male gender
- Post menopausal women
- Age > 50
- HTN, diabetes
- Hypercholesterolemia
- Family history of atherosclerosis or early AMI or sudden death

Coronary Atherosclerosis

**Risk Factors**

- Smoking
- Cocaine usage
- Peripheral Vascular Disease
- Obesity
**AMI/ACS History:**

**Prior Cardiac Catheterization**

- How long of a time does the report of a “negative” cardiac catheterization exclude a likely acute coronary syndrome?

Perhaps 6-12 months, unless patient presents with STEMI or unstable
- Must determine normal coronaries
- Must not have non-occlusive lesions
- Was the cardiac cath negative?
- Or were there non-occlusive lesions?
- This addresses the 30-60% plaque that is prone to disruption and STEMI

**AMI/ACS History:**

**Prior Negative Stress Test**

- In “at-risk” patients, stress annually
- Repeat provocative testing also when symptoms suggest ACS
- Non-invasive diagnosis possible
- Inducible ischemia can be detected
**AMI/ACS Diagnosis**

**Physical Exam**

- Pleuritic, positional pain
- Palpable tenderness
- CHF, DVT, PE, AAA
- New murmur (valve)
- Unequal BP, pulses
- Cardiac dysrhythmia
EMR Documentation

- **ED Chest Pain Patient**
- History and physical that includes every one of these variables can be documented in a matter of minutes
- Chest pain template

AMI/ACS Diagnosis: EKG

- How do we diagnose AMI/ACS?
- How soon after triage should the first EKG be performed?
**AMI/ACS Diagnosis: EKG**

- How do we diagnose AMI/ACS?
  - In part, with diagnostic EKG changes
- How soon after triage should the first EKG be performed?
  - 10 minutes

**EKG Diagnosis**

**Ischemia or NSTEMI (ACS)**

- Ischemia or NSTEMI
  - ST segment depression
  - Symmetric T wave inversions
  - Inverted/biphasic T waves
  - Non-specific ST or T ‘s

**EKG Diagnosis**

**Acute Myocardial Infarction**

- Early Infarction (AMI)
  - Peaked T waves
  - ST segment elevation
EKG Diagnosis

Acute Myocardial Infarction

- Late Infarction (AMI)
  -Normalization of ST segment
  -Development of Q waves

AMI/ACS: EKG Localization

<table>
<thead>
<tr>
<th>Anatomic Location</th>
<th>EKG Location</th>
<th>Coronary Artery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Septal</td>
<td>V1-2</td>
<td>LAD lesion</td>
</tr>
<tr>
<td>Anterior</td>
<td>V3-4</td>
<td>LAD lesion</td>
</tr>
<tr>
<td>Lateral</td>
<td>V4-6, I, aVL</td>
<td>Circumflex branch of LAD</td>
</tr>
<tr>
<td>Inferior</td>
<td>II,III, aVF</td>
<td>RCA (90%)</td>
</tr>
<tr>
<td>Posterior</td>
<td>V8-9</td>
<td>Circumflex or dominant branch of RCA</td>
</tr>
<tr>
<td>Right Ventricular</td>
<td>V4R</td>
<td>RCA</td>
</tr>
</tbody>
</table>
AM/ACS: EKG Localization

- I lateral (V1, V2)
- aVR
- aVL lateral (V3, V4)
- aVF inferior (V5, V6)
- V1 septal
- V2 septal
- V3 septal
- V4 anterior
- V5 lateral
- V6 lateral

AM/ACS: Coronary Arteries

Left:
- Septal wall of LV
- Anterior and lateral walls of LV
- Inferior wall LV (10%)
- Both bundle branches

Right:
- Inferior LV wall
- Posterior wall of LV (90%)
- AV node (90%)
- Right ventricle

Anteroseptal MI

- ST elevation in V1, V2
**ED Chest Pain Patients, AMI/ACS, and ED Evaluation**

**Edward P Sloan, MD, MPH, FACEP**

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**Anteroseptal MI**
- ST elevation in V1, V2

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**Anterior Wall MI**
- ST elevation in V2, V3

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**Anterolateral Wall MI**
- ST elevation in V2-V6
**Anterolateral Wall MI**

- ST elevation in V4, V5

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**Inferior Wall MI**

- ST elevation in II, III, AVF

**AMI/ACS Diagnosis: EKG**

- How do we diagnose true posterior wall AMI? RV AMI?

**True Posterior AMI**

- Initial R waves V1, V2; R/S ratio ≥1
**True Posterior AMI**
- Initial R waves V1, V2; R/S ratio ≥1

**Right Ventricular AMI**
- ST elevation in II, III, aVF
- Also noted in V4R to V6R

**Right Ventricular Leads**
**Posterior Wall AMI**

- ST changes in $V_{1R}$ to $V_{6R}$

**Right Ventricular AMI**

- RCA occlusion
- Inferior and RV infarction

**AMI Diagnosis: EKG**

- What are the indications for additional EKG leads? Which are done? How? Why?
EKG Extra Leads Indications
- EKG shows posterior wall AMI
- Inferior wall MI with hypotension: ??? RV infarction
- Ischemic changes, cardiogenic shock, search for PCI indications

AMI Diagnosis:
EKG V₄R, Inferior ST Elevation

Lead V₄R = diagnostic ST-segment elevation
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EKG Extra Leads: V4R V8 V9
**AMI/ACS: EKG Extra Leads**

- Zalenski extra leads study
- Inf MI, 14% complications
- Inf MI, RV AMI, 26% complications

**AMI/ACS: EKG Extra Leads**

- Zalenski extra leads study
- V4R, V8, V9
- 0.5 mm ST elevation
- Detects RV, posterior infarcts
- Enhances resuscitation
- IVF, nitrate use

**AMI/ACS: EKG Extra Leads**

Assessing the diagnostic value of an ECG containing leads V4R, V8, and V9: The 15-Lead ECG


Zalenski et al

Robert J Zalenski
David Cooke
Robert Rydman
Edward P Sloan
Daniel G Murphy
AMI/ACS: EKG Extra Leads

- Zalenski et al
- Major abnormalities (ST-segment deviation, T-wave inversion, Q waves) were found on the extra three leads in 28.9% (43 of 149) of patients.
- Sensitivity of ST-segment elevation for AMI on 12 versus 15 leads increased from 47.1% to 58.8%, respectively, with no decrease in specificity.
- 22% of patients negative for ST elevation on 12 leads were positive on 15 leads.
- Sensitivity for TT increased from 35.3% to 44.1%.
- 13.5% of patients not meeting criteria on 12 leads did so on 15 leads.

EKG Diagnosis

AMI/ACS Dx in LBBB

- Rules for detecting MI with LBBB
  - ↑ ST > 1mm and concordant with QRS = 5 pts
  - ↓ ST > 1mm in V1, V2, or V3 = 3 pts
  - ↑ ST > 5mm and discordant with QRS = 2 pts
- Higher scores more likely MI present with LBBB
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EKG Diagnosis

ST Elevation in Pericarditis

- Diffuse ST elevation, atypical

AMI/ACS

EKG Diagnosis

- EKG non-diagnostic in 50% of AMI cases (NSTEMI)
- Cardiac enzymes useful
- Serial EKGs useful

AMI/ACS Dx: EKG Timing

- An EKG should be obtained within 10 minutes of presentation and initial triage
When comparing to an old EKG, the old EKG should not be one that demonstrates AMI or ACS. The goal is to establish no change as compared to baseline, non-ischemia EKG. For example, is the LVH with strain pattern new or old?

LVH criteria, ST-T wave changes

- Dysrhythmias
- Bradycardia
- Heart blocks
- Malignant ventricular ectopy
- Ventricular Fibrillation
**Bifascicular Block**

- RBBB: Opposite T waves normal

**Non-sustained VT in AMI**

**Non-sustained VT in AMI**
AMI/ACS Diagnosis: Cardiac Enzymes

AMI/ACS: Lab Evaluation

<table>
<thead>
<tr>
<th>Marker</th>
<th>Elevation</th>
<th>Peak</th>
<th>Duration</th>
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<tbody>
<tr>
<td>Myoglobin</td>
<td>1-4 h</td>
<td>6 h</td>
<td>24 h</td>
</tr>
<tr>
<td>Troponin I</td>
<td>3-12 h</td>
<td>18 h</td>
<td>5-10 days</td>
</tr>
<tr>
<td>Troponin T</td>
<td>3-12 h</td>
<td>12 h</td>
<td>5-14 days</td>
</tr>
<tr>
<td>CK-MB</td>
<td>3-12 h</td>
<td>12-24 h</td>
<td>2 days</td>
</tr>
</tbody>
</table>

Cardiac Enzymes: CPK-MB

- CPK-MB
  - Sensitive and specific
  - Limited potential for early diagnosis?
  - Two hour change in CK-MB may be accurate to predict ACS
Cardiac Enzymes: Myoglobin

- Myoglobin
  - Sensitive but not specific
  - Not used to rule out clinically
  - Peaks earlier than CPK
  - Limited utility given ability to use CPK-MB and troponin

Cardiac Enzymes: Troponins

- Troponins: true cardiac markers
  - Touted to be highly specific
  - More sensitive than CPK-MB at detecting small infarcts
  - Elevation predicts ↑ mortality and complications
  - Troponin T less specific than troponin I

Cardiac Enzymes: Troponins

- Troponins
  - Can troponins be + without ACS?
  - What is the significance of isolated troponin elevation with a non-diagnostic EKG?
  - Cannot overlook higher complication rate if troponin is positive
**Troponin Positive Patients**

- Do these patients have an ACS?
- Should heparin be started?
- Should pts go to the cath lab?
- Should pts be admitted to the ICU?
- How should higher risk be mitigated?

- No clear answers

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**AMI/ACS Diagnosis:**

*Other Acute Tests*

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**AMI/ACS Diagnosis**

*CXR*

- CM, CHF
- Other causes of chest pain
  - Pneumothorax, rib fractures
  - Pneumonia
  - Aortic dissection
  - Carcinoma
AMI/ACS Diagnosis

CXR
- Acute pulmonary edema

Echocardiography
- What are the indications for echocardiography in the ED?
- How might this data alter the acute management of an STEMI patient?
- Uncertain ACS diagnosis
- Uncertain need for acute PCI
- LV dysfunction suggests more gravely ill AMI patient
- Valvular dysfunction diagnosed
AMI/ACS Diagnosis

Echocardiography

- Sensitive in detecting regional wall motion abnormalities
- Cannot differentiate between ischemia, AMI, or old MI
- Can detect valve stenosis, regurgitation, insufficiency

AMI/ACS Diagnosis

Stress Testing

- Measures inducible ischemia
- Must rule out AMI/ACS first
- Two sets of cardiac enzymes
- Two EKGs
- No suggestion of AMI
- No interval chest pain
AM/ACS Diagnosis

**Stress Testing**

- Physical, process limitations
- Have to be able to walk
- Stress thallium may be needed
- Provides better detection of ischemia when EKG detection is limited

**Stress thallium testing**

In general, thallium clears more slowly from regions supplied by stenotic vessels than from normal myocardial regions.

- Areas of significant hypoperfusion will have very slow clearance and may even accumulate thallium.

**Sestamibi (Cardiolyte) Scanning**

- Technetium
- Measures focal ischemia
- Pain for at least 1 hr or ongoing
- Resolution within 30 min only
- Poor perfusion, no uptake
- May lead to early catheterization
- Can be linked with stress testing
AMI/ACS Diagnosis

Sestamibi (Cardiolyte) Scanning

- Myoview™ (technetium Tc-99m tetrofosmin)
- A cardiac imaging agent, is useful in the diagnosis and localization of regions of reversible myocardial ischemia in the presence or absence of infarction under exercise and rest conditions.

AMI/ACS Diagnosis

Coronary Artery CT Scanning

- Useful to rule out coronary artery disease
- Sensitive (few false negatives)
- Less specific (there are false positives)
- Can be technically difficult
- Radiation exposure
- PPV 80% range
- NPV 90% range
AMI/ACS Diagnosis

Coronary Artery MRI Scanning

- Will it replace coronary artery CT scans?
- 94% sensitive
- 82% specific
- MRI is able to detect CAD
- “Long scan time and relatively low spatial resolution have prevented its wide acceptance as a routine test for coronary artery stenosis detection”
- “Motion artifacts”

April 15, 2009 JACC Dr Qi Yang (Beijing, China)

Chest Pain Case #3: Hx

- 43 year old female
- Sharp chest pain, intermittent
- “Chest blowing up like a balloon”
- SOB and tired with exertion
- Dad died at age 45 due to AMI
- This is her third visit to EDs in week
AMI/ACS Diagnosis: Chest Pain Protocols

- Low risk population
- Serial EKGs
- Serial cardiac enzymes
- Provocative testing as needed
- Protocol detects AMI and ACS
- Identifies uncomplicated patients
The Erlanger chest pain evaluation protocol: a one-year experience with serial 12-lead ECG monitoring, two-hour delta serum marker measurements, and selective nuclear stress testing to identify and exclude acute coronary syndromes.

Fesmire F. Ann Emer Med 2002

CONCLUSION: An accelerated chest pain evaluation strategy consisting of SECG, 2-hour delta serum marker measurements, and selective nuclear stress testing in conjunction with physician judgment identifies and excludes MI and 30-day ACS during the initial evaluation of patients with chest pain.
AMI/ACS Diagnosis

**Erlanger Protocol**

- **AMI detection**
  - 93% sensitive, 94% specific
  - Positive LR 15.3
  - Negative LR 0.07
- **30-day ACS detection**
  - 99% sensitive, 87% specific
  - Positive LR 7.9
  - Negative LR 0.01

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**Cardiac Enzyme: CK-MB**

- Delta creatine kinase-MB outperforms myoglobin at two hours during the emergency department identification and exclusion of troponin positive non-ST-segment elevation acute coronary syndromes.
  - Fesmire F. Ann Emer Med 2004

**CONCLUSION:** A 2-hour delta CK-MB level outperforms myoglobin level in the early identification and exclusion of acute myocardial infarction in non-ST-segment elevation chest pain patients. This finding suggests that myoglobin may no longer be the optimal early marker of acute myocardial infarction when troponins are used as the criterion standard.
AMI/ACS Diagnosis

**Cardiac Enzyme: CK-MB**

- **AMI detection**
  - 93% sensitive, 94% specific
  - Positive LR 16.7
  - Negative LR 0.07
- Similar numbers to those seen in Erlanger protocol
- No interval CK-MB change of more than 0.7 ng/ml
AMI/ACS Diagnosis

**Cardiac Enzyme: Troponin**

- Mostly European data
- 2-6 hour chest pain evaluations
- Absolute vs. relative change noted
- Variable results
- Varied by POC test and by assay
- Sensitivity 60-95%

AMI/ACS Diagnosis

**Cardiac Enzyme: Troponin**

- Delta troponin of 20% is a reasonable cutoff
- At what time is uncertain
- Absolute change may be a better predictor of NSTEMI at the extremes (very low or high first troponin)
- Absolute change amounts vary

Test Utilization

- Likelihood ratio develops a risk assessment based on test result
  - A priori (pre test) probability
  - Post test probability
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Likelihood Ratios
- 95% sens or spec leads to LR of 0.1 or 10

Likelihood Ratios
- Negative LR = (1-sens)/spec
  - A test with 95% sens, 95% spec will yield a Negative LR of 0.05
- Positive LR = sens/(1-spec)
  - A test with 95% spec, 95% sens will yield a Positive LR of 19

Clinical Example Principles
- Pre-test assessment of AMI/ACS risk in calculated based on:
  - History and physical exam
  - Initial EKG
- Then the serial EKGs and serial cardiac enzymes are performed
- Use the LR to determine post-test AMI/ACS likelihood
Clinical Example

- Patient with chest pain is assessed to have a 10% AMI/ACS risk (an acute plaque disruption)
- Serial EKG and cardiac enzyme testing is negative for AMI/ACS
- Negative LR is 0.07
- 0.07 x 10% is 0.7% risk
- 7/1000 patients will have AMI/ACS

Clinical Example

- 0.07 x 10% is 0.7% risk
- 7/1000 patients will have AMI/ACS
- 3.5 per 500 patients with this exact presentation will have an AMI/ACS
- In order for this not to be an acceptable approach, the a priori risk for a given patient must exceed 15-20%

Acceptable Risk Assessment

- 20% a priori risk
- Negative LR 0.07
- 0.07 x 20% is 1.4% AMI/ACS risk
- This patient must be admitted for provocative testing
- But this is in a population of patients with a baseline 20% risk!
Clinical Perspective
- A population of patients with a baseline 20% risk:
  - Has a diagnosed AMI/ACS in one of every five instances
  - Would get heparinized
  - Would require coronary angiography and possibly PCI
  - Would pose a significant risk

Clinical Reality
- Most of the chest pain patients that we treat do not have a baseline 20% risk
- Most admitted chest pain patients do not rule in if the initial ED evaluation is negative
- We don't heparinize these patients
- Many don't receive provocative testing
- Most patients have no complications

ED Chest Pain Patients: Rapid Rule Out Protocol
**Rule Out Protocol Steps**

- Document the following:
  - History and physical exam not consistent with AMI/ACS
  - Negative or non-diagnostic initial EKG
  - No ongoing or interval chest pain
  - Negative or not changing 2nd EKG
  - Negative, not changing cardiac enzymes
  - No clinical indication for acute provocative testing

**Rule Out Protocol Population**

- "Low Risk" chest pain patients
- "Moderate Risk" chest pain patients??
- Not a good story, neg EKGs, neg enzymes
- No current chest pain, negative exam
- No significant risk factors
- No significant cardiac history
- You would not start heparin or other Rx

**AMI/ACS Diagnosis**

**Conclusions**

- Important clinical disease state
- Is there an obstructing lesion?
- Risk stratification and AMI/ACS diagnosis can be made
- Exclusion of acute dx possible
- Serial enzymes provide value
- Rapid ED chest pain patient evaluation is feasible, reliable
AMI/ACS Diagnosis

Questions?
- www.acc.org or www.americanheart.org
- www.acep.org
- www.sccep.org (Society of Cardiovascular Patient Care)
- edsloan@uic.edu