Bringin’ On The Heartache: Management of Acute Coronary Syndrome

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Objectives

- Define ACS
- ACS/angina sx
- Risk assessment
- Emergency management of ACS
- In-hospital management of ACS
- Post-hospital management of ACS
- LIFESTYLE!!

What is Acute Coronary Syndrome?

- AHA: “...an umbrella term used to cover any group of clinical symptoms compatible with acute myocardial ischemia.”
  - “Acute myocardial ischemia is chest pain due to insufficient blood supply to the heart muscle that results from coronary artery disease (also called coronary heart disease).”

In Plain English

- Insufficient blood flow to myocardium – most often results from CAD.
  - Compromised blood flow to viable heart muscle.
- Practically speaking: ACS means acute MI or unstable angina.

http://www.americanheart.org/presenter.jhtml?identifier=3010002
Unstable Angina

- Unstable angina:
  - Angina at rest (esp > 20 min)
  - New onset angina limiting physical activity
  - Increasing angina
    - More frequent
    - Longer duration
    - Occurs with lower exertion
- Angina that occurs early after infarction or revascularization is also considered by many to be unstable angina.

Magnitude of the Problem

- Common
  - 17 million Americans w/CAD.
  - > 1.4 million Americans get MI/year.
- Deadly
  - #1 cause of death in men, women, and adults in US.
  - 1 of every 6 deaths = d/t CAD.
- Average age @ 1st MI:
  - Men = 64.5
  - Women = 70.3

Atherosclerosis in ACS

- Unstable plaque rupture
- plt aggregation
- clotting cascade
- So, tx is based on stopping plts & thrombosis
- Plt inhibitors – ASA, clopidogrel
- Antithrombotics – heparinoids (UFH, LMWH), thrombin inhibitors

Symptoms of ACS/MI

- Chest pain/angina
- N +/- V
- Indigestion
- Dyspnea
- Sweating
- Dizziness, lightheadedness
- Fatigue
- Pain in:
  - Either arm
  - Jaw
  - Neck
  - Back
  - Abdomen

http://americanheart.org/presenter.jhtml?identifier=382482
http://circ.ahajournals.org/content/125/1/188.full.pdf+html; Circulation. 2012; 125: e2-e220; Circulation 2011;123:e18-e209; http://circ.ahajournals.org/content/123/4/e18.full.pdf

http://www.uptodate.com/online/content/topic.do?topicKey=acute_co/20093&selectedTitle=1~150&source=s
Angina

- Retrosternal chest discomfort:
  - Squeezing, pressure-like.
  - Not necessarily pain.
  - Also: heaviness, burning, choking.
- Brought on by exertion, emotional stress, cold temperature, eating.
- Relieved by rest, NTG (NOT a specific therapeutic challenge—esophageal spasm responds).

Features Suggesting MI

<table>
<thead>
<tr>
<th>Clinical feature</th>
<th>Likelihood ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain in chest or left arm</td>
<td>2.7</td>
</tr>
<tr>
<td>Chest pain radiation</td>
<td></td>
</tr>
<tr>
<td>Right shoulder</td>
<td>2.9</td>
</tr>
<tr>
<td>Left arm</td>
<td>2.3</td>
</tr>
<tr>
<td>Both left and right arm</td>
<td>7.1</td>
</tr>
<tr>
<td>Chest pain most important symptom</td>
<td>2.0</td>
</tr>
<tr>
<td>History of myocardial infarction or other vascular dz</td>
<td>1.5-3.0</td>
</tr>
<tr>
<td>Nausea or vomiting</td>
<td>1.9</td>
</tr>
<tr>
<td>Diaphoresis</td>
<td>2.0</td>
</tr>
<tr>
<td>Third heart sound (S3) on auscultation</td>
<td>3.2</td>
</tr>
<tr>
<td>Hypotension (systolic BP ≤80 mmHg)</td>
<td>3.1</td>
</tr>
<tr>
<td>Pulmonary crackles on auscultation</td>
<td>2.1</td>
</tr>
</tbody>
</table>

JAMA 1998;280:1256-63; Circulation 2002;106:3143-3421

Chance of MI in Pts W/O ST Elevation

- High likelihood:
  - Primary symptom = pain or discomfort in chest or left arm AND current pain = similar to pain of prior documented angina/MI
  - Patient w/know CAD OR MI
  - Transient mitral regurgitation OR hypotension OR pulmonary edema OR rales OR diaphoresis
  - New (or presumably new) transient ST depression (≥1 mm)
  - New T-wave inversion in multiple precordial leads
  - Elevated cardiac troponin or CK-MB levels

Circulation 2007;116:e148-e304

Chance of MI in Pts W/O ST Elevation

- Intermed: lack of high likelihood findings and:
  - Primary symptom is pain or discomfort in chest or left arm
  - Older than 70 years of age
  - Male
  - Diabetes mellitus
  - Extracardiac vascular disease
  - Fixed Q waves
  - ST depression 0.5 to 1 mm or T-wave inversion >1 mm
  - Normal cardiac troponin or CK-MB levels
**Sx Suggesting Non-Ischemic CP**

- **Pleuritic**
- Located only in mid- or low- **abdomen**
- Can be **localized** w/1 finger
- **Reproducible** by **movement** or palpation
- **Duration:**
  - Days
  - Few seconds or less
- **Radiates** above mandible or to legs

**Clinical Prediction Rules for CP**

**Vancouver CP Rule (road tested-Iran).**
- Low risk for MI (must have all):
  - Normal EKG
  - No prior ischemic CP
  - Age <40
- If ≥40 + EKG WNL:
  - No prior ischemic CP
  - Low-risk pain
  - CK-MB <3.0 OR CK-MB ≥ 3.0 BUT no EKG or biomarker chg @ 2 hr
  - 98.8% sens, 32.5% spec. Validation = 95.1% sens, 56.3% spec.

**Mayo ED**
- No new ischemic EKG changes
- No H/O CAD
- No pain typical for ACS
- Initial or 6 hr troponin < 99th percentile
- Age <50
  - 100% sens, 20.9% spec.
  - Not road tested.
- Swiss group: 85.6% sens.

**Angina in Women**

- Women are **more likely than men** to have atypical sx:
  - Pain: more intense, sharp, burning
  - Location: more often in neck, throat than men
  - Provocative factors: more likely associated with sleep, rest, mental stress
- **NB:** Women still get typical angina, too!
- **Pearl:** High index of suspicion for CAD in women with risk factors or sx.
Unrecognized MI in Women

- MI is more likely to go undetected in women, esp young women (40% unrecognized 35 – 39 yo vs 27% @ 75-79).
- Women more likely than men to have pain in neck, jaw, back, & to have nausea w/CP.
- Study of 515 women w/MI:
  - Only 30% had prodromal CP.
  - CP during MI in only 57%.
  - Dyspnea in 58%.

Atypical MI Symptoms

- 1/3 had no CP.
- Atypical sx:
  - Dyspnea alone
  - Weakness
  - Nausea and/or vomiting
  - Palpitations
  - Syncope
  - Cardiac arrest
- More likely to be older, diabetic, women.

DDx of Chest Pain

- Cardiovascular
  - Ischemic (<20-30%, but 2-4% of MI’s are missed)
  - Non-ischemic
    - Aortic dissection *
    - Myocarditis
    - Pericarditis
- Pulmonary
  - PE *
  - Tension pneumothorax *
  - PNA
  - Pleurisy/pleuritis
- Psych
  - Depression
  - Anxiety d/o’s
  - Somatoform d/o’s
  - Delusional d/o’s

Other Causes of CP

- Musculoskeletal
  - Cervical disc disease
  - Costochondritis
  - Fibromyalgia
  - Herpes zoster (before the rash)
  - Neuropathic pain
  - Rib fracture
  - Sternoclavicular arthritis

http://www.uptodate.com/online/content/image.do?imageKey=CARD%2F2500%7ECARD%2F1479
GI Causes of CP
- Biliary
  - Cholangitis
  - Cholecystitis
  - Choledocholithiasis
  - Biliary colic
- Peptic ulcer disease
  - Nonperforating
  - Perforating*
- Esophageal
  - Esophagitis
  - Spasm
  - Reflux
  - Rupture*
- Pancreatitis

Life-Threatening Causes of Chest Pain
- Acute coronary syndrome*
- Aortic dissection*
- Pulmonary embolism*
- Tension pneumothorax*
- Esophageal rupture
- Perforated peptic ulcer

Life-Threatening Causes of Chest Pain
- Dissection (aneurysm)
- Embolism (pulmonary)
- Acute coronary syndrome
- Tension PTX
- Hole in GI tract
  - Esophageal rupture
  - Perforated ulcer

Etiology of CP in Primary Care Practice
- Musculoskeletal, incl costochondritis: 36%
- Gastrointestinal: 19%
- Cardiac 16% (up to 50% in elderly)
  - Stable angina 10.5%
  - Unstable angina or MI 1.5%
  - Other cardiac 3.8%
- Psychiatric 8%
- Pulmonary 5%
- Other/unknown 16%
Remember to Look at Risk Factors!

http://www.diabetespharmacist.com/articles/obesity/

CAD Risk Factors

- Per JNC-7:
  - Age—65 F (or premature menopause), 55 M
  - BP (HTN)
  - Cigarettes (smoking)
  - DM
  - Exercise lack (physical inactivity)
  - FH—65 F, 55 M
  - Good cholesterol too low (HDL-C <40)
  - High LDL-C
  - Chronic Kidney Disease
  - Obesity (BMI > 30)

HDL-C: The Good Cholesterol

- In both JNC-7 & NCEP ATP III, HDL-C >60 is a negative risk factor – removes 1 point.

CAD Equivalents (NCEP ATP III)

- DM
- Symptomatic carotid artery dz
- AAA
- Peripheral arterial dz
- 10-yr risk of 1st event >20%
- (probably CKD—not in ATP III yet)

- Think vasculopathy! (Vasculopaths = @ risk.)

Circulation 2002;106:3143; image: digitalart via freedigitalphotos.net
More CAD Risk Factors

- **hsCRP > 3 – 4**
  - Higher CRP → higher risk at any LDL level.
  - May be most useful in those w/intermediate CAD risk to direct further eval & therapy for 1° prevention (per CDC & AHA).
  - My **caveat** FYI: most CRP studies done by Ridker, who holds patent on hsCRP assay.
- **Hot off the press (10/4/12):** 400 – 500 CV dz-free, intermed risk pts would need to be screened w/CRP to prevent 1 CV event/10 years (high NNT).

More CAD Risk Factors We Can’t Change

- **Homocysteine** (though Rx does **not** ↓ risk).
- **Coronary artery calcification (CAC)** on electron beam CT
  - Becoming less expensive.
  - Growing popularity.
  - NNT 24 for CAD, 19 for overall CV dz in highest vs lowest tertile.
  - I do not recommend for routine screening.

Likely Risk Factors We Know About But Don’t Include

- **CKD (~ risk to prior MI?)**
- **Probably microalbuminuria**
- **LVH**
- **H/O pre-eclampsia**
- **Diet:** high sat fat/low fiber/low vege + fruit
- **Sedentary lifestyle**
- **HR – resting >90 or peak too high or low**

Likely Risk Factors We Know About But Don’t Include

- **Metabolic syndrome**
- **RA, SLE, other collagen vasc dz’s**
- **Psoriasis**
- **Obstructive sleep apnea**
- **Psychosocial—depression, stress, anger**
- **Socioeconomic status**
- **Air pollution**
Additional Likely CV Risk Factors

- HIV/AIDS – dz & treatment
- NAFLD (non-alcoholic fatty liver disease)
- PTSD
- Adults who were SGA infants
- Migraine w/aura ➔ ischemic stroke in women
- Gestational DM
- Social isolation/poor relationships
- Pessimism
- (BNP, nT-pro-BNP predict MI mortality)

Risk Factor Assessment

- The ACC, AHA, and AAFP recommend that all pts undergo an assessment of CAD risk factors every 3 – 5 years.

Risk Factor Calculators

- Some options include:
  - Framingham risk score ([link](http://hp2010.nhlbihin.net/atpiii/calculator.asp?usertype=prof)).
  - ARIC risk calculator ([link](http://aricnews.net/riskcalc/html/RC1.html)).
  - Reynolds risk score (requires CRP; better validated in women than men): [link](http://www.reynoldsriskscore.org/).
  - PROCAM Risk score: [link](http://www.chd-taskforce.com/procam_interactive.html)
  - QRISK: [link](http://www.qrisk.org/index.php)
  - Just count the risk factors.
Caveat: Young Patients

- 10-year CV risk calculators **may not be appropriate for use in young pts**, who may have low 10-year risk (risk calculators may overestimate in short term), but might have greater long-term risk.


UA vs MI

- **Unstable angina:**
  - NO elevation in cardiac enzymes
  - +/- ischemic ECG changes—**transient**

- **MI:**
  - Elevated cardiac enzymes – rise & fall
  - Evolving ECG changes

- Cardiac enzymes may not rise for several hours, so UA may be indistinguishable from non-ST elevation MI at presentation.

Steps in ACS Management

- **1-2-3**
  - A-B-C.

Steps in ACS Management

1. Emergency management.
   a) Monitor.
   b) MONA.
2. EKG.
   a) STEMI ➔ reperfuse.
   b) NSTEMI ➔ generally do not reperfuse.
3. **ABC** meds.
Acute Coronary Syndrome: Step 1

- ABC’s (Airway, Breathing, Circulation) – now CAB
- ECG
- Monitor
- IV, labs
- MONA
  - Morphine
  - Oxygen (Keep $S_{p}O_2 \geq 90\%$)
  - NTG
  - ASA

Step 1a: Initial Meds

- Immediate ASA 162 – 325 mg chewed.
- CURRENT-OASIS 7 Trial:
  - No difference in outcomes or bleeding using low dose (75-100 mg) or higher dose (300-325)
  - There may be a change in the air...
  - Need rapid absorption—do NOT use EC.
  - Do not use if anaphylactic reaction.

Initial Meds—Not ASA

- Sublingual NTG 0.4 mg q 5 min X3
- Morphine 2 – 4 mg IV. Repeat prn.
  - Relieves pain, anxiety.
  - Reduces sympathetic stimulation caused by pain, anxiety

Nitrate Precautions in ACS

- Contraindicated if PDE-5 inhibitors within 24 hrs (hypotension):
  - Sildenafil (Viagra™ and Revatio™)
  - Vardenafil (Levitra™)
  - Tadalafil (Cialis™, Adcirca™) – may need to wait 36 hrs.
- Extreme caution if inferior MI & possible R ventricular involvement
  - RVMI dependent on preload to maintain cardiac output (RV not working well).
  - Give FLUIDS – ↑ neck veins are NOT due to fluid overload in this situation!
Step 2: Look at the ECG

- ST elevation
  - ST-elevation MI (STEMI)
  - Prinzmetal’s angina (transient ST ↑)
- No ST elevation
  - ST depression – angina or NSTEMI
  - T wave inversions – NSTEMI or increased risk for acute MI – includes Wellens’ syndrome

Initial ECG in ACS

- Initial ECG may be non-diagnostic in 45%, normal in 20%
- Early abnormalities include hyperacute T waves
- If initial ECG is non-diagnostic in a pt in whom there is high suspicion of MI (including continued sx), repeat ECG every 5 – 10 minutes (ACC/AHA).

Serial ECG’s

- The key to electrocardiographic diagnosis of myocardial ischemia & infarction is serial ECG’s.
  - Remember to order follow-up ECG’s.
- Even serial ECG’s are only 87% sensitive for MI – use other criteria in addition to ECG.

Diagnosis of Acute MI

- Rise & fall of cardiac biomarkers AND at least one of:
  - Ischemic sx
  - ECG changes
  - Imaging evidence of new myocardial loss or wall motion abnormality


Criteria for ST Changes

- **ST Elevation:**
  - Men:
    - ≥ 40: 2 mm in V2-V3; 1 mm in other leads.
    - < 40: 2.5 mm in V2-V3; 1 mm in others.
  - Women:
    - 1.5 mm in V2-V3; 1 mm in other leads.
- **ST depression (men & women):**
  - 0.5 mm in V2-V3, 1 mm in other leads.
  - Horizontal or downsloping ST depression of 0.5 mm in 2 contiguous leads.

A Note on ST Depression

- **Downsloping or horizontal ST depression is significant; upsloping is not.**

What is the Baseline For Comparing ST Segments?

1. A
2. B
3. C

What’s Elevation & Depression?

- **Baseline is the TP segment** – electrical neutrality.
  - T wave is ventricular repolarization.
  - P wave is atrial depolarization.
  - Between T & P is the only point in ECG when heart is not “doing” anything, electrically speaking.
Pathological Q Waves

- Any Q in leads V2-V3 ≥20 msec (1/2 box wide)
- QS complex in leads V2-V3
- Any Q wave ≥30 msec and ≥0.1 mV deep (1 box) or QS complex in leads I, II, aVL, aVF, or V4-V6
- Changes must be present in any two leads of a contiguous lead grouping.
- 1 box wide/1 box deep—easier to remember.

Where is the MI?

Extensive Anterior + Lateral

- V1 – V2: septal, anteroseptal
- V3 – V4: anterior, anteroseptal
- V4 – V6: lateral
- I, aVL, V5, V6: lateral
- II, III, aVF: inferior
  - Check V4R – V6R – RVMI (ACC/AHA)

- ST depressions in V1-V2: consider posterior MI (check V7-V9)

ECG Localization of MI: ST Elevations
Reciprocal ECG Changes

- **ST depressions**
  - Anterior MI: II, III, aVF (inferior leads).
  - Lateral MI: II, III, aVF (inferior) + V1-V2 (anterior).
  - Inferior MI: V1-V3, I, aVL (anterior, +/- lateral).

- “Inferior partners with everything.”

Inferior MI ➔ R-Sided EKG

Where is the MI?
Where is the MI?

13:01

14:32

15:37
Steps in ACS Management

1. Emergency management.
   a) Monitor.
   b) MONA.
2. EKG.
   a) STEMI ➔ reperfuse.
   b) NSTEMI ➔ generally do not reperfuse.
3. ABC meds.

More Meds in STEMI

- **β-blockers** if not contraindicated.
- IV NTG if persistent pain, CHF, HTN
  - D/C NTG if BP too low—more important to give β-blockers.
- Replete K if below 4 (2X ↑ in VF if < 3.6).

Step 3: ABC’s of MI Drugs

- ASA
- β-blockers
- Clopidogrel (esp if reperfusion)

β-blocker Contraindications

- Active bronchospasm
- Severe bradycardia
- Heart block > 1° (if no pacemaker)
- Pulmonary edema
- Hypotension with or without shock
- Overt heart failure should be brought under medical control 1st
- Most pts w/MI d/t cocaine should not be treated with beta blockers (risk of coronary artery spasm, or severe HTN)

[www.acc.org/qualityandscience/clinical/statements.htm](http://www.acc.org/qualityandscience/clinical/statements.htm)

JACC 2007;50(7):652-726

More Meds in STEMI

[http://www.uptodate.com/online/content/topic.do?topicKey=acute_co/7620&source=see_link](http://www.uptodate.com/online/content/topic.do?topicKey=acute_co/7620&source=see_link)
How & When to Give β-Blockers

- Early β-blockade is preferred.
  - IV β-blockers are recommended only for MI pts w/o contraindications and with HTN @ presentation.
  - Otherwise, oral β-blockers within 24 hrs of presentation.
- Pt w/early contraindications to β-blockers should be reassessed after 24 hrs for β-blocker appropriateness.


Meds in STEMI

- Already received ASA.
- Clopidogrel (added to ASA):
  - Benefits (↓ death, MI) in pts undergoing thrombolysis.
  - No controlled studies of clopidogrel in PCI, though cardiologists use it based on studies of PCI + TL or studies where all received PCI.


Clopidogrel Controversies

- Duration of Rx:
  - Drug-eluting stent: 12 months (?more)?.
  - Bare metal stent: 6-12 mo (?).
- High dose clopidogrel (may overcome drug resistance)
  - 300 mg load if TL or no reperfusion → 75 mg/day
  - 300 – 600 load if PCI, then 150 mg daily X 6 more days, then 75 mg daily.


New Antiplatelet Agents

- Prasugrel (Effient™)
  - In STEMI & NSTEMI treated w/PCI:
    - Reduces CV death, nonfatal MI, or nonfatal stroke (combined endpoint) vs clopidogrel.
    - Reduces stent thrombosis vs clopidogrel.
    - ↑ bleeding, esp w/CABG.
    - NNT = 45, NNH = 167.
  - Avoid in:
    - High bleeding risk, incl H/O CVA or TIA.
    - ≥ age 75
    - < 60 kg
    - Not studied in pts not receiving revascularization.

New Antiplatelet Agents – 2

- Ticagrelor (Brilinta™)
  - Binds reversibly to P2Y12 platelet receptor (clopidogrel & prasugrel irreversible).
  - Vs clopidogrel (PLATO):
    - ↓ combined endpoint (MI, stroke, CV death).
    - ↑ stroke risk, ↑ CABG-related bleeding.
    - NNT=71, NNH=143 (but low incidence of stroke).


New Antiplatelet Agents – 3

- Neither prasugrel nor ticagrelor has been studied in thrombolysis.

New Data & Why I’m Not An Early Adopter

- TRILOGY-ACS—eval prasugrel:
  - High risk pts, medical mgmt (no revascularization).
  - Prasugrel equivalent to clopidogrel.
  - Non-significant ↑ survival at 17 months w/prasugrel.
  - More expensive than generic clopidogrel.
  - Industry-supported study.
  - [Meta-analysis of dabigatran (Pradaxa™) for Afib → 33% ↑ MI (NNH = 250).]

  NEJM 2012;367:1297-1309; ArchIM 2012;172(5):397-402

New Data & Why I’m Not An Early Adopter – 2

- Primum non nocere.
  - (probably originated in mid-17th C from Thomas Sydenham).
  - Unintended consequences (SE’s).
  - More data needed on both prasugrel and ticagrelor (IMHO).

**Adverse Effects of Plt Inhibitors**

- **Bleeding**
- **Nonbleeding:**
  - Hypersensitivity (more common w/clopidogrel).
  - Neutropenia – esp w/ticlopidine, rarely used
  - Thrombotic thrombocytopenia purpura/hemolytic uremic syndrome (TTP-HUS)—esp w/ticlopidine.

**Diagnosis of Acute MI**

- Rise & fall of cardiac **biomarkers** AND at least one of:
  - Ischemic sx
  - ECG changes
  - Imaging evidence of new myocardial loss or wall motion abnormality

**Cardiac Enzymes**

- **Troponins** most sensitive & specific
- **CK-MB & myoglobin** rise first
- Low sensitivity until ≥4 – 6 hrs after sx onset
- Enzymes may not rise for 12 hrs

**Timing and Cardiac Enzymes**

<table>
<thead>
<tr>
<th>Enzymes</th>
<th>Onset</th>
<th>Peak</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myoglobin</td>
<td>1 – 4 hr</td>
<td>6 – 7 hr</td>
<td>24 hr</td>
</tr>
<tr>
<td>CK-MB</td>
<td>3 – 12 hr</td>
<td>18 – 24 hr</td>
<td>36 – 48 hr</td>
</tr>
<tr>
<td>Troponins</td>
<td>3 – 12 hr</td>
<td>18 – 24 hr</td>
<td>7 – 10 days</td>
</tr>
</tbody>
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Nonischemic Causes of Elevated Cardiac Enzymes

- CHF
- Myocarditis
- Cardiac ischemia/injury without infarction
- Rapid atrial fib
- PE
- Proximal aortic dissection
- Chronic (or acute) renal insufficiency

Look for rise & fall – not just elevation.

STEMI

- ST elevation ≥1 mm in 2 contiguous leads
  - Not due to pericarditis or LV aneurysm
- If ST elevation resolves after NTG, repeat ECG while pain-free to R/O variant angina.
- In setting of high suspicion of ACS, new LBBB should be considered STEMI.

Algorithmic Approach to STEMI
Management of STEMI

- **Job #1: Reperfusion**
  - If <12 hrs since onset of sx, PCI or thrombolysis is indicated.
  - PCI is preferred if it can be accomplished within 90 minutes.
  - If PCI not available or not within 90 min, thrombolysis is an acceptable alternative.

- Do not wait for cardiac biomarkers. Just do it!

Absolute Contraindications to Thrombolysis

- H/O any intracranial hemorrhage
- H/O ischemic stroke w/in 3 months*
- Cerebral vascular malformation or 1° or metastatic intracranial malignancy
- Sx/signs suggestive of aortic dissection
- Bleeding diathesis or active bleeding (except menses***)
- Significant closed-head or facial trauma w/in 3 months

Relative Contraindications to TL

- H/O chronic, severe, poorly controlled HTN, or uncontrolled HTN @ presentation (>180/110)
- H/O ischemic stroke >3 months previously
- Dementia
- Other intracranial disease
- Traumatic or prolonged (>10 min) CPR
- Major surgery w/in 3 weeks
Relative Contraindications – 2

- Internal bleeding w/in 2-4 weeks or active peptic ulcer
- Noncompressible vascular punctures
- Pregnancy
- Current warfarin therapy (risk of bleeding incr w/INR)
- For streptokinase or anistreplase - prior exposure (more than five days previously) or allergic reaction to these drugs

Thrombolysis Summary

- Use thrombolysis only if you have a hospital or other institutional protocol or worksheet. It’s too hard to remember all the contraindications & requirements.

More Meds in STEMI

- Heparin (per ACC/AHA, even though evidence mediocre).
  - PTT goal = 50 – 70 sec
- GP IIb/IIIa inhibitors (eptifibatide = IntegriLin, abciximab = ReoPro) prior to PCI – only if heparin used.
  - May add little to dual anti-plt therapy (ASA+clopidogrel or prasugrel or ticagrelor).
  - Evidence changing rapidly.
  - Consult cardiologist.

GP 2b/3a Inhibitors—11/9/12

- STEMI: prior to PCI & w/heparin (unless high bleeding risk)
- In NSTEMI (consult cardiologist): 
  - After angiography.
  - Troponin + ➔ UFH + clopidogrel + GP2b/3a-inhib.
  - Troponin - ➔ no GP2b/3a-inhib.
  - Maybe some others.
- Higher bleeding risk w/renal insufficiency.
- Higher bleeding risk in women & elderly.


JAMA 2006;295(13):1531-8
More on GP 2b/3a Inhibitors

- Thrombolysis:
  - No evidence to support use of these agents in pts receiving thrombolysis.
- STEMI w/o reperfusion:
  - No benefit.


Key Principle of Heparinoids

- If pt will have a procedure (e.g., CABG) which might require reversing the anticoagulant (heparin or related med), use unfractionated heparin, as it is completely reversible w/protamine.

Heparinoids in STEMI

- CABG: heparin
- STEMI w/PCI: heparin or bivalirudin
- STEMI w/thrombolysis: enoxaparin
- If reperfusion, continue anticoagulation for at least 48 hours.
- STEMI w/o reperfusion: enoxaparin, heparin, or fondaparinux
- Recommendations are changing.

http://www.uptodate.com/online/content/topic.do?topicKey=acute_co/5253&source=see_link

“PATCH” The Ailing Heart

- PCI (or thrombolysis w/protocol)
- ASA
- Thrombsis inhibitor (GPIIb/IIIa)?
- Clopidogrel
- Heparin(oids)

More Meds in STEMI

- Prior to D/C:
  - **ACEI**, esp if abnormal LVEF or uncontrolled risk factors.
  - **ARB** may be used if ACEI contraindicated or not tolerated.
  - **Statin**—high dose (atorvastatin 80 mg).
    - A**RMYDA-ACS** (industry-sponsored) ➔ early atorvastatin (12 hr before PCI) improved 30-day outcomes
    - Caution: high-dose statins associated w/new DM—but benefit > risk

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Testing After STEMI

- **LVEF**: ↓ EF ➔ ↑ mortality.
  - **Echo**—wait for recovery after reperfusion (stunned myocardium) ➔ 14 days.
- **Stress test** (guide CV rehab, eval for residual ischemia):
  - If revascularization: few wks after D/C.
  - No revascularization: pre-D/C, if no recurrent angina or CHF.

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Activity Level After STEMI

- Encourage daily activity.
- Activity guided by cardiac rehab.
- Most return to work within 2 weeks.

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Non-ST Elevation ACS

- ACS without ST elevation – unstable angina or NSTEMI
  - UA: little/no rise in cardiac enzymes
  - NSTEMI: enzymes rise—wait 4 – 12 hrs
- Risk stratification: TIMI risk score
  - High risk: may benefit from early PCI
TIMI Risk Score

- Age ≥65 years
- ≥3 risk factors for CHD (HTN, DM, dyslipidemia, smoking, + FH of early MI)
- Prior coronary stenosis of ≥ 50%
- ST segment deviation on admission ECG
- ≥ 2 anginal episodes in prior 24 hours
- Elevated serum cardiac biomarkers
- Use of ASA in prior seven days

http://www.uptodate.com/online/content/topic.do?topicKey=acute_co/20093&selectedTitle=1~150&source=search_result#H19

Reperfusion in NSTEMI

- Pts w/TIMI risk score ≥5, and maybe ≥3, may benefit from early PCI.
- NSTEMI pts w/hemodynamic instability or LV dysfunction (or sustained ventricular arrhythmias or persistent sx) may also benefit from PCI.
- Thrombolysis is NOT useful in NSTEMI, and may be harmful.

NSTEMI Management: Differences vs STEMI

- Enoxaparin instead of heparin (if no renal failure, and no CABG within 24 hr)
- No reperfusion ⇒ pre-D/C stress test (as in STEMI)
- Measure LVEF – echo (same)
- Statin (same)
- ?ACEI if EF <40%, DM, HTN (same, less evidence)
- GP2b/3a inhib depends on troponin, anticoagulant, other emerging factors.

NSTEMI Management

- Similar to STEMI except usually no reperfusion
  - If reperfusion → PCI, but not thrombolysis.
- ABC’s (stabilize)
- ASA
- ß-blocker
- Oxygen
- Morphine
- NTG
Summary of ACS Management

- **Everybody** (unless contra) gets:
  - ASA
  - β-blocker
  - Clopidogrel
  - Morphine
  - O₂
  - NTG
  - Statin
  - Stress test:
    - Pre-D/C if no reperf
    - Few weeks later if PCI

- Low EF: ACEI
- Heparinoid:
  - Poss C ABG: UFH
  - STEMI + PCI: UFH
  - STEMI + TL:
    - enoxaparin
  - STEMI w/o reperf:
    - UFH or enoxaparin
  - NSTEMI:
    - Invasive: UFH, bivalirudin
    - Non-invasive: enoxaparin, fondaparinux

Other Post-ACS Management: Secondary Prevention

- **Healthy diet** – ≥ 5 servings of vegetables & fruits/day, low saturated fat, olive oil as main fat, low in starches, high in fiber.
- Appropriate **exercise**.
- Control of risk factors.
  - ASA.
  - Statin – LDL < 70.
  - β-blocker.
  - Smoking cessation.

Lifestyle in 2° Prevention

- Mediterranean diet
  - Lyon Heart Study: 27 mo f/u → 73% ↓ in nonfatal MI, 70% ↓ overall mortality.
  - Lyon Heart Study f/u: 46 mo f/u → 72% ↓ cardiac death + nonfatal MI (combo), 56% ↓ overall mortality, 47% ↓ all endpoints combined; 61% ↓ CA!!.
  - Indo-Mediterranean Diet Heart Study:
    - ~50% ↓ total cardiac endpoints, SCD, nonfatal MI vs NCEP diet.

Mediterranean Diet – 2

- Italian study: 2.8 kg more wt loss than “prudent diet”, lower insulin resistance, almost 50% ↓ metabolic syndrome @ 2 yrs.
- Esposito et al: obese women, 2 yrs Med diet + exercise vs general info on diet/exer → BMI ↓ by 4.2 more, lower CRP in Med diet group
Mediterranean Diet – 3

- Heart Institute of Spokane Diet Intervention & Evaluation Trial: small study, Med diet = low fat, both ~70% ↓ events vs usual care.
- Greek Study of ACS (GREECS): validated diet score – closer to Med diet ➔ less severe MI (troponin), 19% ↓ recurrence

Mediterranean Diet – 4

- GISSI-Prevenzione Study: advice to increase their consumption of fish, fruit, raw and cooked vegetables and olive oil, 42 mo:
  - Those most adherent to Med diet had 49% ↓ mortality vs worst quartile.
  - Ω-3 ↓ SCD 58% in pts w/systolic HF.
  - Ω-3 save more lives than pravastatin
  - 30% ↓ CAD death/45% ↓ SCD.

Mediterranean Diet – 5

- EPIC-Elderly Study: 2700 pts >60 yo, 6.7 yrs – 20% better adherence to Med diet (standardized scale) ➔ 18% ↓ mortality
- Trichopoulou et al: 20% better adherence to Med diet ➔ 27% lower mortality in pts w/preexisting CAD.

Mediterranean Diet – 6

- Systematic review:
  - ...benefits from the Mediterranean diet were significant in all studies.... reduction in the risk of coronary heart disease varied from 8% to 45%...”
  - “The systematically reviewed studies reveal a cardio-protective effect of the Mediterranean diet and point to this dietary pattern as highly appropriate for public health objectives.”
**More Lifestyle in 2° Prevention**

- **GOSPEL Study:** 3200 p-MI pts (Italy), 3-yr intensive intervention (exercise, diet, psychosocial stress, less deterioration of body weight control) → ↓ in 2° endpoints:
  - 48% ↓ nonfatal MI.
  - 33% ↓ CV mortality + nonfatal MI and stroke
  - 36% ↓ cardiac death + nonfatal MI

**ArchIM 2008;168:2194-204**

**Meditation**

- **Hot off the press:** 201 African-American pts w/>50% stenosis on cath.
  - 20 min TM daily (after full training) vs
  - 20 min “heart-healthy behaviors”/day (exercise, relax, healthy meal, etc).
  - 5.4 yrs f/u → 48% ↓ in combined endpoint (total mortality + MI + CVA).
  - 24% ↓ 2° endpt (CV mortality + revasc + CV hospitalization).
  - SBP ↓ by 5.

**CircCVOutcomes online epub 11/13/12, CIRCOUTCOMES.112.967406**

**Meditation – 2**

- Mindfulness meditation reduces CAD adverse outcomes.
- TM mostly 1 prevention studies in high risk elderly pts w/HTN or pre-HTN, 36 mo survival:
  - TM = 100%
  - Mindfulness meditation = 87.5%
  - “Relaxation” = 65%
  - F/U 7.6 yrs (max 18.8 yrs) → 23% ↓ total mortality, 30% ↓ CV mortality.


**Meditation – 3**

- TM vs usual care, pilot study in CAD pts:
  - Less exercise-induced ischemia.
  - Better exercise tolerance.
Because I’m From Wine Country…

- *Alcohol consumption and mortality in patients with cardiovascular disease: a meta-analysis.*
  - J-shaped curve for CV mortality.
  - 22% ↓ in CV mortality – max protection at 26 g/day.
  - 18% ↓ in all-cause mortality – max protection at 5-10 g/day.
  - Conclusion: mod alcohol consumption of 5-25 g/day in pts with CV dz = assoc w/ ↓ CV & total mortality.

Primary Prevention: 5CDE

- Cessation of smoking
- Control BP
- Control Cholesterol
- Control DM
- Control weight (obesit-ee)
- Diet: ↑ fiber, veges, fruits, whole grains, Ω-3
- Exercise

Summary

- Lifestyle works!
- Primary prevention (Over 7-22 years)
  - CV Dz
  - DM
  - HTN
  - All-cause mortality
  - Cancer
  - MI
  - Stroke—total, ischemic
  - CAD
  - CHF
  - SCD
  - 70-87%
  - 91-93%
  - 78%
  - 65-69%
  - 36-70%
  - 81%
  - 50-55/71-80+
  - 59-70%
  - 52-57%
  - 92%

Are Those #’s Real?

- NEJM 2000;343:16-22
- NEJM 2001;345:790-797
- JAMA 2009;302:401-411
- NEJM 2002;346:393-403
- JAMA 2004;292:1433-1439
- Lancet 2004;364:937-952
- Lancet 2004;364:953-962
- ArchIM 2006;166:1403-9
- Circulation 2006;114:160-7
- Circ 2008;118:947-54
More on real #'s

- JAMA 2009;302:394-400
- AmJMed 2007;120:598-603
- http://www.plosmedicine.org/article/info:doi/10.1371/journal.pmed.0050012

Primary Prevention Rules!

- Primary prevention is best, when possible – prevent dz before it is evident.
- 1° prevention reduced death by 4-fold vs 2° prevention in study that synthesized data & used mathematical modelling.

Take-Home Points

- ACS definition
- Risk factors
- Management of STEMI & NSTEMI – 1-2-3, A-B-C.
- Primary care f/u.

Shameless Self-Promotion

- www.BlogTalkRadio.com/DrDaveS
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