

EVALUATION AND TREATMENT OF CHEST PAIN IN THE ED

Potential Non-ACS Cardiac Etiologies

- Aortic Stenosis (older patient with a murmur)
- Aortic Dissection (unrelenting severe CP, severe HTN)
- Pulmonary hypertension (cardiomyopathy with decompensated CHF)
- Pericarditis (pleuritic CP that is positional)
- Hypertrophic Cardiomyopathy (younger patient)

Common Non-Cardiac Etiologies (by organ system):

- Gastrointestinal—Gastric (ulcer, gastritis), Esophageal (reflux, spasm, esophagitis), Gall bladder dz
- Musculoskeletal--Costochondritis, Fibrositis, Rib fracture, Herpes zoster
- Psychogenic--Anxiety disorders, Panic disorder, Hyperventilation, Somatiform disorders
- Pulmonary--Pulmonary embolism, Pneumothorax, Pneumonia, Pleuritis

Diagnostic Tools--History and Physical, ECG, Myocardial Markers, and Imaging Techniques

ECG

Advantages

- Best initial test for chest pain patients as it is quick and inexpensive. Should be done w/in 10 min of presentation
- Provides useful diagnostic and prognostic information
 - identifies patients who will benefit from early reperfusion
 - significant ECG changes (ie, ST depression, deep T wave inversion) identify high patients

Disadvantages

- Low sensitivity-----ST elevation present in only approximately 20% of patients with MI
- Low specificity-----if criteria other than ST elevation used for estimating MI risk (particularly T wave inversion, which should be 2-3 mm or more before being considered consistent with ischemia; will improve specificity)

Alternative Methods

- Extended lead ECGs (15 or 18 lead ECG):
 - Posterior leads—useful in those who have anterior ST depression on the ECG or posterior/inferior perfusion defect on rest MPI
 - Right ventricular leads—useful only in acute inferior MI (and should be done in all inferior STEMIs), as it identifies those who have increased risk of complications (hypotension with NTG, shock, brady arrhythmias)
 - ST elevation in AvR—high specificity for multi-vessel and left main disease
- Serial or continuous ECG monitoring—should repeat ECG every 10-15 minutes when suspicion of ischemia is high

History

Predictors of MI, in order of importance:

Chest pain characteristics

- chest pain quality—key is to differentiate into typical or atypical characteristics.
 - typical—pressure, tightness, squeezing, burning, indigestion, or similar to prior MI pain
 - atypical—pinprick, pleuritic, stabbing, reproducible. Minor increase in risk for ACS

caveat—in some pts, sharp pain means severe or bad pain (typical), rather than knife-like (Atypical)
—should ask patient to define better—substantial decrease in risk for ACS
symptom duration -if it last seconds or days it is unlikely to be cardiac
caveat—stuttering chest pain or late MI presenter
response to therapy (eg, NTG) not useful in the acute setting as numerous studies have demonstrated that response does not predict chest pain etiology

History of coronary artery disease

previous MI—often useful to obtain additional information, particularly in younger patients, as many think they have an MI every time they are admitted, or when told they are being admitted to exclude an MI. Should obtain prior tests—cath, stress to better differentiate revascularization (CABG or PCI)
prior coronary angiography
prior stress testing

Age—caveat—older (ie, >70) patients more likely to have atypical presentations; SOB is the most common presentation of AMI, with CP occurring in the minority of older pts

Traditional risk factors—less important in acute setting, as ECG and H/O CAD much more important.

Number of RF rather than any particular RF is the most important predictor. Presence of DM and HTN in patients diagnosed with ACS identifies a higher risk cohort
other important risk factors

diabetes—have accelerated atherosclerosis. Common misconception that these patients present atypically (Find a study showing this and win a prize!). Patient groups with highest likelihood of atypical/silent MI are elderly and women (although atypical sx more comone, women still more likely to present with typical sx)

peripheral vascular disease—up to 50% likelihood of having concomitant sig CAD

Cocaine Chest Pain

MI risk low (<2%) but important to know to avoid beta-blockers (including labetalol) as all BB are currently contraindicated

Treatment— IV or topical NTG and calcium channel blockers (diltiazem or verapamil if EF is normal, amlodipine if EF reduced). Both are excellent coronary vasodilators. Avoid beta-blockers. Benzodiazapines may be useful if tachycardic and hypertensive

Risk Stratification Scores—currently multiple exist. Probably the most well known is the 7 point TIMI Risk Score ([+] biomarkers, ST depression, ≥ 2 episodes of CP in last 24 hrs, ASA in last week, known coronary disease, ≥ 3 traditional RF, age ≥ 65 older). Increasing score associated with higher risk. This likely works because ASA use is a surrogate for prior CAD and ≥ 2 CP episodes identifies pts with typical sx
Caveats—highest risk predictors of bad outcomes in ACS patients (renal failure and CHF) not included. ST depression and Tn (+) are standard criteria for admission, and therefore should not be counted alone.

Physical Exam (These findings are indicative of increased risk in setting of MI)

Acute CHF

New or worsening mitral regurgitation murmur

Hypotension

MARKERS

Myoglobin—no longer used on a routine basis

low molecular weight marker with early rise and fall

- rises as early as hour, peaks at 4-6 hours, and falls after 20-24 hours

b Myoglobin limitations:

Sensitivity--low if the patient presents very early or late (> 24 hrs)

Specificity—no difference between cardiac and skeletal muscle myoglobin, so increases with

muscle damage

Only cardiac marker excreted through the kidney, so increased often in renal failure

Is no longer considered a routine cardiac marker than needs to be ordered

CK-MB

- Upper range of normal 6 ng/dL
- Relative index calculated as CK-MB x 100/total, with upper limit of 4; no longer reported as has limited utility.
- Not as important now as TnI can separate cardiac and non-cardiac damage

Troponin

Structural Proteins that are Cardiac specific and highly sensitive

Prolonged elevations post MI, so CK-MB is preferred marker for diagnosing re-infarction

Analytical False Positives

hemolysis and release of red cell stroma; less common, interfering antibodies (rheumatoid factor, heterophile antibodies)

Biological False Positives

other sources of cardiac damage (myocarditis, contusion, pulmonary embolism, etc)

renal disease—rare. Should be considered secondary to ACS until proven otherwise. One way to

differentiate is if there is a rise and fall in TnI (or MB) likely secondary to ACS than myocarditis

What is a significant elevation?

0.03 to 0.09 ng/ml Negative for myocardial damage

0.01 to 0.39 ng/ml “Gray zone”. **If values increase** by 20-30% between serial samples, this strongly suggests acute injury, and is **usually consistent with ACS**. Most patients with increasing values from the first to the second sample with ACS sx should be admitted to the CCU for further evaluation.

≥0.4 ng/ml are almost always secondary to ACS

Reasons for Prolonged TnI Elevations after MI

- Although serum half-life is approximately 5 minutes, there is an initially a early release from cytosolic pool, then a later release from continued release from contractile apparatus
- Elevations present for prolonged periods, which are dependent on size of MI
 - 90% of Q wave MI had Tn elevations on day 6
 - 90% of non-Q wave MI had Tn elevations on Day 4
- Use CK-MB and TnI trends on serial sampling to determine if early or late
- No need to send further markers after CK-MB peaks (usually at 24 hours)

Patients with elevated TnI usually belong in CCU unless:

deemed to be MI > 24 hr (MB decreasing from first to second sample)

No ongoing sx

How to interpret an isolated elevated TnI

Always get a CK-MB at the same time

Repeat both markers 2-3 hours later

Possible scenarios:

CK-MB increased, and increases on repeat-----Acute MI

CK-MB increased, and decreases on repeat-----Sub-acute MI

CK-MB is low:

TnI increases between the two samples-----Subacute MI

TnI decreases between the two samples-----Late MI

No change in CK-MB between the two samples

Unclear—late MI, low grade necrosis or myocarditis

Needs to be interpreted in combination with clinical scenario

How Rapid Can MI Be Excluded?

Look at the first 2 TnI values—if increases by 20-30% between 2 samples, even if both are < 0.4 ng/ml, highly suspicious for acute MI. this will pick up > 75% of patients with MI

Can also do this with MI. Look at 0 and 3 hour CK-MB. If either elevated, or if it increases by 3 ng/ml or doubles over that time period, it has a sensitivity of 93% and specificity of 98% for MI

How Frequent should Markers be Sampled?

- No data which says need to get markers after TnI turns positive
- Convention is to send until CK or CK-MB (not TnI) peaks and begins to decrease
- For transfers in, no need to send 0, 3, 6, and 8 hrs, instead get one set for a baseline; if negative no more
- if positive see above

What is a “big MI”?

Percentile	MB, ng/mL	CK, U/L
10th	10.8	161
25th	17.8	267
50th	42.7	584
67th	94	1036
70th	107	1158
75th	137	1420
80th	180	2103
90th	300	4060

CHEST PAIN LEVELS

Level 1 – ST elevation Myocardial Infarction (STEMI)

Very High Probability of AMI and Ischemia

Diagnostic Criteria--Ischemic ST elevation, Posterior MI, or ?new LBBB with highly suggestive symptoms

Will be notified by:

STEMI pager (cardiac ALPHA Page)—go to ED as soon as possible; will be responsible for assisting in care, make sure adequate history taken, meds given prior to transporting patient to cath lab; during off hours need to wait until sufficient cath lab staff available; they will call ED to let you know

How to treat primary PCI (see medication hand-out for additional details):

Heparin--UFH

60 U/kg bolus (no more than 4000 U)

12 U/kg/hr infusion (max initial dose 1000 U/hr)

enoxaparin **NOT** used for STEMI

ASA-- 324 mg po (4 baby aspirin)

Prasugrel--60 mg load F/B 10 mg a day. Used in the ED ONLY for STEMI. **Contraindicated** in pts with: prior TIA or stroke; Age >75, weight < 60 kg [132 lbs]; high bleeding potential; severe renal failure, inability to give a history

Clopidogrel—600 mg po load (only STEMI pts; others loaded with 300 mg) F/B 75 mg q day. Used in patients with contraindications to prasugrel

Beta-Blockers not used prior to emergent PCI unless hypertensive and without evidence of CHF, or other contraindications. Usually should be initiated as po, not IV. **THIS IS A RECENT RECOMMENDATION CHANGE**

Contraindications—shock, CHF, SBP < 120 mmHg, HR >110 bpm, age >70

IV dosing—only in patients with severe HTN without contraindications listed above; otherwise, start with po (usually 12.5 mg po)

5 mg IV repeat up to 15 mg—probably should avoid in STEMI pts until EF known

PO dosing--12.5 for initial dosing for most pts; if BP high and EF ok, then can use 25 mg po

Target heart rate (ie amount of BB given) dependent on presence of CHF, size and type of MI. Contraindicated in patients with MI and active CHF, especially if EF is abnormal

If EF is low consider using carvedolol as initial treatment

Initial CCU resident’s responsibilities for STEMI patients

- When receiving “Cardiac ALPHA alert” page, respond to the ED immediately.
- On ED arrival, evaluate the patient, making sure the primary PCI checklist is complete.
- Review Cerner for prior important labs (ie, creatinine, Hgb), and prior x-rays (ie, CT reports)

- 4 Make the fellow aware of any issues that arise that would impact on the immediate treatment of the patient
- 5 Make sure that bed control and the CCU charge nurse are aware of the admission.
- 6 Stay in the ED to assist in transport of patient to the cath lab, once sufficient cath lab staff arrive (at least 2 MDs and 2 nurses). They will call when they are ready for the patient. DO NOT transport until they notify that the cath lab is ready
8. If called away by an emergency, have the intern assist in transfer

JCAHO QA indicators

Important to note specific allergies/contra-indications to: ASA, beta blockers, ACEI
Listed in allergy section of H&P form

Current quality indicators:

ASA within 24 hr and at DC,
BB at DC
EF assessment
ACEI or ARB for EF < 40%
documentation of smoking cessation counseling
Statin for patients with elevated cholesterol at DC

If a patient is not considered a candidate for a specific treatment, the medication must be linked with the treatment (For example, No ACEI/ARB due to hypotension). Chart abstractors are not to assume reasons for why a med is not given, even if it appears fairly obvious

Level 2 – Definite Unstable Angina

High Probability of ACS

Diagnostic Criteria:

Ischemic ST depression or T wave inversion
Typical sx in patient with known CAD but non-ischemic ECG
Acute Severe CHF
Abnormal MPI with MI or ongoing symptoms in level 3 or 4 pt

Treatment Strategy—Heparin (UFH or enoxaparin), B-blockers, NTG, clopidogrel, IIb/IIIa inhibitors (see handout for specific dosing recommendations)

Acute Perfusion Imaging in the ED (pertinent for level 3 and 4 patients, and some level 2 patients)

Technetium-99m sestamibi and tetrofosmin are radioisotopes that do not redistribute

Patients can be injected during symptoms and imaged after stabilization

Images will provide a “snapshot” of the blood flow at the time of injection

Information Obtained:

Myocardial perfusion
Wall motion and Wall thickening (gated images; 8 images taken during cardiac cycle put into movie format allowing for wall motion and EF assessment)
Ejection fraction

Myocardial Perfusion Imaging Interpretation

Negative for ischemia

Static images demonstrate normal perfusion, and
Gated images demonstrate normal wall motion, wall thickening, and systolic function

Or

Static images demonstrate a defect, but gated images demonstrate normal wall motion (most consistent with soft tissue attenuation), or

Images are unchanged from previous studies, or
Normal perfusion but systolic dysfunction

Abnormal (c/w myocardial ischemia)

Static images demonstrate a perfusion defect, while gated images demonstrate abnormalities in wall motion and/or thickening

If abnormal consistent with ischemia, advance to level 2 treatment protocol

Level 3 – Probable Unstable Angina

Moderate probability of MI or ischemia

Diagnostic criteria

ECG-non-ischemic

Symptoms-usually prolonged (>30 min)

typical symptoms w/o known CAD, or
atypical symptoms in pt with known CAD, or
atypical symptoms in pt with multiple risk factors

Disposition

- Observe on M10E, admitted to cardiology hospitalist--fast track protocol, or
- Observed in Orange ED
- Transferred to CCU if markers (+), or if MPI (+) and patient continues to have ongoing sx with likely ACS

Diagnostic strategy—if markers and MPI negative, then stress imaging

Level 4 – Possible Unstable Angina

Low probability of MI and low-moderate probability of unstable angina

Diagnostic criteria

ECG-non-ischemic

Symptoms

suggestive symptoms < 30 min, or
prolonged atypical symptoms, or
cocaine associated chest pain (in absence of hx/o CAD or ischemic ECG changes)

Disposition--ED evaluation, admit to CCU only if MPI positive with ongoing sx c/w ACS or (+) TnI (and advance to level 2 treatment protocol)

Role of Perfusion Imaging

Level 3—Probable Unstable Angina

If MPI is (+), Rules in ACS – early intervention

If MPI is (-), Rule out ACS – early stress testing and discharge

Level 4—Possible Unstable Angina

Rules in unsuspected ACS if (+) – prevent “missed MI”

MPI and other xray Reports

Preliminary reports are available in Cerner within about 1 hour of imaging. If not, first call nuclear medicine at 8-6828 to see if the patient has been imaged. They should also be able to give you the imaging resident’s pager number.

Considerations When Ordering Stress Testing

Make sure the patient has ruled out

Order the appropriate test (ie, exercise, persantine or dobutamine)

K should be ok (>3.5) and Hgb (>8 and not dropping acutely)

No SEALs—If a patient can have a stress test, they don’t need additional monitoring. Also there is insufficient staffing to monitor patients during the stress test, so cannot be done if require SEALs

Check Hgb (should be >10, or > 8 if chronic)

Patient needs to be able to give consent and not be DNR

IF the patient has an ICD this should be noted on the request

How to Choose which Stress Modality

Exercise

Preferred method because of additional info obtained (peak HR, BP, chest pain or not, ECG response)

Should be able to go up two flights of steps without stopping

Dipyridamole

Preferred pharmacologic method for nuclear imaging

No wheezing, asthma, bronchodilators

Dobutamine

Preferred pharmacologic method for echo

Used for nuclear imaging if unable to exercise and have contraindications to dipyridamole

Need to know if AICD in place, as target heart rate aimed for is dependent on AICD firing threshold

Criteria for Floor transfer for Patients Who Rule Out in ED (occurs rarely)

Has to be stable:
ruled out
no recurrent chest pain
considered low risk

Process:

- a. CCU resident/intern writes admission note
- b. Patient discussed with fellow and/or attending
 - c. Can be transferred to Team B
 - d. Cardiology floor team (either Team B or cardiology hospitalist) notified about patient by CCU resident
- e. Plan for further testing put into place

Limitations of Acute Imaging

- d. Can't tell the difference between
 - acute ischemia
 - acute infarction
 - old infarction
- e. Lower sensitivity when symptoms absent
- f. Need for experienced physicians for proper interpretation

Limitations of Acute Imaging Injection Without Symptoms

Sensitivity for MI unlikely to be altered by pain status
By 8-10 hours, sensitivity for MI for markers and MPI equal
Sensitivity for ischemia likely to decrease as pain free period increases
Can be evaluated using:

Subacute chest pain pathway:

send one set of markers (CK-MB, Tnl)
if positive-- admit to CCU or floor
if negative
D/C patient home or admit to floor (no need for CCU admit)
If DC'd home, schedule out-patient stress test
Page or email Mike Kontos (mckontos@vcu.edu) and with patient's name

CHF—Risk Stratification

- a. Acute versus chronic onset—if progressive PND, orthopnea, likely not acute.
- b. Severe symptom onset within 1-4 hours more likely acute. If there is a question, send one set of markers (CK-MB and TnI)
- c. When trying to decide whether to obtain myocardial perfusion imaging, ask these questions:
 - Can patient lay flat?
 - Will the patient be able to go home the next day?
 - Will the patient be able to exercise?

Acute CHF Treatment

- a. IV lasix—there is no such thing as being “lasix naïve”. Response to lasix is determined by extent of underlying renal dysfunction and EF. Initial dose dependent on renal function, EF, and should be at least 40 mg or home dose. To determine response threshold, should double dose every 2 hours if no response (ie, < 5-800 ml urine output) until 160 mg reached. If no to minimal response, next steps are to try a lasix drip, add metolazone, add nesiritide, or other vasoactive agent, or even to use a combination of all three
- b. IV NTG--Can titrate up quickly to 80-120 ug/min, depending on BP
- c. IV Nesiritide--Consider if no to minimal response to adequate IV lasix dose, or the patient is nearing intubation, but still on face mask. With bolus, wedge pressure will decrease by 20-25% within 20 minutes. To avoid hypotension, stop NTG for 10-15 minutes prior to starting. Important to confirm that patient is actually volume overloaded

Brain Natriuretic Peptide

- a. High sensitivity for diagnosing CHF
 - < 100 pg/ml- CHF not likely
 - > 800 pg/ml-CHF highly likely
 - In between--depends on clinical suspicion

Exceptions: acute CHF, mitral stenosis, burned out cardiomyopathy

- b. Other conditions also associated with increases-- Renal failure, valve problems, LVH, RV abnormalities (RVH, PE, COPD)--essentially anything that results in stretch of the left or right ventricle
- c. Optimal uses
 - Unknown if SOB secondary to CHF or not
 - Increasing Cr-?over diuresis or low cardiac output
 - Prior to discharge to see if optimally treated

Why is it Important to obtain Cholesterol and LDL at the time of admission?

LDL and total cholesterol may decrease within 12 hours of MI onset

Admission LDL thus provides better estimate of true value

Important for selecting initial statin dose, to try to get to target (< 70 mg/dL for MI patients)

We use a direct assay for LDL, which is not affected by fasting state, so LDL can be sent at the time of admission

IF using a calculated LDL, look at the TG to make sure that it is not high as result of not fasting, as this will artificially lower the LDL

Remember, AST (and to a lesser extent, ALT) are found in skeletal muscle, and will be elevated in patients with large MI. If there is a question about liver damage, can send a GGTP (specific for liver disease).

Rule of 5 and Rule of 7

- d. Initial dose of drug will lower:
 - a. Total cholesterol by: 22%
 - b. LDL by: 27%
- e. Subsequent doubling will further reduce:
 - a. Total cholesterol by: 5%
 - b. LDL by: 7%

Expected effect on LDL and total cholesterol based on choice of statin and dose

Atorvastatin	Simvastatin	Pravastatin	Rosuvastatin	TC	LDL
5 mg	10 mg	20 mg		22%	27%
10 mg	20 mg	40 mg	5 mg	27%	34%
20 mg	40 mg	80 mg	10 mg	32%	41%
40 mg	80 mg		20 mg	37%	48%

Admissions Information

- Review CCU memos and handouts for policies
- Notify CCU nurse bed control and admissions as soon as possible about admissions
- **In general, don't argue about admissions**

Determine if the question is a consult or an admission

- However, if there are questions about appropriateness of admissions can discuss with fellow or attending. If more appropriate for MRICU, should page their resident directly and give info, rather than going thru the ED. This will save time and confusion
- For any patient in whom the ED wishes to admit to the CICU who is not considered an appropriate candidate, must be discussed with the CICU attending directly before refusing
- Patients who do not need to come to CCU:
 - No chest pain in >6-8 hours
 - DNR if required treatments can be given on floor
- Can order tests and meds on ED patients, but let ED attending know. If there are disagreements, call fellow and/or attending