AMERICAN COLLEGE OF EMERGENCY PHYSICIANS
SCIENTIFIC ASSEMBLY
1999

Chest Pain: Clinical Decision Making

Patients presenting with a complaint of chest pain can provide a clinical decision making challenge. Not all of these patients must be admitted. This course reviews cardiac and noncardiac causes of chest pain. The management, documentation, and practice protocols will also be discussed, including the most recent findings from clinical research.

- Formulate a differential diagnosis for chest pain presentation.
- Develop and implement a management plan for patients with obvious cardiac chest pain.
- Identify and discuss the key elements of charting for patients with chest pain.
- Discuss the current outcomes of clinical research and its application to practice in the emergency department.

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FACULTY

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1999 ACEP Scientific Assembly

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I. Course Objectives

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Part I

A. Introduction

1. Chest pain - why do we care
   a. Patients die or become disabled
   b. Lawsuits for failure to diagnose MI
      - 10% of all paid claims
      - 20% of all malpractice dollars
2. Epidemiology
   a. 2-5% of MI patients presenting with chest pain are sent home
      - Usually young with misread EKG
   b. Only 31% of missed MI returned to same hospital
   c. Increased mortality of missed MI at 72 hours
      - 26% if sent home
      - 12% if admitted

B. Differential Diagnosis

1. Chest Wall Pain
   a) History
      - elicit history of trauma
      - pleuritic component may be present
      - often chronic symptoms
   b) Physical examination
      - usually anterior chest wall involved
      - chest wall palpation reproduces pain
      - occasionally local swelling

c) Key points
- local causes of chest pain are often overlooked
- palpation reproduces pain, not just elicits pain
- heart disease can coexist with chest wall pain

2. Epidemic Pleurodynia (Devil's grip)
   a) Epidemiology
   - acute viral infection of skeletal muscle (Coxsackie B, Echovirus)
   - occurs sporadically in epidemics
   - person to person transmission

   b) History
   - viral prodrome
   - sudden onset of sharp, catching, stabbing pain
   - paroxysmal episodes (2-10 hours length, recurrent up to 3 wks)

   c) Physical Examination
   - febrile (38 degrees - 40 degrees)
   - cutaneous hyperesthesia over involved muscles
   - pleural rub (rare)

   d) Diagnosis
   - normal EKG and CXR
   - Virus isolated from throat or stools

   e) Key points
   - initial diaphoresis and severe chest pain may mimic AMI
   - cutaneous hyperesthesia

3. Esophageal Rupture (Boerhaave's)
   a) Epidemiology
   - lower esophagus lacks serosal covering
   - after rupture only mediastinal pleura limits esophageal contents
   - pressure gradient with respirations

   b) History
   - vomiting then severe chest pain
   - pain increased with swallowing or neck movement

   c) Physical Examination
   - sudden collapse
   - appears acutely ill
   - gradual development of signs (subcutaneous emphysema, mediastinal air, Hamman's crunch, pleural fluid)

d) Diagnosis
- chest radiograph:
  mediastinal air
  pleural effusion
- widened mediastinum
- pneumothorax
- esophagram with gastrografin or dilute barium
- endoscopy can have false negative study

e) Key points
- consider the diagnosis
- physical exam signs may appear late

4. Mitral Valve Prolapse

a) Epidemiology
- redundant valve tissue and elongated chordae tendineae
- common (5% of population)

b) History
- variable symptoms (weakness, palpitations)
- chest pain in 50%:
  prolonged, sharp, nonexertional
  may mimic angina

c) Physical examination
- hallmark is midsystolic click and murmur (late systolic, holosystolic)
- murmur prolonged with valsalva, decreased with squatting

d) Diagnosis
- EKG frequently abnormal (most commonly inverted T's in II, III, AVF)
- Holter may show variety of dysrhythmias
- Echocardiogram

e) Key points
- MVP is common echo finding
- variable clinical symptoms

5. Aortic Dissection

a) Epidemiology
- males 40-70 years old
- 2000/year in United States
- intimal tear then dissection

b) History
- chest pain is universal finding (severe, tearing)
- pain radiates to back or abdomen (rare in AMI)

- pain onset is most severe (vs. crescendo for AMI)
- syncope
- neurologic symptoms

c) Physical examination
- hypertension (60%)
- dissection of hematoma (CVA, hemiplegia, pulse deficits, aortic insufficiency)
- cardiac tamponade

d) EKG (LVH, ischemia)

e) CXR - progressive mediastinal widening

f) Angiography
- considered the diagnostic "gold standard" because of its sensitivity (90-98%), specificity (95-98%) and widespread use
- allows assessment of aortic regurg and coronary arteries
- invasive, time consuming, expensive

g) CT Scan
- fair sensitivity (94%) and specificity (90%)
- noninvasive, fast, widely available
- less accurate than other studies

h) Transesophageal echo
- excellent sensitivity (97%), fair specificity (75-90%)
- noninvasive, fast, widely available
- need trained technician, may need sedation
- becoming increasingly popular

i) MRI
- very accurate with sensitivity and specificity of about 98%
- time consuming, not emergently available, difficult to perform in unstable patients

j) Diagnosis
- do one reliable imaging procedure rather than a stepwise approach
- Angio or MRI for stable patients
- TEE for stable patients
- Test of choice depends on experience/availability at your hospital

6. Pericarditis

a. Etiology
- most commonly idiopathic

- infection: viral (coxsackie and echo) bacterial (staph, strep, TB)
- malignancy (breast, lung, leukemia, lymphoma)
- systemic disease (RA, SLE, uremia, RF)
- drugs (procainamide, hydralazine, INH, methydopa)

b. History
- pleuritic pain - worse lying down, better sitting
- sharp retrosternal pain may radiate to back, neck or shoulder.
- may have pain with swallowing and with inspiration

c. Physical Examination
- may have fever if infectious etiology
- tachycardia
- signs of tamponade - Kussmaul's sign, pulsus paradoxsus
- friction rub (transient scratching sound, "leather-on-leather", heard best while leaning forward)

d. EKG
- Stage I (ST elevation and/or PR depression in I, V5,V6 and PR depression in II, AVF, V4-6)
- Stage II (ST isoelectric and T flattens)
- Stage III (Isoelectric ST and T inversion in same leads previously elevated)
- Stage IV (returns to normal)
- also low voltage or electrical alternans

e. Chest X-ray
- usually not helpful
- if old CXR is available, comparison may reveal enlarged silhouette
- may reveal other etiology (pneumonia, pneumothorax)

f. Disposition
- consider admitting all unless effusion/tamponade are ruled out
- echocardiogram is procedure of choice
- unstable patients may need pericardiocentesis

7. Spontaneous Pneumomediastinum

a. Etiology
- either negative (inhalation) or positive (valsalva) pressure causes rupture of alveous or bleb

- air tracks along vessels and bronchi and coalesces in the mediastinum
- associated with inhalational drug use, asthma, coughing, vomiting, weight lifting, parturition

b. History
- sore throat, dyspnea
- pleuritic pain worse with lying, better sitting forward

c. Physical examination
- subcutaneous emphysema
- Hamman's crunch (up to 50%)

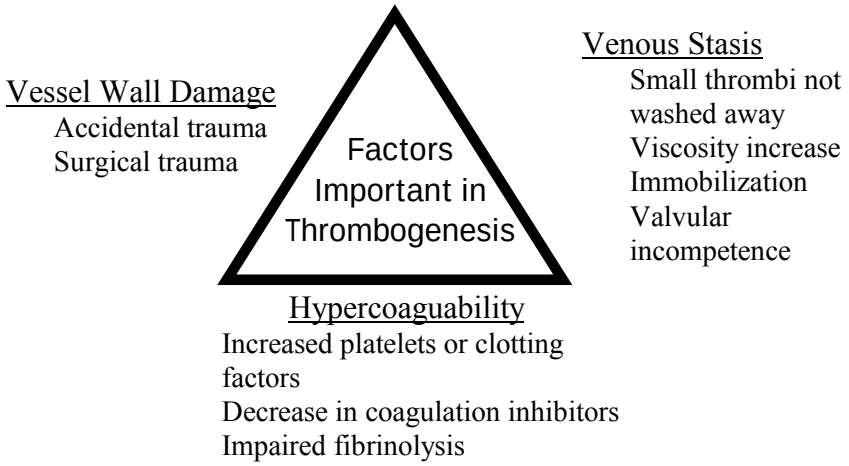
d. Diagnosis
- chest x-ray (need lateral as up to 50% missed on AP, also look for pneumothorax)
- R/O esophageal perforation
- R/O tension pneumomediastinum (not seen with idiopathic spontaneous pneumomediastinum)

e. Disposition
- classically all were admitted, recent articles support selective discharges

8. Hyperventilation
   a. Common, but must be a diagnosis of exclusion
   b. Beware the traditional treatment with a paper bag
      - Callaham documented a 1 PE and 2 AMI's treated with paper bag rebreathing based on history
      - Paper bag rebreathing with 20 volunteers (mean drop in pO_2 - 26, mean pCO_2 41)
      - Case report of paper bag being placed on a "hyperventilating" DKA patient
9. Pulmonary Embolism
   a. Epidemiology
      - third most common cause of death (200,000 annual US deaths)
      - mortality ↑ from 5% to 30% if missed in ED
      - mortality ↑ with age (2.5% at 40 yrs, 40% in elderly)
      - history, physical, EKG, ABGs are nonspecific
      - mimics other illnesses
   b. Risk Factors
      - cardiac (CHF, a fib, AMI)
      - stasis (obesity, immobilization, bedrest)
      - travel > 4 hours in any conveyance a 4-fold

- increased risk in normal patients
- smoking
- estrogen (polycystic ovaries, OCPs, etc.)
- hypercoagulability (pregnancy, post-partum, neoplasm, prior DVT, deficiencies of ATIII, protein C & protein S)
- rate increases with age

Virchow's Triad—Why Thrombosis Happens



d. History
- chest pain 80-90%
- dyspnea 75-85%
- apprehension 50-65%
- cough 40-50%
- hemoptysis 20%
- syncope 5 - 10%

e. Physical Examination
- respirations (16) 80-90%
- rales 50%
- tachycardia 40-50%
- fever (100.4) 40%
- also diaphoresis, edema, abnormal cardiac exam

f. Arterial Blood Gases
- most patients are hypoxic (average pO_2 62-72) BUT
- significant numbers of patients are not hypoxic (20% pO_2 above 80, 5% pO_2 above 90)

- most patients (~ 95%) have decreased pCO_2
- some suggest abnormal A-a gradient or decreased pCO_2 picks up 98%
- A-a gradient = 150 - (1.2 pCO_2 + pO_2)
- Normal A-a gradient = (age/4)+4

g. EKG
- 80% have abnormal EKG's but the findings are nonspecific
- sinus tachycardia is the most common finding
- right sided strain (RBBB, RAD) or ST-T changes mimicking AMI
- new onset a fib
- classic $S_1Q_3T_3$ found in less than 20% but very suggestive when present and known to be new

h. Chest X-ray
- most have abnormal CXR but the findings are nonspecific
- an elevated hemidiaphragm is the most common finding (50%)
- Hampton's hump (pleural based infiltrate pointed towards hilum)
- Westermark's sign (dilated proximal pulmonary vessels with a distal cutoff)
- pleural effusion is common

i. V/Q scan
- physician must first determine a clinical probability then the VQ scan is independently read as normal or near normal, low probability, intermediate probability, high probability
- concordant readings give an answer
- low suspicion and normal scan - 4% PE
- high suspicion and high scan - 96% PE
- discordant readings require further WU
- only 40% of PE patients have high probability scan
- 12% of low probability scans have PEs

j. Lower extremity color flow duplex
- 90% of PEs arise from peripheral venous thrombotic disease
- used to evaluate patients with nondiagnostic VQ scans

k. D-dimer
- degradation product of fibrin that indicates presence of thrombosis
- rapid latex agglutination test available
- study of 1177 suspected PE (Ginsburg 1998) found 85% sensitivity and 68% specificity.
- also helpful in stratifying risk for proximal lower extremity DVT (93% sensitivity/77% specificity, Wells 1995)
- can be used in conjunction with noninvasive studies to reduce the need for angiography

l. Angiography
- the "gold standard" - 98% sensitive
- mortality 0.1-0.5%, usually unstable patients
- consider when discordance between clinical probability and VQ reading

m. Transesophageal Echo
- sensitive for hemodynamically significant emboli:
- insensitive (13%) for small PEs
- useful for patients too unstable for VQ scan

n. CT scan
- helical and ultra fast CT becoming popular but not yet a 1st line test
- 86% sensitive for central emboli, 63% sensitive for subsegmental vessels
- difficult to use in severe dyspnea

o. MRI
- 90% sensitive for proximal emboli
- 10% unable to get adequate study4
- may be useful if absolute contraindications to angiography or in pregnancy

Part II

A. Acute Coronary Syndrome: A New Way of Thinking

We are conditioned to think in terms of the "rule-out MI". Much of the literature focuses on predicting AMI from various combinations of historical factors, EKG findings, and a seemingly endless procession of different protocols for cardiac markers—six, eight, twelve, and zero hour rule-outs, Δ-CK and Δ-myoglobin, etc. This is an attractive approach because it seeks a clean, relatively reproducible, binary answer—infarction/no infarction.

The problem with this way of thinking is that it ignores the pathophysiology of the acute coronary syndrome (ACS)—atherosclerotic plaque rupture and thrombus formation resulting in a spectrum of outcomes, from no change in coronary artery luminal diameter to complete occlusion with ST-elevation myocardial infarction. ACS is a dynamic process, with patients spontaneously moving up and down the ACS spectrum over the hours to days following a plaque fissuring event. With most of our thinking and tools geared towards detecting evidence of infarction, it is likely that a large percentage of our "missed MIs" are patients with unrecognized ACS that progress to AMI after discharge. We look only for AMI, and, finding none, send home a patient at very high risk for a significant coronary event. Our challenge is to learn not only how to identify acute MIs, but how to identify patients with a significant risk of subsequent coronary events.

B. Risk Stratification

Risk stratification requires that one have a clear answer to the question—"risk of what?" The only questions that matter in chest pain assessment are the ones that make a therapeutic difference. Some of those questions may change or become obsolete, and new questions may develop with development of new therapies. In 1999, the emergency physician has three questions to answer:

1. Does this patient need thrombolytic therapy?

2. Should this patient get anti-thrombin and/or anti-platelet agents?

If the answer to the preceding questions is NO, then

3. Can I safely send this patient home? (i.e. Distinguishing between those who are at high risk of a near-term significant coronary event and those who are not.)

A new question which we now have to answer revolves around who will should get Gp IIb/IIIa inhibitors. With the currently available literature, it seems clear that anyone with unstable angina [defined by (1) clear cut ST depression or transient ST elevation or (2) enzymatic evidence of infarction] benefits, but what about patients without definite unstable angina whom we can identify as

high risk? What treatments will benefit patients further down on the risk spectrum? Can we find a reproducible way to categorize chest pain patients by risk in order to guide workup and treatment decisions? Other questions might be institution or system dependent, like what type of bed the admitted patient should have and how soon and with whom followup should occur for a given risk level. Whatever our local practice circumstances, it is imperative that we stop thinking in terms of AMI vs. no AMI and start thinking in terms of risk of serious coronary events in our chest pain patients.

C. A Quick Review of Bayes Theorem

No discussion of risk stratification can proceed very far without bumping up against Bayesian probability. Bayes Theorem states that if we know the incidence of the disease of interest in a given population (pre-test probability), and know the outcome of a given test for the disease and its characteristics, we can calculate the post-test probability of disease from a positive test result as follows:

$$posttest\ probablity = \frac{(pretest\ prob)(sensitivity)}{(pretest\ prob)(sensitivity) + (1 - pretest\ prob)(1 - specificity)}$$

Positive and negative predictive values are the Bayesian post-test probabilities from this equation. In more familiar form:

$$PPV = \frac{TruePositives}{TruePositives + FalsePositives} \quad and \quad NPV = \frac{TrueNegatives}{TrueNegatives + FalseNegatives}$$

It is important to remember PPV and NPV vary with the pre-test probability of disease in the population in which the test is being done. PPV and NPV is how we really think in the clinical setting, but we must understand the effect of pre-test probability on these test characteristics. Sensitivity and specificity are stable characteristics of the test.

In clinical practice, one must be cautious about applying Bayes Theorem sequentially for multiple tests. Doing so is only valid if the tests are truly independent of one another.

Likelihood ratios (LR) are another means of expressing the meaning of a test result. Like sensitivity and specificity, are independent of the pre-test probability of the tested population, but are more intuitive and therefore more clinically useful to apply in practice. If LRs are not reported in a study, it is easily calculated from the following formulas:

$$LR\ of\ a\ positive\ test = \frac{Sensitivity}{1 - Specificity}$$

$$LR\ of\ a\ negative\ test = \frac{1 - Sensitivity}{Specificity}$$

To determine post-test probability using LR, first convert to odds, multiply by the LR, then convert back to probability. A clinical example:

Following a history, physical, and an EKG, a patient is felt to be at low risk (5%) for acute coronary syndrome. A resting sestamibi has a sensitivity of ~ 95% and a specificity of ~ 85%. This translates to a LR of 6.5 for a positive study and 0.06 for a negative study. Applied to the patient in question:

$$pretest\ probability = 0.05$$

$$pretest\ odds = \frac{pretest\ probability}{1 - pretest\ probability} = \frac{0.05}{0.95} = 0.053$$

$$posttest\ odds+ = pretestodds * LR+ = 0.053 * 6.5 = .344$$
$$posttest\ odds- = pretestodds * LR- = 0.053 * 0.06 = .00318$$

$$posttest\ probability = \frac{posttest\ odds}{posttest\ odds + 1}$$

So, the posttest probabilities are 25% for a positive sestamibi 0.3% for a negative sestamibi.

D. Clinical Tools for ACS Risk Stratification

1. History

   No single historical factor or combination of factors is consistently more predictive of AMI than the "gestalt" of an experienced physician. The likelihood ratios of some common historical factors as independent predictors:

Symptom	LR
Nausea or Vomiting	1.9
Diaphoresis	2.0
Pleuritic chest pain	0.2
Stabbing chest pain	0.3

These are all relatively low LRs, and as such aren't helpful in risk stratification. Other that the presence of chest pain as the chief complaint (OR=12.1), individual historical findings aren't particularly predictive.

Taken as a whole, the clinician's judgement based on history, physical, and EKG in the ACI-TIPI study (Selker 1991) showed performance characteristics of 95% sensitivity and 73% specificity for detection of coronary ischemia. In the Goldman study, clinicians had a sensitivity of 88% and a specificity of 71% for detection of AMI.

The five classic coronary artery disease risk factors of diabetes, hypertension, family history, smoking, and hypercholesterolemia convey lifetime risk of coronary artery disease. They have little or no risk stratification value for evaluating the likelihood of ACS in a chest pain patient. In a study of 5773 chest pain patients in 6 EDs, there was no significant increased risk of ACI in women from any of the risk factors, and the ORs of 2.4 and 2.1 for diabetes and family history in men were overwhelmed by the OR of 12.1 for having a chief complaint of chest pain and 8.7 and 5.3 for any ST or T abnormalities. (Jayes 1992)

2. Physical

Very helpful in the risk stratification of patients <u>with</u> AMI (i.e. the Killip classification *Am J Cardiol 1967;20:457-64*), but almost no help in risk stratifying patients with ACS. Likelihood ratios of some common physical exam findings:

Physical Finding	LR
Hypotension (SBP ≤80 mmHg)	3.1
Positional Chest Pain	0.3
Chest Pain Reproduced by Palpation	0.2-0.4
Pulmonary crackles	2.1

3. Examples of the Futility of the History and Physical Exam
   a. 23% of AMI's described as indigestion or burning pain
   b. 5-20% of AMI's described as stabbing or sharp pain
   c. 8-10% of AMI's pain fully reproduced with palpation
   d. 60% of AMI's complain of nausea
   e. Esophageal pain radiates to left arm in 11% and response to NTG in 30-50% of cases
4. EKG

   The risk stratification value of the ECG depends greatly on what criteria constitute a positive test, and what population it is being applied to. Also, most of the studies have looked only at the predictive value for MI. It is very difficult to make comparisions from paper to paper because everyone is studying a slightly different patient population and using slightly different ECG criteria. Panju attempted to draw these diverse studies together and generate LRs for specific ECG criteria. Some selected results from this paper:

Criteria	Positive LR
New Q-wave	5.3-24.8
Any Q-wave	3.9
Any ST elevation	11.2
Any ST depression	3.2
Any conduction defect	2.7
Any abnormality (Lee ,1988)	1.23

Calculating the value of a negative ECG using the "any abnormality" criteria yields an LR of 0.043, a very strong effect.

Additional right-sided leads increase the sensitivity of ECG for AMI without reducing specificity.

Some EKG test characteristics from the literature:

Reference	Test	Outcome	Sens	Spec
Lee 1985	Evidence of AMI	AMI	61%	95%
	Any abnormality	AMI	99%	23%
Zalenski 1993	ST Elevation (12 leads)	AMI	47%	93%
	ST Elevation (15 leads)	AMI	58%	93%
Lee 1989	ST elev or Q-waves not known to be old	AMI	41%	98%
	ST elev or depression, T-wave abnormalities of ischemia or strain	AMI	72%	85%

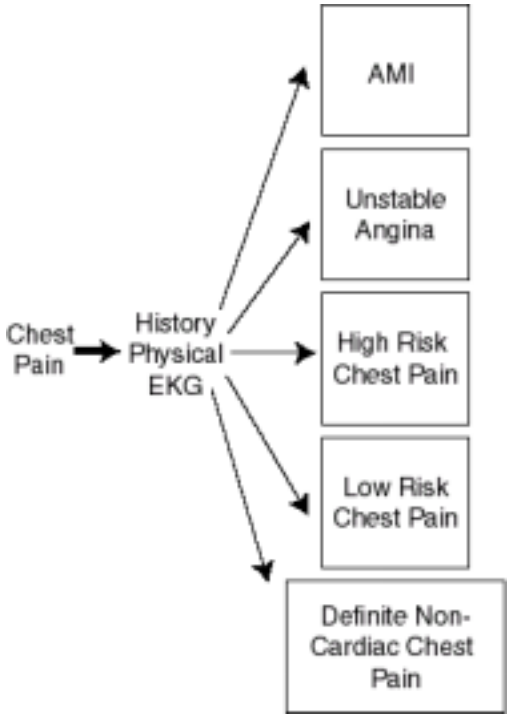
5. The Algorithmic Approaches to Risk Assessment

Clinical decision rules such as Goldman (1982) or ACI-TIPI (Selker, 1991 & 1998) combine all the information available on the initial encounter—history, physical, and EKG—and generate a probability. They have not been widely accepted in clinical practice, probably because they are somewhat cumbersome, and in the case of ACI-TIPI, have to be computer based due the complexity of the calculation. They are however, very powerful and well-validated risk stratification tools using simple and reproducible clinical criteria. Goldman was specifically designed to look at the risk of AMI, while ACI-TIPI was intended to predict the likelihood of ACS, a more clinically useful tool for the emergency physician. The addition of ACI-TIPI software to EKG machines may encourage its broader use. One potential use of these tools would be to guide the assignment of patients to quantifiable risk strata to more carefully select further testing.

6. Initial Risk Stratification

Once a risk level has been assigned based on the initial clinical data, decisions can be made regarding further testing. There are at least five easily definable groups:

1. Acute ST-elevation MI
2. Definite unstable angina
3. High risk chest pain
4. Low risk chest pain
5. Definite non-cardiac chest pain



Groups 1 and 2 are treated appropriately and admitted, and group 5 is treated appropriately for the diagnosed condition. The chest pain patients in groups 3 and 4 present the greatest diagnostic difficulty for the emergency physician.

The low risk patients of group 4 could potentially receive only one further test, with a high specificity, such as a sestamibi or two troponin I levels 6 hours apart. A

negative result would put them in such a low risk category that they would clearly be safe for discharge and outpatient followup. A positive result would merit further testing, because in this low risk population, the positive predictive value of a single test is relatively low, even with a very high specificity test

High risk patients should receive two or more tests to adequately risk stratify, for example a set of troponin Is and a treadmill or sestamibi. Patients who are not stratified to a low risk group after two or tests should be admitted for definitive workup.

5. Cardiac Enzymes
   a. WHO criteria for AMI (need 2 of 3)
      - history of characteristic chest pain
      - evolving EKG changes
      - elevation of serial cadiac enzymes (usually CK-MB used)
   b. Reasons for intense interest in newer cardiac markers that might outperform CK-MB
      - potential expanded market for thrombolytics (but at present no data for thrombolytics or angioplasty in absence of EKG changes
      - may allow identification of lower risk patients who could be admitted to telemetry rather than CCU
      - might safely allow discharge of chest pain patients from ED or chest pain unit
   c. Time course of cardiac enzymes in AMI:

	Rise(hours)	Peak(hours)	Duration
CK	4-6	12-24	4-5 days
CK-MB	4-6	12-24	2-3 days
Myogl.	1-2	4-6	1-2 days
Troponin	3-6	20	5-14 days

   d. CK-MB
      - newer monoclonal CK-MB can be performed in less than 30 minutes and measures mass rather than activity
      - Subforms of CK-MB may prove useful
      - CK-MB2 which is released from the heart is converted to CK-MB1
      - A high CK-MB2 (>1.0) or an increased MB2 - MB1 ratio may prove to be both sensitive and specific, but the patient still needs serial blood draws
   e. Myoglobin

- a low molecular weight oxygen carrying protein ubiquitous to cardiac and skeletal muscle
- monoclonal antibody and latex agglutination tests are available and can be performed in less than 10 minutes
- rapidly released from muscle, but also cleared quickly (can see stacatto pattern)
- the rapid release gives the potential to pick up AMI's earlier
- lower specificity because it is also released from skeletal muscle
- attempts to reduce false positive tests by using the myoglobin-carbonic anhydrase III ratio.
  - CAIII found only in skeletal muscle
  - Release pattern similar to myoglobin
  - High myoglobin with low CAIII implies myocardial myoglobin release

f. Troponin

- ♥ 3 subunits located on the thin filament of the contractile apparatus of the myocyte
- ♥ Troponin C binds to calcium, is not cardiospecific
- ♥ Troponin T binds troponin complex to tropomyocin, is cardiospecific
- ♥ Troponin I inhibits actinomyosin ATPase, is cardiospecific
- ♥ Both have been shown to have risk stratification value beyond just the detection of AMI.
- ♥ Both more predictive of near term events than CKMB
- ♥ Beware of the artificial cutoff values used by some labs—the troponin I of 0.35-1.0 predicts a two-fold increased risk of 42 day mortality (Antman et al., 1996). The point of care tests suffer from this problem also—always know what the cutoff value is!
- ♥ Probably detect smaller infarcts than we have previously been able to see—perhaps microinfarction from brief vessel occlusions in ACS
- ♥ Both Troponin I and Troponin T gain their full risk stratification value AFTER 6 hours from symptom onset, and remain elevated for 7-10 days.

g. Conclusions about markers

- their sensitivity and specificity changes rapidly over time in a given patient and you must appreciate the kinetics of the various markers
- may be especially helpful when serial levels are drawn in

the setting of an ED observation unit
- the conjoint use of multiple markers or multiple markers and ancillary tests (echo, ETT) may increase specificity and prove cost effective
- new markers may replace a diagnostic problem (is the pain cardiac in origin?) with a therapeutic one (should the patient with an elevated Troponin-T be thrombolysed?)

6. Ancillary Studies
   a. Imaging studies utilized
      1. Echocardiography
      2. Nuclear Medicine
      3. Exercise tolerance test
      4. Electron beam computed tomography
   b. Pertinent questions
      1. Which of these studies provide real-time, clinically useful information?
      2. How many of these studies are available to the ED physician around the clock?
   c. Stress studies
      1. Used in the stable, pain free patient
      2. Exercise is preferable if tolerated by patient (treadmill or bicycle)
      3. Pharmacologic agents
         - dipyridamole
           - direct coronary vasodilator
           - relatively long acting
         - adenosine
           - direct coronary vasodilator
           - short acting
         - dobutamine
           - the best test, precipitates true ischemic response
           - positive inotrope, moderate chronotrope
           - commonly dyspnea, ventricular ectopy
   d. Echocardiography
      1. Advantages - noninvasive, quick, portable, relatively inexpensive, early detection of wall motion abnormalities
      2. Disadvantages – reduced sensitivity in patients without ongoing chest pain, operator dependant, problems in interpretation in patients with prior MI (can't tell old from new), 10-15% of exams are inadequate due to body habitus or cooperation
      3. What can Echo tell us?
         - wall motion abnormalities
           - early finding in ischemia (usually before EKG

changes)
- sensitivity is about 90% of transmural and 60-80% for subendocardial MI
- ejection fraction
- detection of complications (aneurysms, VSD, papillary muscle rupture with mitral regurgitation, myocardial rupture, pericardial effusion, LV thrombus formation, infarct extension, RV infarction)

4. Use of Echo in AMI
   - Diagnosis: patients with ongoing chest pain, transmural MI, no prior MI
   - Prognosis: baseline for development of complications, potential discharge from CP units
   - Monitoring of response to therapeutic interventions
5. Stress Echo
   - baseline echo then stress study for new wall motion abnormality

e. Nuclear imaging studies--coronary blood flow delivers agent to the myocardium then thrombosis, spasm or scar shows up as an imaging defect
   1. Advantages
      - can identify the extent of ischemia or infarction
      - can study reperfusion (repeat study in 24 hours)
   2. Disadvantages
      - availability of personnel and tracer
      - must leave the ED
      - cost ($600 - $800)
   3. Thallium
      - analog of potassium which was previously the most commonly used agent
      - difficult to prepare the thallium
      - long half-life (73 hours)
      - since it rapidly washes out it must be given, essentially while the patient is still symptomatic
      - impractical for ED use
      - only ED study showed poor sensitivity (<70%) and specificity (<50%)
   4. Technetium
      - Two commercially available perfusion agents: sestamibi (Cardiolite) and tetrafosmin (Myoview)
      - has many advantages over thallium for ED use:
        - taken up by myocardium with a very slow redistribution allowing agent to be injected at time of initial evaluation/stabilization with imaging

done 1-4 hours later
- shorter half-life (6 hours) allows larger dose than thallium getting higher quality image
- higher energy emitter=less scatter and better images
- high photon flux allows for gating and assessment of ejection fraction and wall motion abnormalities

- excellent sensitivity (90-95%) and specificity (80-85%)
- Negative scan highly predictive of lack of near-term significant coronary events
- Tatum, Ann Emerg Med 1997
  - 438 low or moderate risk patients injected in ED
  - Sensitivity for AMI within 30 days 100%, specificity 78%
  - 0.9% normals had event between one and twelve months, 13% of abnormals
  - 2/58 AMIs were initial placed in low risk group and were only identified on the sestamibi scan. This is consistent with the missed MI rate reported in the literature (Lee 1987)
- Cost effectiveness studies suggest ~$1000 savings over an admit & rule out strategy.

5. Immediate exercise tolerance testing
   - selected patients at low risk for AMI can have immediate ETT done to identify patients who don't require admission
   - no prior history of coronary artery disease
   - normal initial cardiac enzymes
   - EKG normal or minor nonspecific ST-T changes
6. Electron Beam Co mputed Tomgraphy
   - 100 msec acquisition time gated to cardiac cycle eliminates motion artifact
   - Detects coronary artery calcifications
   - Lack of calcium = no angiographically detectable coronary narrowing
   - Potential advantages: fast, very high negative predictive value, relatively cheap (~$500)
   - Disadvantage: Not widely available
   - Only study in ED to date (Laudon et al, Ann Emerg Med 1999;33:15-21)
     - 100 chest pain patients at low to moderate risk

	Cardiac Eval +	Cardiac Eval-
EBCT +	14	32

EBCT-	0	54

B. Special Groups

1. Elderly
   a. With increasing age the chief complaint of AMI frequently becomes weakness, confusion or syncope rather than classical chest pains.
   b. Diagnostic enzyme changes occur less commonly due to decreased muscle mass and underlying diseases.
   c. Mortality in elderly with AMI higher than in younger patients (32% at 75 years, 5% if younger than 55 years)
2. Cocaine induced chest pain
   a. Pathophysiology
      - exact cause unknown, several theories
      - vasospasm has been shown on angio in several studies and 45% of patients have a normal angiogram
      - damaged coronary arteries subjected to increased myocardial oxygen demand
      - enhanced thrombogenesis and platelet aggregation
   b. Epidemiology
      - a disease of young (average age 33) males (70-90%)
      - incidence of AMI parallels incidence that route is used (60% nasal, 30% crack, 10% IV)
      - first reported case was 1982
      - 2 prospective studies show the incidence of AMI in admitted patients to be 6%, other studies give range of 0-31%
   c. History and physical
      - 67% develop chest pain within 3 hours of cocaine use
      - (range 1 minute - 4 days)
      - ask about cocaine use in the young male with chest pain (17% positive drug screens in one study)
      - 58% present with normal vital signs
   d. Diagnosis
      - EKG usually normal or nondiagnostic (complicated by early repolarization)
      - enzymes may be confusing since 50% of cocaine users without chest pain have elevated CK (Troponin - T may prove to be more specific)
      - echo might be helpful in the young patient with persistent pain
   e. Prognosis
      - very few complications due to young age of patients

- low recurrence of symptoms if cocaine use is stopped

f. Treatment
- use standard treatment modalities of aspirin, oxygen and nitrates
- benzodiazepines should be used in hypersympathetic patients to decrease oxygen demand
- calcium channel blockers can be considered if pain persists after above methods used
- thrombolytic use is controversial, should be considered if the suspicion of AMI is high (but remember these patients have low mortality anyway)
- avoid beta blockers since they will result in an unopposed alpha effect
- "caines" (lidocaine, procainamide) are not contraindicated
- patients without EKG changes are good candidates for observation units

3. Women and heart disease
   a. An unacknowledged epidemic
      1. AMI is not just a male disease
      2. 1/3 of all US deaths in women are from heart disease
      3. Annual United States deaths in women
         500,000 heart disease
         245,000 AMI
         227,000 cancer
          90,000 stroke
          46,000 lung cancer
          42,000 breast cancer
   b. How ACS differs in women
   c. Women presenting with AMI are:
      1. Older than men and more likely to be diabetic and hypertensive
      2. Less likely have the classic chest pain pattern and more likely to have nausea, jaw pain, and dyspnea
      3. Higher in-hospital mortality than men
      4. Less likely to die out-of-hospital
      5. Have less ST deviation for a given degree of ischemia (J Electrodiol 1994;27 Suppl:42-45.)
      6. More likely to have bradydysrhythmias (JACC 1998;31:301-306)
      7. Less likely to get beta-blockers during an AMI
      8. Less likely to get an acute revisualization strategy
      9. Die less often from dysrhythmia and are more likely to have a cardiac rupture

d. Increased relative risk (RR) subgroups
   1. polycystic ovary 7.4 RR
   2. irregular menses 1.8 RR
   3. early first pregnancy (<20 years) 2.3 RR
   4. short stature (<59 inches) 1.5 RR
   5. heavy smoker with toxemia history 41 RR

Part III. Disposition

A. CCU vs. Telemetry

1. CCU admission is indicated for all high risk patients
   a. ongoing chest pain
   b. new EKG findings
   c. abnormal vital signs
   d. rales on physical examination

B. ED Observation

1. Goal
   a. Opt out of the "damned if you do--damned if you don't" chest pain scenario
   b. Decrease unnecessary costly admissions - $3-$6 Billion/year
   c. Avoid inappropriate discharge of ACS patients with its morbidity/liability
2. Protocols required
   d. Entry criteria - low to intermediate risk of MI, evolving to include all patients without enzymatic or ECG evidence of ischemia
   e. Coordinated support
      1. hospital administration
      2. nursing
      3. cardiology
      4. emergency medicine

C. Transfers

1. Hospitals unable to perform angioplasty and emergency cardiac surgery should have prearranged referral plans
2. The receiving hospital should have special skills, services and equipment not available at the sending hospital
3. Patients should be considered for tertiary referral if evidence of continuing ischemia of viable myocardium after thrombolysis
4. Patients being transferred out for acute intervention should be considered for low molecular weight heparin, a glycoprotein IIb/IIIa inhibitor, or both, in consultation with the receiving cardiologist.
5. Always remember, the transferring physician is responsible for making sure the transfer is medical appropriate and that the

patient has been stabilized within the capabilities of the transferring hospital.

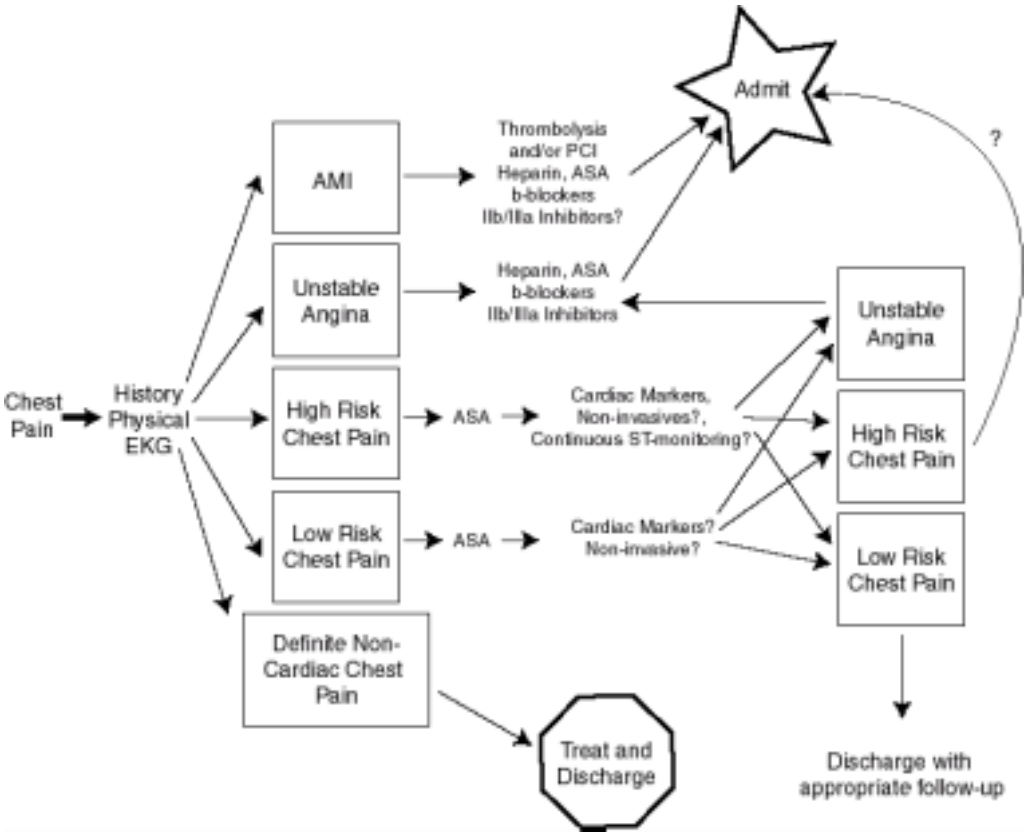
D. Avoiding Litigation in the Chest Pain Patient

1. Communication
   a. Discuss results with patients and family
   b. Emphasize the uncertainty of the workup
   c. Identify and resolve unmet expectations
2. Documentation
   a. Historical factors
   b. Decision making process
   c. AMA patients require especially detailed notes
   d. Dictated charts provide more complete and legible documentation
3. Knowledge
   a. Atypical presentations may represent AMI
   b. Normal EKG and initial enzymes doesn't exclude AMI

E. Conclusions

1. The patient who comes to the ED with chest pain presents a high medicolegal risk in an era of cost containment
2. A careful history and physical exam along with some simple diagnostic studies can allow you to make a number of noncardiac diagnoses in the patient with chest pain
3. It is becoming possible to safely reduce the number of CCU admissions using:
   a. newer cardiac markers
   b. ancillary tests
   c. shorter stays in observation units with well thought out, complete evaluation protocols

Putting it All Together



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