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Episode # 015 - ACS

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- **Canadian AMI miss rate** is 0.8-8% depending on practice location
- **ED differential diagnosis for immediately life-threatening causes of CP**: ACS, PE, Pericarditis w/ tamponade, Tenesion Pneumothorax, Aortic Dissction, Esophageal Rupture (Boerhaave's Syndrome)

Atypical Presentations

- 1/3 of pts with ACS have no CP and **risk factors for No CP**: elderly, women, DM, Hx of CVA or CHF

- **most frequent anginal equivalents**: SOB>Weakness>Unusual Fatigue>Sweats>Dizziness

- therefore, elderly & diabetic pts with SOB should get ECG within 10min of arrival in ED just like CP pts

- Assoicated symptoms most predictive of MI: ED observed sweating>vomiting>radiation to both arms>radiation to R arm

- Radiation to the L arm did not increase the likelihood of MI in recent study in *Resuscitation,* 2010

- 7% of pts with ACS will have pleuritic chest pain & 7% of pts with ACS have their pain partially or fully reproduced on chest wall palpation

Conclusion: "Atypical is Typical" ie. Atypical presentations of ACS are common

Diagnostic Value of ED Meds for ACS

- 'Pink Lady' (Maalox & Lidocaine mixture) improving chest pain has no predictive value in ACS

- GERD is the most common misdiagnosis of missed MI (20% of pts describe their pain as "burning" or "heartburn", 8% of MI begin while eating, and 15% of pts with MI will respond to 'Pink Lady')

-Response to Nitroglycerin has no predictive value in ACS

ACS Risk Factors

- In the ED, traditional cardiac risk factors are poor predictors of ACS in pts >40y/o: the only traditional risk factors that ***predict*** ACS in the ED patient with CP are DM & Family History of premature MI in male patients (not obesity, smoking, hypertension, hyperlipidemia)

- pts < 40y/o in whom the absence of all traditional risk factors makes the pre-test probability of ACS very low and the presence of >4 risk factors, does help predict ACS

- Important Non-traditional Risk Factors (esp in young pts): Pregnancy, CRF, HIV (esp if on protease inhibitors), Cocaine/Amphetamine use, chronic steroid use, Lupus (50x risk from Framingham data)

Diagnostic Utility of Recent Cardiac Testing for ED pts with CP

- up to 2/3 of AMIs caused by stenotic plaques <50% occlusive, so that tests based on stenosis can be misleading

- treadmill stress test has only 68% sensitivity and 77% specificity for single vessel disease

- a recent "negative" angiogram does ***not*** rule out ACS in the ED pt

Value of Physical Examination in CP Patients

1. Exclude other diagnoses: pulse deficit, new aortic regurg murmur and neuro deficit in aortic dissection,
   unilateral decreased air entry and JVD in tension pneumothorax
   friction rub, muffled heart sound and JVD in pericarditis with tamponade
2. Prognostication: look for signs of AHF (JVD, HJR, S3, crackles and peripheral edema) as worse Px
   aortic stenosis murmur (critical AS with ischemia carries very poor prognosis)
   new mitral regurg murmur (ominous sign of papillary muscle rupture/flail leaflet)

ACS ECG Pearls from our experts

- 7% of pts with MI will have a normal ECG & 1/3 of pts with MI will have 'non-specific changes'

- elderly pts more likely to have LBBB, LVH and non-diagnostic ECG

- Indications for 15 lead ECG: All inferior STEMI to rule out RV infarct, ST depression in V1/V2 with or without tall R wave in V1 to rule out Posterior MI

  - Some experts extend indications to any pts with non-specific changes (literature is mixed)

- Differentiating the ECG of Pericarditis from AMI: ST elevation Lead ll > Lead lll in pericarditis and vice versa in MI, diffuse concave (as apposed to convex in MI) STE without reciprocal changes, PR depression in lead ll + V6 and PR elevation in aVR, maintain T wave morphology (as apposed obliteration of T wave morphology in ACS), ST elevation in Lead l **and** Lead ll at the same time

LBBB & ACS

- new LBBB in the setting of a good clinical story for MI is an indication for re-perfusion therapy according to the AHA guidelines, however only 1/3 of these pts will rule in for MI, and only a small minority of pts will fulfill Sgarbosa's criteria making the diagnosis of MI more likely

- Sgarbosa's criteria that make MI more likely in the setting of LBBB and Ventricular-Paced Rhythm

  1. STE 1mm or more and concordant (in the same direction as the main deflection) with the QRS
  2. STD 1mm or more in leads V1 V2 or V3
  3. STE 5mm or more and discordant (in the opposite direction) with the QRS complex

- new RBBB has worse prognosis than new LBBB in the setting of ACS

- pts with CP or CP equivalent and new LBBB ***or old LBBB*** both have ***same rate of MI (***Kontos MC, et al. Outcomes in patients with chronicity of left bundle-branch block with possible acute myocardial infarction. *Am Heart J.* 2011;161(4):698-704)

aVR ST Elevation

- STE in aVR > STE in V1 with ST diffuse ST depression in other leads suggests Left Main or Triple Vessel Disease in the setting of ACS which carries a poor prognosis

- also consider Pulmonary Embolism, WPW in CP pts with STE in aVR

- while STE in aVR is not part of most STEMI protocols for transfer for immediate PCI, our experts believe that pts with STE in aVR and ACS should be considered very high risk and transferred immediately for PCI via a STEMI protocol

Right Ventricular Infarction

- Clinical diagnosis: triad of elevated JVP, clear chest and low BP + 15 lead shows V4R elevation on ECG

- preload dependent, so avoid nitrates, morphine and B-blockers and consider fluid bolus

Cardiac Biomarkers

- **When to use CK-MB**: CK-MB returns to baseline in 2-4days vs Troponin in 1-2wks so if pt presents with CP 5 days after being discharged from hospital with MI, the CK-MB is helpful to determine whether the Troponin rise is from the MI that occured 5 days ago vs. pt has re-infarcted today

- 2 sets of Troponins (6-8hrs apart) are indicated for all pts (except those that have isolated CP >12hrs prior to ED visit and are symptom free for 12hrs prior to their ED visit, who only need 1 Troponin)

- always repeat a detectable Troponin leak even if below the cut-off level for MI

- new 4th generation ultra-sensitive Troponins turn positive sooner than older Troponins and a 3hr & 6hr post CP ultra-sensitive Troponin has a near 100% sensitivity to rule out MI in low risk patients, however they are less specific than the traditional Troponins (N Eng Jour Med, Aug 27th, 2009)

- Troponin levels reflect increased risk of death/MI regardless of renal dysfunction (N Engl J Med 2002; 346:2047-2052)

- adverse outcomes increase with increased Troponin even at values below the cut-off regardless of cause

- differential of elevated Troponin: PE, myocarditis, sepsis, post-cardioversion, CHF, RF

Medications in ACS

1. **Oxygen** indicated only if pt 1. SOB 2. signs of AHF 3. Shock 4. O2sat<94% (AHA guidelines)

   - hyperoxia causes decreased coronary blood flow, so high flow O2 is ***not*** indicated if O2sat>94%

2. **ASA** 160-320mg chewed: NNT=19 saves one life at 30 days

3. **Nitroglycerin:** never shown to decrease mortality

   - indicated in pts with CHF with high BP, cocaine associated ischemia, failure to reperfuse after lytics

4. **Morphine**: helps to dampen pt's sympathetic response and decrease preload but may increase mortality in NSTEMI pts (CRUSADE study - number needed to harm = 125)

5. **IV B-blockers**: AHA guidelines advises against routine use in MI because increases incidence of cardiogenic shock in pts with 1. AHF 2. age >70 3. SPB<120 4. HR>110 or <60 5. increased time since onset of STEMI symptoms, but our experts believe their may be a role in pts who are hypertensive in the absence of the above listed risk factors and other C/Is (cocaine induced ischemia, Inferior MI with RV extension, large Anterior MI, reactive airways etc)

   - older studies show NNT=31 to save one life at 90 days

6. **Clopidogrel**:

- in STEMI with Fibrinolysis, NNT = 15 for recurrent MI or death (CLARITY), dose: <75y/o 300mg, >75y/o 75mg

- in STEMI with PCI, dose: <75y/o 600mg (OASIS-7), >75y/o consider 300mg

- in NSTEMI or UA with ischemic ECG changes NNT 47 for CV death, nonfatal MI, stroke (CURE) dose: <75y/o 300mg, >75y/o consider 75mg

Prasugrel & Ticagrelor – newer faster acting platelet inhibitor; compared to Clopidogrel may reduce ischemic events but have higher risk of bleeding complications

7. Anticoagulants: Unfractionated Heparin (UFH), Low Molecular Weight Heaprin (LMWH eg. Enoxaparin), & Fondaparinux prevent infarct related re-thrombosis, indicated in MI and UA with any ischemic ECG changes or positive biomarkers

- STEMI pts going for PCI: IV UFH 60IU/kg preferred by interventional cardiologists

- STEMI with lytics: LMWH and Fondaprinux preferred over UFH

- NSTEMI & UA: LMWH or Fondaprinux preferred over UFH

- Fondaprinux may have lower risk of major and minor bleeding complications compared to LMWH

<u>Management of Cocaine-induced Ischemia</u>

- patients who are chronic cocaine users are more likely to have atherosclerotic dz and acute cocaine use causes coronary vasospasm

- mainstays of medical therapy are benzodiapines and nitrates

- PCI is the re-perfusion treatment of choice in cocaine-related ishcemia

<u>Fibrinolysis vs PCI for STEMI</u>

Fibrinolysis is generally preferred if: early presentation (<3hrs from symptom onset) AND door-to-balloon time >90mins OR door-to-balloon time minus door-to-needle time >1hr, AND no C/I to fibrinolysis

PCI is generally preferred if: late presentation (>3hrs from symptom onset), door-to-balloon time <90mins door-to-balloon time minus door-to-needle time <1hr, C/I to fibrinolysis, high risk STEMI (AHF, Killip class 3+), diagnosis of STEMI in doubt

- additionally, PCI is generally preferred for cocaine-induced STEMI and pts with LBBB who do not fulfill Sgarbosa's criteria, as there is uncertainty of the diagnosis of MI

- risk of IC bleed with fibrinolysis ranges from 0.25% to 2.5% depending on: age >65, weight <70kg, HTN in ED >180/110

- Absolute C/Is to fibrinolysis in STEMI: prior ICH, known structural cerebral vascular lesion, known malignant intracranial neoplasm, ischemic stroke within 3 months, suspected aortic dissction, active bleeding or bleeding diathesis (excluding menses), significant closed-head trauma or facial trauma within 3 months

- for Emergency Departments located a few hours from a PCI centre, a 'pharmaco-invasive strategy' (facilitated PCI) can be considered according to the TRANSFER-AMI study in which high risk STEMI pts received standard-dose tenecteplase, ASA and either UFH or Enoxaparin and Clopidogrel. Pts were randomized to either standard treatment (including rescue PCI, if required, or delayed angiography) or a strategy of immediate transfer to a PCI centre within 6 hours after fibrinolysis. Pts in the 'pharmaco-invasive strategy' group had fewer ischemic complications, but there was no mortality benefit.

Disposition Decisions in Low Risk Chest Pain Patients

- while the risk of a cardiac event or death in 30 days is <1-2% after a a negative ED cardiac work-up (normal serial ECGs and 2 sets of normal cardiac biomarkers), it is not zero

-the decision to admit a pt who is at low risk for ACS after a negative ED cardiac work-up for invasive testing must be weighed against the potential complications of invasive cardiac testing (radiation exposure, bleeding etc)

-Jeffery Kline (of PERC rule fame) determined with computer modelling that with a pretest probability of ACS less than or equal to 2%, the risk of testing will exceed its benefits; this has yet to be validated

- there are no chest pain decision rules that have a low enough acceptable miss rate to be used clinically in the ED as the sole means for making disposition decisions

- TIMI score: 7 point score based on age>65, 3+ CVS Risk Factors, prior coronary stenosis >50%, ST deviation on ECG >0.5mm, 2+ anginal equivalents in 24hr, ASA use in the last 7 days, elevated cardiac biomarkers

-originally developed for use in pts with UA or NSTEMI as apposed to the undifferentiated ED CP pt

-CMAJ, 2010. *Diagnostic accuracy of the TIMI risk score in patients with chest pain in the emergency department: a meta-analysis.* Conclusion: "Although the TIMI risk score is an effective risk stratification tool for pts in the ED with potential ACS, it should not be used as the sole means of determining patient disposition"

- pts who are discharged from the ED who are considered to be low risk for ACS and require further cardiac testing, should have that test performed and interpreted within 48-72hrs

- ideally low risk CP pts should have a stress test **before** discharge

- treadmill stress test, while having poor sensitivity and specificity is inexpensive and easily accessible compared to nuclear testing, stress Echo and CT coronary angiography

- CT coronary angiography positives: excellent negative predictive value, identifies other diseases, shows vessel wall abnormalities (not just stenosis) which can lead to unstable plaques, ideal for moderate risk pts (>10% ACS risk)

- CT coronary angiography negatives: poor specificity with many false positives in low risk patients, radiation exposure

- Clinical Decision Units or Chest Pain Observation Units have been shown to reduce length of stay, hospital admissions, diagnostic accuracy and healthcare costs but have never been shown to reduce adverse cardiovascular outcomes, particularly mortality