Chest pain pathways and the Acute Medicine Assessment Units

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Non cardiac chest pain is a common presenting complaint

- In the UK, chest pain is responsible for roughly 1 million visits to emergency departments each year
- 10-25% are due to cardiac ischemia
- In general practice, proportion is even lower
- Problem is of identifying patients with
  - acute coronary syndromes (incl STEMI)
  - vs stable anginal chest pain
  - vs non-cardiac chest pain

Stable Angina versus Acute Coronary Syndrome

Stable angina
- Predictable symptoms, on set level of exertion
- No pain at rest
- ECG normal at rest (or old changes)
- No elevation in cardiac troponin

ACS
- Rapidly worsening symptoms
- Pain lasting more than 20 minutes
- Pain at rest
- Dynamic ECG changes
- Raised cardiac troponin
High-sensitivity troponin

- Cardiac enzyme detectable in serum which indicates myocardial cell damage
- High precision of troponin levels at very low concentrations
- Need to be aware of the local values for 99th centile
- Potential for "rule out" clinical algorithms

6 h/3 h rule-out algorithm of non-ST-elevation acute coronary syndromes using high-sensitivity cardiac troponin assays.

Criteria for Acute Myocardial Infarction

- Detection of a rise and/or fall of cardiac biomarker values (preferably cardiac troponin) with at least one value above the 99th percentile upper reference limit (URL) and with at least one of the following:
  - Ischaemic symptoms;
  - ECG changes of new ischaemia (new ST-T changes or new LBBB);
  - Development of pathologic Q waves in the ECG;
  - Imaging evidence of new loss of viable myocardium or new regional wall motion abnormalities;
  - Identification of an intracoronary thrombus by angiography or autopsy.

Cumulative incidence of myocardial infarction or cardiac death in patients with troponin concentrations less than the 99th centile Patients without index myocardial infarction were stratified into two groups based on the troponin concentration

- Musculoskeletal
- Costochondritis
- Xiphodynia
- Tietze’s syndrome
- Bornholm disease ("Devil’s grip", "Grip of the Phantom")
- "Precordial catch"
- Fibromyalgia
- Mastalgia
- Herpes zoster
- Somatization / chronic pain syndromes

"If it’s not my heart, what is it doctor?"

- Other cardiac causes – pericarditis, valvular heart disease, dissection
- GORD / oesophagitis / gastritis
- Pleuritic chest pain: LRTI, PE, Trauma,
- Cholezystitis, liver abscess

Using cardiac troponin as a first step "rule-out"

- 4870 patients enrolled with suspected ACS in EDs
- In this derivation cohort, a hsTnI< 5ng/l had a negative predictive value for type 1 MI or cardiac death at 30 days of 99·6% (95% CI 99·3–99·8)
- Replicated in the validation cohort
- Valid across risk factor status and previous CHD
- Not as clear cut if there is an ischemic ECG
- If pain <= 2hours ago, consider repeat
Back to the diagnostic conundrum...

- Problem of false negatives: up to 25-30% of patients with ACS may have a normal ECG at entry
- Acceptable “miss rate” deemed at <1% (survey of 1029 ED physicians)
- Using history, examination, ECG & initial troponin
- HEART / TIMI / Grace risk scores...
- Chest pain presentations as an opportunity

Chest pain assessment units

- Ring-fenced area with admission criteria for chest pain assessment and investigational protocols
- CPAUs were mostly instituted before the high sensitivity troponin protocols
- Safe & do not increase cardiac events in intermediate risk patients
- However admission criteria can be breached (NZ – 73% didn’t reach criteria) and further functional testing can be performed unnecessarily
- Treadmill tests are widely used for diagnosis: but sensitivity for obstructive CAD is only 61-68%
Summary

- History of the nature of the chest pain is key
- Early rule-out Cardiac troponin algorithms allow rapid risk stratification of patients with chest pain presentations
- Patients with stable chest pain presentations with risk factors for CAD will require further testing: consider CT CA +/- functional testing
- AMAUs with access to diagnostic pathways may provide a new route for chest pain patient assessments