HeFSSA Practitioners Program 2014

- 08:00 – 08:30  Registration
- 08:30 – 09:15  Clinical Case Presentation 1
- 09:15 – 10:00  Clinical Case Presentation 2
- 10:00 – 10:30  Tea Break
- 10:30 – 11:15  Clinical Case Presentation 3
- 11:15 – 11:45  Clinical Case Presentation 4
- 11:45 – 12:00  Questionnaire
- 12:00 – 14:00  Lunch
• Elderly lady
• Intermittent chest pain over few days
• Associated
  – Shortness of breath
  – Fatigue
• Syncope x 1
• Presents to Casualty 00h30
• Past history
  – Hypertension
  – Dyslipidaemia
    • Refused statins – too old

• Returned from Australia 36 hours ago
CHEST PAIN

• Coronary artery disease
• Pneumonia
• Pulmonary embolus
• Aortic dissection
• Pericarditis
• Reflux
• Ulcer

• Costochondritis
• Intercostal neuralgia
• Muscle strain
• Fracture
• Bony pathology – met
• Could this be stress, Dr?
• Intermittent chest pain over a few days
  – Localized or diffuse
  – Duration
  – Central or lateral
  – Burning, sharp, crushing
  – Aggravated by movement or breathing
  – Aggravated by exertion
  – Rest or on exertion
  – Supine or recumbent
  – Cough – productive (of what)
CHEST PAIN

• Examination
  – BP - high or low or normal
  – Pulse – fast, slow, normal, weak, absent
  – Respiratory rate
  – Temperature
  – Crackles
  – Bronchial breathing
  – Localized tenderness
CHEST PAIN

• Investigations
  – ECG
  – CXR
  – Bloods
    • FBC
    • U + E + Cr (always do!)
    • D Dimer
    • Troponin / enzymes
  – (Echocardiography, Spiral CT, V/Q scan, CT brain)
Coronary? Pulm embolus? Myopericarditis? Dissection?
Not our lady, but a man with shortness of breath, similar circumstances
RESULTS

• ECG – normal
• CXR – pulm congestion
• Hb    12.3
• WCC 12.2
• Troponin T 12
• NTProBNP 1980
• D Dimer 0.64
• ECHO EF  54%

• Rules out CAD?
• Acute Heart Failure?
• Normal
• Pneumonia? CAD? PE? Dissection? Pericarditis?
• Rules out CAD?
• Acute Heart Failure? PE? Pneumonia?
• Confirms PE?
• Excludes Heart Failure?
NTProBNP

• Chronic setting < 125 pg/ml excludes HF*
• Acute setting < 300 pg/ml excludes HF*
• Produced by left AND right ventricle
• Rises rapidly
• “Diagnostic” of Heart Failure
• PE, AMI, Pneumonia, Aortic stenosis

*Mpe, MT et al. SAMJ 2013;9(Suppl 2):661-7;
ACUTE HEART FAILURE?

- ECG – normal
- CXR – pulm congestion
- Hb 12.3
- WCC 12.2
- Troponin T 12
- NTProBNP 1980
- D Dimer 0.64
- ECHO EF 54%
12.1 Initial assessment and monitoring of patients

Three parallel assessments must be made during the initial evaluation of the patient, aided by the investigations listed in Figure 4.

(i) Does the patient have HF or is there an alternative cause for their symptoms and signs (e.g. chronic lung disease, anaemia, kidney failure, or pulmonary embolism)?

(ii) If the patient does have HF, is there a precipitant and does it require immediate treatment or correction (e.g. an arrhythmia or acute coronary syndrome)?

(iii) Is the patient’s condition immediately life-threatening because of hypoxaemia or hypotension leading to underperfusion of the vital organs (heart, kidneys, and brain)?
TROPONIN T

• “Specific” for cardiac muscle injury
• Rises fairly early – within 4 – 6 hours
  (not as early as NTProBNP)
• Rises earlier than CK, CK-MB
• More sensitive than CK, CK-MB
Suspected acute coronary syndrome

1st sample

- TnT

- <15
  - Pain >6 hours
    - Discharge
  - Pain <6 hours
    - 2nd sample after 3 hours
      - Δ<50%
        - Discharge
      - Δ>50%
        - Admit
      - Δ<20%
        - Discharge
      - Δ>20%
        - Admit

- <53
  - Pain <6 hours
    - 2nd sample after 3 hours
      - Δ= rise or fall
        - Admit

- 53-100
  - Discharge

- >100ng/L
  - Admit

• Repeated 6 hours later
  – 288 ng/L

• Home and dry?
  – Acute coronary syndrome precipitating heart failure?
# CARDIAC TROPONIN ELEVATION (other than ACS)

<table>
<thead>
<tr>
<th>Acute</th>
<th>Acute</th>
<th>Chronic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ischaemic mechanism</td>
<td>Other mechanisms</td>
<td>Myo-pericarditis</td>
</tr>
<tr>
<td>Acute heart failure</td>
<td>Cardiac contusion</td>
<td>Endocarditis</td>
</tr>
<tr>
<td>Pulmonary embolism</td>
<td>Procedural trauma:</td>
<td>Stroke</td>
</tr>
<tr>
<td>Tachy-arrhythmias</td>
<td>Cardiac surgery</td>
<td>Tako-tsubo cardiomyopathy</td>
</tr>
<tr>
<td>Brady-arrhythmias</td>
<td>Uncomplicated PCI</td>
<td>Rhabdomyolysis</td>
</tr>
<tr>
<td>Accelerated hypertension</td>
<td>ASD closure</td>
<td>COPD exacerbation</td>
</tr>
<tr>
<td>Hypotension / shock</td>
<td>Endomyocardial biopsy</td>
<td>Acute renal failure</td>
</tr>
<tr>
<td>Sepsis</td>
<td>Pacing</td>
<td>Burns &gt;30%</td>
</tr>
<tr>
<td>ARDS</td>
<td>ICD shocks</td>
<td>Snake venoms</td>
</tr>
<tr>
<td>Aortic dissection</td>
<td>RF/cryo ablation</td>
<td>Chemotherapy: Adriamycin, 5-fluoro-uracil, herceptin</td>
</tr>
<tr>
<td>Carbon monoxide poisoning</td>
<td>External cardiac massage</td>
<td>Sympathomimetic drugs</td>
</tr>
<tr>
<td></td>
<td>External cardioversion / defibrillation</td>
<td>Strenuous exertion</td>
</tr>
<tr>
<td></td>
<td></td>
<td>After non-cardiac surgery</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chronic heart failure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chronic renal failure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hypertension/ LV hypertrophy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pulmonary arterial hypertension</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Aortic valve disease</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hypertrophic cardiomyopathy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Infiltration: amyloidosis, haemochromatosis, sarcoidosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Peri-partum cardiomyopathy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hypothyroidism</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Diabetes</td>
</tr>
</tbody>
</table>
Advised CABG
Elderly lady – asked if “there was anything else”
DUAL ANTIPLATELET THERAPY

• DAPT
  – Dispirin + Clopidogrel
    • Very effective
      – Platelet resistance
    • Prodrug
      – 2 steps of activation
    • Relatively quick onset of action
    • Prolonged duration of action
      – 5 days
    • Delay surgery
    • Operate with higher risk of bleeding
Gurbel P A, Tantry U S Circulation 2010;121:569-583
TICAGRELOL

- Non-thienodipyridine drug
- P2Y\textsubscript{12} inhibitor
- Active
- Reversible
- Faster onset of action
- Shorter duration of action
- bd dosage (90mg bd)
- Dyspnoea, pauses / bradycardia
PLATO: Primary Efficacy Endpoint (Composite of CV Death, MI, or Stroke)

Cumulative Incidence (%)

- **0–30 Days**
  - Ticagrelor: 4.8%
  - Clopidogrel: 5.4%

- **0–12 Months**
  - Ticagrelor: 9.8%
  - Clopidogrel: 11.7%

**ARR=0.6%**
**RRR=12%**
**P=0.045**
**HR: 0.88 (95% CI, 0.77–1.00)**

**ARR=1.9%**
**RRR=16%**
**NNT=54***
**P<0.001**
**HR: 0.84 (95% CI, 0.77–0.92)**

No. at risk

<table>
<thead>
<tr>
<th>Group</th>
<th>0</th>
<th>2</th>
<th>4</th>
<th>6</th>
<th>8</th>
<th>10</th>
<th>12</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ticagrelor</td>
<td>9,333</td>
<td>8,628</td>
<td>8,460</td>
<td>8,219</td>
<td>6,743</td>
<td>5,161</td>
<td>4,147</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>9,291</td>
<td>8,521</td>
<td>8,362</td>
<td>8,124</td>
<td>6,650</td>
<td>5,096</td>
<td>4,047</td>
</tr>
</tbody>
</table>

*Both groups included aspirin.
***NNT at one year.

PLATO: Primary Safety Endpoint

![Graph showing comparison of PLATO-defined Total Major Bleeding between Ticagrelor and Clopidogrel]

- **Ticagrelor**: 11.6%
- **Clopidogrel**: 11.2%

**P=NS**

**P=0.43**

HR: 1.04 (95% CI, 0.95–1.13)

**No. at risk**

<table>
<thead>
<tr>
<th></th>
<th>Ticagrelor</th>
<th>Clopidogrel</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–30 days</td>
<td>9,235</td>
<td>9,186</td>
</tr>
<tr>
<td>31–90 days</td>
<td>7,246</td>
<td>7,305</td>
</tr>
<tr>
<td>91–180 days</td>
<td>6,826</td>
<td>6,930</td>
</tr>
<tr>
<td>181–270 days</td>
<td>6,545</td>
<td>6,670</td>
</tr>
<tr>
<td>271–360 days</td>
<td>5,129</td>
<td>5,209</td>
</tr>
<tr>
<td>361–360 days</td>
<td>3,783</td>
<td>3,841</td>
</tr>
<tr>
<td></td>
<td>3,433</td>
<td>3,479</td>
</tr>
</tbody>
</table>

Statins: Life Savers or Big Pharma Con?

The risks, the benefits and the conspiracy theories.

Some claim they’re nothing but a con, marginally effective drugs foisted on the public by pharmaceutical companies out to make a profit at the expense of our health; others say they are so effective in the fight against cardiovascular disease that they should be added to our drinking water.

Detractors oppose the ‘medicalisation’ of people who are not ill, and also cite grave side effects as reason enough to avoid the drugs.
• Statins
  – Bad press
    • Memory loss
    • Muscle aches and pains
    • Fatigue
    • Depression
    • My friends have all had side effects
    • No benefit

• Did Noakes say “throw away your statins”? 
STATINS IN THE ELDERLY

- Fewer people > 75 in statin trials
- Continue statin if already on
- Starting high intensity statin >75 for $2^0$ prevention, not clearly supported on the few data available
- Moderate intensity statin Rx supported ASCVD >75
- Few data available to indicate event reduction in $1^0$ prevention >75, without clinical ASCVD
- Starting statin for $1^0$ prevention >75; requires additional factors, must consider increasing comorbidities, safety,
- Can look at 10 year risk of ASCVD
CONCLUSION

• Elderly lady, chest pain, short of breath
  – Wide differential

• Acute Heart Failure
  – CXR congestion
  – ProBNP 1980

• Precipitated by an acute coronary syndrome
  – Coronary angiogram

• Stent
  – Dispirin + Clopidogrel (Ticagrelor soon to be here)