Challenge 1: Diagnosis

Chest pain >20 minutes → ECG
Challenge 1: Diagnosis

Chest pain >20 minutes

ECG

Treat as STEMI
STEMI - management

- Direct paramedic transfer to PPCI centre
- DGH attenders transferred to PPCI centre
- Exclusions:
  - ‘Overriding comorbidity’
  - Terminal cancer
  - Severe dementia
  - Ventilated patients
  - LBBB/paced rhythm

- NB: exclusion from PPCI protocol does not preclude discussion with PPCI centre
DIRECT TRANSFER PROTOCOL TO UHW FOR PRIMARY PCI

CARDIAC CHEST PAIN
(for more than 20 minutes and persisting)\textsuperscript{1,2}

12 lead ECG shows ST elevation on two or more contiguous leads – STEMI confirmed (performed within 10 min)

12 lead ECG does not show ST elevation, STEMI not confirmed or strongly suspected

Overriding co-morbidity (eg severe dementia, terminal Ca, end stage organ disease)

YES → STANDARD LOCAL ACS TREATMENT

NO → DO NOT ADMINISTER THROMBOLYSIS

WHILE WAITING FOR AMBULANCE PLEASE ENSURE:
- IV access in left arm
- 300mg aspirin
- Prasugrel 60mg to all (irrespective of age/weight and even if clopidogrel preload already)
- Analgesia + anti-emetic
- If delay call WAST control manager to ascertain which call category patient is in and source of delay

PRE-ALERT UHW
CCU 02920744343

THEN CALL 999 FOR IMMEDIATE TRANSFER TO UHW
- Ask for emergency (blue light) transfer to Cath Lab
- State reason as “confirmed Heart Attack with ST elevation on ECG”;
- Answer “yes” when asked “does this condition present an immediate threat to the patient’s life”
- If nearest available blue light ambulance is an Urgent Care Service Vehicle (no paramedic) consider providing nurse/medical escort rather than waiting for paramedic
- Confirm ETA and update UHW

NOTES:
1) Post-arrest STEMI patients who are not ventilated should be referred immediately. Post-arrest STEMI patients who are ventilated should be discussed with UHW on-call cardiology SpR or consultant prior to transfer.

2) The identification of a STEMI and requirement for transfer must as a minimum involve the senior resident on call A+E Doctor and/or the RMO. For borderline cases or suspected myocardial infarction and LBBB/paced rhythm the patient needs to be discussed first with the on call Physician/Cardiologist. If the local consultant is unavailable contact UHW cardiology SpR (sleep 5770) prior to referral/acceptance. The direct CCU number is an activation line not an advice line.
DIRECT TRANSFER PROTOCOL TO UHW FOR PRIMARY PCI

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(for more than 20 minutes and persisting)\(^1,2\)

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PPCI access challenges

- Geography
- Public education - dial 999 not attend A&E
- Ambulance availability for PPCI transfer from DGH
Challenge 1: Diagnosis

Chest pain >20 minutes → ECG → Treat as STEMI

ECG

History
Examination
Biomarkers
Repeat ECG
Clinical presentation

**Typical:**
- Prolonged chest pain (> 20 mins)
- New onset severe CCS III angina
- Destabilisation of stable angina to CCS III

**Atypical:**
- Epigastric pain, recent onset indigestion
- Pleuritic chest pain
- Recent onset dyspnoea
- Pain reproduced on palpation
- More common in women, DM, elderly, CKD, dementia
Examination in ACS: what are you looking for?

- Often normal
- Aim to exclude non-cardiac / non-ischaemic causes of chest pain (remember acute aortic syndromes)
- Spot potential haemodynamic instability
- Assess for LV dysfunction
- Stratify risk for appropriate management
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- Spot potential haemodynamic instability
- Assess for LV dysfunction
- Stratify risk for appropriate management
Risk stratification – high risk patients

- ↑ age
- Male
- Previous CAD
- DM
- ↑ BP
- LVF/CCF
- Recurrent symptoms despite treatment
- ST segment depression
- Dynamic ST changes
- Elevated troponin
- Haemodynamic instability
- Major arrhythmias
Biomarkers – Highly Sensitive troponin T

- Early detection – within 1 hour of myocyte necrosis
- Advantages: second assay at 6-9 hours not 12, high negative predictive value for ACS
- Values >5 x ULN have >90% positive predictive value for ACS
- Disadvantages: confusing normal range, concept of ‘delta change’
- Still requires presence of a fully functional brain in the requestor to interpret results
# HS troponin T reference ranges

<table>
<thead>
<tr>
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Mr L

- “We think he’s having a STEMI”
- HS trop 3150
- Fit and well 44 yr old
- NO chest pain history
- EF<15% on bedside echo
- Dx: viral myocarditis
Challenge 2: Troponinitis

- Myocarditis / myopericarditis / pericarditis
- Severe CCF
- Aortic dissection
- Exacerbation of COPD
- PE
- Cardiotoxic chemotherapy
- Severe sepsis
- Prolonged acidosis
- Ultra-endurance exertion
- HCM variants
- Hypertensive crisis
- Tachy/bradyarrhythmias
- Infiltrative e.g. Sarcoid
- Renal failure

Non-coronary myocardial injury has prognostic implications
Mrs X

- 55 yr old female, smoker, positive family hx CAD, taxi driver
- 2/7 hx central chest pain at rest
- Initial dx “ACS”
- Given aspirin, clopidogrel, fondaparinux
- Initial hs trop 35 then 2nd hs trop 21
- “troponin profile not suggestive of ACS”
- Sent home (3am), Dx – “oesophageal spasm”
- No follow up
- Meds unchanged
- No advice re: driving (taxi driver)
Mrs X

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- Meds unchanged
- No advice re: driving (taxi driver)
ECG:

- Suggests severe proximal LAD stenosis
- T inversion extends to I and aVL indicating large ischaemic territory involved
- Ischaemic QTc prolongation (524ms)
- High probability of sudden death from future ischaemic events:
  - sudden severe pump failure
  - polymorphic VT
- Recalled and admitted
- Discharging Dr given opportunity to reflect
Pathophysiology – a reminder

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Challenge 3: Which Anti Platelet Agents?

Size of the circles denotes sample size
Perimeter of the circles denotes type of investigated population
- Mixed clinical presentation at the time of stent implantation
- Acute coronary syndrome at presentation
- DAPT initiated in patients with prior myocardial infarction
- DAPT for primary prevention

LEGEND
2K pts 5K pts 10K pts 20K pts
Clopidogrel

- Prodrug requiring CYP3A4 mediated oxidation to active form (2-6 hours)
- Selectively and irreversibly inhibits platelet ADP P2Y12 receptors
- 85% is hydrolysed to an inactive form
- High individual variability in response due to genetic polymorphisms

Prasugrel

- Also a prodrug irreversibly inhibiting platelet ADP P2Y\textsubscript{12} receptors
- Faster onset than clopidogrel (30 mins vs. 2-6 hours to clinical effect)
- Therefore utilised in PPCI for STEMI
- Increased bleeding risk
Ticagrelor

- Reversible inhibitor of platelet ADP P2Y$_{12}$ receptors
- Faster onset than clopidogrel (30 mins vs. 2-6 hours to clinical effect)
- Faster recovery of platelet function on cessation
- May cause dyspnoea and bradyarrhythmias
- Reduced mortality without increased bleeding (PLATO)
Which DAPT at the front door?

- ALWAYS ASPIRIN

- If STEMI for PPCI use prasugrel 60mg with aspirin 300mg

- If NSTE-ACS consider ticagrelor......but clopidogrel more likely to be available

- Cardiologist may switch from clopidogrel to ticagrelor when patient reviewed

- For patients undergoing coronary intervention DAPT regime and duration should be determined by operator
Challenge 3: ACS in the elderly

- Proportion of those >80 yrs to triple by 2050 (CDC data)
- Underrepresentation in clinical trials – those that do participate may not be representative of ‘real elderly’
- Multi-morbidity common
- Frequently present atypically (dyspnoea) and late
- Challenging for stress testing
- Higher risk of complications following any form of revascularisation:
  - Bleeding
  - Hypotension
  - Bradycardia
  - Renal failure
Challenge 3: ACS in the elderly

- Elderly less likely to undergo invasive risk stratification\(^1\)
- Patients >75 years derive largest benefit in risk reduction from invasive strategy\(^2\)

- Individualised approach needed:
  - frailty
  - patient choice
  - renal function
  - bleeding risk
  - cognitive function

- Trial data pending: SENIOR trial

**Challenge 4: ACS in CKD**

- Many CKD patients have CAD risk factors
- Diagnosis more challenging – ECG changes plus troponin elevation are common
- Worse prognosis from ACS
- Little safety data for P2Y$_{12}$ inhibitors in CKD 5 (use clopidogrel)
- Fondaparinux contraindicated if Cr clearance <20mls/min (use enoxaparin)

- Less likely to receive evidence-based Rx$^1,^2$
- Concern re: contrast nephropathy

1: Esekowitz J *et al.* Association of renal insufficiency with outcome in HF and CAD. *JACC.* 2004; **44:** 1587-92
Mr P

- Mr RP, 71 yr old male, new chest pain at rest
- CKD 4, asthma, endocarditis 2013 (MV repair)

- Initial trop 43, 6 hour trop 53
- Cr 204 (stable)
- Troponin rise attributed to CKD

- Discharged home with ISMN and referral to RACPC
- Attended routine yearly review in my OPD 2/52 later
Challenge 4: ACS in CKD

- Serial and old ECGs useful to determine new changes
- Rise and fall pattern of HS trop suggests myocardial injury
- Requires individualised assessment of relative risk of invasive assessment
- Prehydration for all patients with eGFR<60
Summary

• History taking is still crucial to diagnosis in ACS

• Beware late presenters with a falling troponin

• Always aspirin; DAPT regime should be guided by cardiology

• One size does not fit all for high risk groups