Early vs delayed angiography & complete vs staged PCI

When to perc up

Nepean WTET summary 22/9/20

Background and Rationale

- Troponin elevation may indicate myocardial strain or injury without necessarily ischaemia or infarction. In the critically ill it can be prognostic rather than diagnostic. Myocardial injury (acute troponin rise/fall without ischaemia e.g. myocarditis, pulmonary embolism), type 2 myocardial infarction, MI (ischaemia secondary to supply-demand mismatch, may have coronary artery disease, CAD, but no acute plaque disruption) and non-ST elevation MI, NSTEMI (most frequently type 1 MI; CAD, with acute plaque disruption) have differing aetiologies, prognoses & management
- Primary percutaneous coronary intervention (pPCI) can facilitate emergent coronary reperfusion optimising myocardial recovery in type 1 MI. Ischaemic heart disease (IHD) is the most common cause of cardiac arrest with 70% of patients resuscitated and referred for angiography exhibiting coronary artery disease (CAD). Up to 80% of patients with cardiogenic shock have multivessel disease and this carries a higher mortality when compared to single vessel disease

Advantages and Disadvantages

- Advantages of more aggressive approach to PCI
    - Good physiological rationale; early reperfusion maximises myocardial function / recovery
- Disadvantages of more aggressive approach to PCI
    - Re-opening non-culprit lesions to include chronic total occlusions may not improve myocardial function whilst subjecting the patient to greater risk of volume overload, high doses of contrast (and associated nephropathy / renal failure) and further ischaemia
    - More challenging to tightly control physiology in cardiac catheterisation lab (flat, no TTM ...)

Key studies

Out of hospital cardiac arrest (OOHCA) not caused by STEMI

- COACT 2019 NEJM
    - MC RCT; OOHCA with initial shockable rhythm, ROSC but unconscious with no ECG changes of STEMI, no shock and no obvious non-coronary cause for arrest. Randomised to standard Mx and immediate (<2h randomisation) vs delayed (after neurological recovery; usually after ICU discharge – unless cariogenic shock, recurrent life-threatening arrhythmias or recurrent ischaemia) angiogram +/- PCI (revascularisation and anticoagulation at discretion of treating physician e.g. culprit vs multivessel etc.; recommended in >70% stenosis). N=552
    - Primary outcome; survival at 90d 64.5% vs 67.2% (OR 0.89, 95% CI 0.62-1.27, P=0.51)
    - Time to angio from randomisation 0.8h vs 119.9h & acute thrombosis found in 3.4% vs 7.6% with PCI performed in 33% vs 24.2% & CABG in 6.2% vs 8.7% respectively

Cardiogenic Shock secondary to acute myocardial infarction (AMI)

- CULPRIT-SHOCK 2017 NEJM
    - MC RCT; acute STEMI or NSTEMI with associated cardiogenic shock (BPs<90mmHg >30mins or need for catecholamines; pulmonary congestion; impaired end organ perfusion), for planned early revascularisation by means of PCI with multivessel CAD identified (=/> 2 major vessels; =/>2mm diameter with >70% stenosis) and an identifiable culprit lesion, randomised to PCI of culprit lesion only (+/- staged PCI nonculprit lesions) vs immediate multivessel PCI. N=706
    - Primary endpoint; composite of death from any cause or severe renal failure needing RRT <30d; 45.9% vs 55.4% (RR 0.83; 95% CI 0.71-0.96, P=0.01) predominantly made up by increased risk of death (43.3% vs 51.6%, P=0.03) rather than RRT (11.6% vs 16.4%, P=0.07)
    - 1y follow up study NEJM 2018; no difference in mortality at 1y (50% vs 56.9%, RR 0.88, 95% CI 0.76-1.01) but more revascularisation & more admissions with heart failure in culprit arm

Non ST elevation acute coronary syndromes (NSTEACS)

- FRISC 2 Lancet 1999
    - MC RCT; 2x2 factorial design (invasive <7d vs non-invasive treatment & long term dalteparin vs blacebo for 3m) unstable coronary artery disease. N=2457
    - Primary endpoint (ITT); 6m composite endpoint of death or MI 9.4% (invasive) vs 12.1% (risk ratio 0.78, 95% CI 0.62-0.98, P=0.031)
- TACTICS TIMI 2001 NEJM
    - MC RCT; NSTEACS randomised to angiogram +/- PCI <48h vs conservative Mx. N=2220
    - Primary endpoint; composite of death, non-fatal MI, readmission with ACS at 6m, 15.9% vs 19.4% (OR 0.78, 95% CI 0.62-0.97, P=0.025)
- RITA 3 Lancet 2002
    - MC RCT; NSTEACS randomised to intervention or conservative strategy. N=1810
    - Primary endpoint; death, MI and refractory angina at 4m; 9.6% vs 14.5% (risk ratio 0.66, 95% CI 0.51-0.85, P=0.001) and combined rate of death or non-fatal MI at 1y; 7.6% vs 8.3% (risk ratio 0.91 95% CI 0.67-1.25, P=0.58)
- ICTUS 2005 NEJM
    - MC RCT; NSTEACS randomised to angiogram +/- PCI <48h (CABG if indicated) vs medical Mx and selective intervention (angiogram +/- PCI if refractory angina, arrhythmias, haemodynamic instability, or clinically significant ischaemia on exercise test). N=1200
    - Primary endpoint; composite of death, MI or rehospitalisation for anginal symptoms at 1y; 22.7% vs 21.2% (RR 1.07, 95% CI 0.87-1.33, P=0.33)
- SWEDEHEART 2017 European Heart Journal
    - Observational prospective MC cohort study; NSTEMI requiring CCU admission post PCI 2006-2013 in Swedish PCI centers. Compared early (d1-3) vs late PCI
    - Lower risk of all-cause mortality with early PCI. Less recurrent MI and severe bleeding also

Summary (my practice)

- Studies have shown that multivessel (vs culprit vessel only) pPCI in STEMI is associated with lower re-infarction risk but no difference in all-cause mortality (at present; ?emerging); unless associated cardiogenic shock where multivessel PCI was associated with increased mortality when compared to culprit vessel only PCI (CUPRIT-SHOCK trial). Timing of early non-culprit multivessel PCI therefore in STEMI without cardiogenic shock remains controversial (immediate vs staged <7d / in-hospital)
- In OOHCA with ROSC in absence of STEMI or cardiogenic shock (or other clear non-cardiac aetiology for arrest), delayed angiography +/- PCI is a reasonable approach. Timing then is dependent on clinical picture, haemodynamic profile and clinical likelihood of a type 1 MI. This allows in the initial period focus on strict neuroprotection and optimal end-organ supports
- In type 1 NSTEMI /NSTEACS it is reasonable to decide upon invasive vs conservative (medical) strategies based on risk factors (recurrent ischaemia, heart failure, LVEF<0.4, serious ventricular arrhythmias, large /multiple regional wall abnormalities on echocardiography) as per AHA/ACC