Topic Overview: Cardiac Module
Sub-Module: C8 – Cardiogenic Shock and the Management of Patients with Unstable Myocardial Infarction
Last Updated August 3 2012

This handout is designed as pre-reading for the simulation session C8 - Cardiogenic shock and the management of patients with unstable myocardial infarction. This session involves a presentation and an immersive simulation scenario. Use this document to jog your memory or to aid in your reflection of the session, and in particular the simulation.

Session Objectives
- Demonstrate a structured method of assessment, diagnosis and management of patients presenting to the Emergency Department (ED) with cardiogenic shock
- Apply non-technical skills
    - Teamwork
    - Communication
    - Decision Making
    - Situational Awareness

Cardiogenic shock
Cardiogenic shock is defined as "Circulatory insufficiency that creates an imbalance between tissue oxygen supply and oxygen demand… caused by inadequate cardiac pump function" (Tintinalli). Pump failure leads to reduced cardiac output, hypotension, impaired tissue perfusion, cellular hypoxia, anaerobic metabolism and production of lactate. The feedback loop responds by releasing hormones (noradrenaline, adrenaline, dopamine, etc) to cause vasoconstriction and tachycardia in an attempt to improve cardiac output to vital organs (brain, heart, lungs) increasingly at the expense of the less vital ones. In cardiogenic shock there is pump failure restricting the effect of these responses, the acidosis contributes to worsening pump failure, and the effect of systemic vasoconstriction is variable.

The most common cause is acute myocardial infarction (AMI) causing pump failure. Other causes include:
- Acute MR (mitral regurgitation)
- Acute VSD (ventricular septal defect)
- Acute valve disease – severe AS (aortic stenosis), MR, aortic incompetence
- Drugs
- SIRS response (systemic inflammatory response syndrome)
- Cardiomyopathy – viral myocarditis, sarcoid, peripartum
- Pulmonary hypertension
    - PE (pulmonary embolus)
    - Cor pulmonale
- Congenital – HOCM (hyper-obstructive cardiomyopathy)
- Pericardial tamponade
- High output failure
    - Beri beri
    - Thyrotoxicosis
    - Sepsis

Assessment
Assessment and management occur simultaneously in the critically unstable patient in the ED.

Assessment aims to establish the diagnosis, assess severity, seek underlying causes, identify secondary complications and impact on co-morbid conditions.

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History

This should include the duration of presenting symptoms and evidence of the underlying cause. Most commonly this is AMI but other causes should be excluded including viral myocarditis, alcohol abuse and thyrotoxicosis, HOCMand cardiac arrhythmias). Current medication use and allergies are essential to guide treatment choices (especially beta blockers and calcium channel blockers); likewise allergies are essential to know when prescribing any medication.

Often a collaborative history is required with sources including the ambulance officers, family or friends, the general practitioner and old notes.

Examination

This should reveal the severity of the illness and haemodynamic stability; guides therapy; seeks the underlying cause and complications of both the illness and of the management. Careful cardiovascular examination may reveal a wide pulse pressure of Aortic Stenosis; elevated jugular venous pulse of right heart failure; displaced apex beat of a chronically dilated Left Ventricle (not suggestive of an acute course); systolic murmurs of AS, MR or VSD; distinctive murmurs of HOCM with dynamic manoeuvres (louder with valsalva); diastolic murmurs of aortic incompetence; rales in the lung fields (LV failure) and peripheral oedema of the sacrum or lower limbs (suggesting RVF). Further clinical examination may highlight the underlying pathology, e.g. obesity, IVDU, thyrotoxicosis.

The findings on examination give the Killip classification (and predicted mortality) for those with AMI.

- Killip I – No clinical signs of heart failure (6% mortality)
- Killip II – Rales or crackle < 50% of the chest, S3 audible and elevated JVP (17%)
- Killip III – Rales or crackles > 50% of the chest (38%)
- Killip IV – Hypotension, cardiogenic shock (67%)

Clear documentation at the time of presentation is essential in guiding the initial management and subsequent assessment of complications during this presentation and those in the future.

Investigations

Immediate investigations should be performed on arrival.

A 12 lead ECG should be urgently obtained and reviewed by someone competent in assessing ECGs, with early specialist advice if there is any doubt about the presence of STEMI and its location. Comparison with old ECGs is often insightful. Early intervention improves outcomes, especially in those with cardiogenic shock as a result of AMI (SHOCK trial). Specific avoidance of nitrates in RV infarction may improve outcomes. Seek arrhythmias, electrolyte abnormalities and LVH.

When IV access is obtained bloods should be sent for urgent assay, however there are no biochemical markers that are specific for cardiogenic shock and the cardiac enzymes may not be elevated on initial presentation if that occurs early. Drug levels should be sent including digoxin levels.

A mobile CXR should be rapidly obtained to assess for pulmonary oedema/effusions, cardiomegaly and other diagnoses including focal infection, pneumothorax, and radiographic evidence of dissection.

A bedside ultrasound should be obtained, dependent on available skills. This should be confirmed with formal echocardiography as soon as practicable after this. This imaging looks for the LV and RV function, evidence of pericardial effusion and valve function. Free wall rupture and acute MR may also be identified.

The patient should remain on a monitor with regular serial measurements including BP, ECG, Oxygen Saturation, Urine Output, BSL, lactate clearance and serial blood tests as warranted.

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Management Goals

- The treatment aim is to improve cellular respiration by increasing oxygenation, and tissue perfusion to remove anaerobic metabolites.

- Ultimately the underlying cause needs treatment. Early intervention from AMI requiring Percutaneous Coronary Intervention (PCI) or urgent Thrombolysis reduces mortality. Surgical intervention may be required for valve or free wall rupture, or effusion causing Tamponade. Sepsis will require early goal directed therapy.

- Seek and treat physiological changes including cardiac arrhythmias, electrolyte abnormalities and mechanical complications.

- Right ventricular failure requires specific mention. In this instance you should avoid nitrates and use cautious fluid loading.

Management

Cardiogenic shock is a result of pump failure causing global tissue hypoxia as a result of hypoperfusion. The PaO2 of circulating blood should be increased in an attempt to meet tissue oxygen demand. High flow oxygen should be applied, whilst non-invasive ventilation is prepared.

Ventilation

- CPAP has been shown to reduce intubation rates and improve symptoms (3CPO study), although does not have a short-term mortality benefit.

- Non-Invasive Ventilation (NIV) may worsen hypotension secondary to increased intrathoracic pressure and decreased venous return. This usually occurs in the initial phases of its use and may require removal of the NIV and preload with cautious fluid resuscitation or early inotropic support.

- Intubation is the final step in airway management to improve oxygenation and ventilation. Pharmacological induction may cause further hypotension when the sympathetic drive is removed and contribute to worsening myocardial depression. Intubation maybe unavoidable for multiple reasons including when NIV is not tolerated by the patient or otherwise contraindicated; the patient must be supine for a prolonged procedure (including PCI) and the transport restrictions of NIV. Care should be taken to minimise the apnoea period and the extent of hypotension, deterioration during induction should be anticipated thus precise preparation and experienced assistance is advantageous.

Fluids

Cautious fluid boluses of 100-250ml normal saline may be attempted if hypotensive with minimal pulmonary congestion. Preparation of inotropic support should be anticipatory and may assist in temporarily stabilising the patient en route to definitive care.

Drugs

The choices of drugs to use will be variable dependent on clinical condition, clinician knowledge and local practise. Early senior input should be sought as required.

<u>Targeted treatment – Based on Systolic Blood Pressure (SBP)</u>

- If the SBP >80mmHg, start with dobutamine (5mcg/kg/min) which improves myocardial contractility and coronary blood flow. It may cause hypotension so draw up noradrenaline at the same time. With acute MR or a VSD you might need to consider Sodium Nitroprusside, to reduce after load.

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- If the SBP is 70-80 mmHg, you might consider dopamine (5mcg/kg/min) as it can aid in vasoconstriction.

- If the SBP is <70 mmHg then use noradrenaline (2mcg/min) which is mainly a vasoconstrictor and doesn't improve CO. You may need to add in Milrinone or dobutamine to accomplish this.

Targeted treatment of AMI

As most cardiogenic shock is a result of acute myocardial infarction, treatment for this should be initiated at the initial presentation, unless there are clear contraindications. This treatment will include:

- Aspirin 300mg orally
- Clopidogrel 600mg loading (unless aVR lead elevation suggestive of triple vessel disease which may require coronary artery by-pass grafting -CABG)
- Heparin – weight based loading bolus, followed by infusion
- Nitrates may be helpful but should be avoided in RV infarction and be used very cautiously in the hypotensive patient.

Beta Blockers are *contraindicated* in those in cardiogenic shock or at risk for cardiogenic shock; there is an increased mortality with their use in these patients.

Reperfusion

The Australian Resuscitation Council guidelines advise that revascularisation by PCI/CABG should be first line, if able to be performed in a reasonable time (not specified, in other parts of the document 2 hours is the acceptable time if >3 hours of pain).

The SHOCK study and registry highlight that there is a significant mortality benefit at both 3 and 6 months for the early revascularization group. This is reflected in both the Australian and American Guidelines for cardiogenic shock. Alternately Thrombolysis then IABP for transfer to centre capable of PCI. Thrombolysis alone, if no suitable alternative, will also improve mortality.

Mechanical Support

Options include:

- Intra Aortic Balloon Pump (IABP)
    - Increases diastolic blood pressure, reduces afterload and reduces myocardial oxygen demand. It is used as a bridge to definitive therapy – catheterisation or surgical correction of mechanical failure or stenosed coronary arteries.
    - Insertion requires experience and early notification of this need is suggested
- Left Ventricular Assist Device
- Extra Corporeal Membrane Oxygenation and cardiac by-pass
    - Temporising measure until definitive treatment

Summary

- Acute myocardial infarction is the leading cause of cardiogenic shock seen in the ED
- These patients require a structured approach to assessment and management
- It is important to know you hospital's policies, guidelines and available resources
- Early specialist help and intervention may be vital
- A multidisciplinary team approach is vital to effectively and efficiently treat critically unwell patients
- Good use of non-technical skills allows teams to function smoothly and decreases the risk of errors

References and Further Reading

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