

CASE 5

Short case number: 3.2.5

Category: Cardiovascular

Discipline: Medicine

Setting: Emergency Department

Topic: Coronary artery disease – acute myocardial infarction, cardiopulmonary arrest

Case

Maud Gyer, aged 79, presents by ambulance with severe chest pain. On arrival she is distressed and breathless. The ambulance driver tells you Maud has central crushing chest pain and is hypotensive and tachycardic. Her oxygen saturation is 80% on pulse oximetry and she is cold and clammy. Maud has a past history of hypertension and non insulin dependent diabetes mellitus.

Questions

1. What further history and examination would you undertake?
2. What emergency management would you commence?
3. Maud has classic ECG changes of and inferior acute myocardial infarction. Describe what these changes would be?
4. What cardiac enzymes would you order and why?
5. Maud suddenly develops acute cardiopulmonary arrest in the emergency department. Describe your immediate series of responses?
6. What is the evidence base behind the use of thrombolytic and antiplatelet drugs in the emergency department in the setting of acute myocardial infarction?

Suggested reading:

- Colledge NR, Walker BR, Ralston SH, Penman ID, editors. Davidson's Principles and Practice of Medicine. 22nd edition. Edinburgh: Churchill Livingstone; 2014. Chapter 18.

Advanced Reading

- Stiles MK, Dabbous OH, Fox KA, GRACE investigators. Bleeding events with antithrombotic therapy in patients with unstable angina or non-ST-segment elevation myocardial infarction; insights from a large clinical practice registry (GRACE). Heart, Lung & Circulation. 17(1):5-8, 2008 Feb.
<http://www.sciencedirect.com/locate/nd.edu.au/science/article/pii/S1443950607001540>

ANSWERS

1. What further history and examination would you undertake?

Further history must try and ascertain whether this could be a presentation of a myocardial infarction. This includes type and nature of the pain, radiation of the pain, whether there was a change in the pain with the application of the nitrate patch, associated physical symptoms such as breathlessness, light-headedness, nausea, vomiting, sweating, change in the pain with the application of the nitrate patch or a sense of fear or impending doom?

Exam for signs of sympathetic activation i.e. pallor, sweating and tachycardia, or vagal activation i.e. bradycardia and vomiting.

Check for pallor and cool peripheries, pulse (character and rate), blood pressure (hypotension), JVP, character of the apex beat. The most common auscultation findings in AMI are muffled heart sound, decreased intensity of S1, audible S4, S3 and paradoxical splitting of S2.

Auscultation of base of both lungs may reveal late or pan-inspiratory crackles due pulmonary congestion because of left ventricular failure.

2. What emergency management would you commence?

The most important concern is that this patient is at risk of death from acute myocardial ischemia (and/or malignant arrhythmias) and therefore requires urgent admission to hospital. Patients with suspected myocardial infarction require immediate access to medical care with defibrillation facilities.

On arrival, O2 and aspirin are administered and immediate IV access established. Blood should be taken immediately and IV analgesia (e.g. morphine) administered if there is ongoing pain.

A 12-lead ECG is taken and continuous ECG monitoring established.

If there are ST-elevations consistent with acute myocardial infarction (STEMI) or a newly onset left bundle branch block, acute re-perfusion therapy is indicated either with PTCA (PCI) (if facility is available) or thrombolysis therapy.

Patients are best managed in specialist units where they be monitored and receive therapy for acute complications should they develop.

3. Maud has classic ECG changes of an acute inferior myocardial infarction. Describe what these changes would be?

An inferior myocardial infarct presents with the ECG changes of ST elevation on II, III and aVF. It is important to remember that up to 50% (10-50%) of inferior infarcts are associated with a right ventricular infarct and it is therefore important to look for ST segment elevation in leads V3R and V4R) and tall R waves & ST depressions in V1-V3.

4. What cardiac enzymes would you order and why?

Myocardial infarction causes detectable rises in plasma concentrations of some enzymes and proteins that are usually only detectable in myocardial cells.

The markers that are most used are;

- The cardio-specific proteins troponin T and I
- The cardio-specific creatine kinase enzyme CKMB.

The troponins are the most sensitive makers of myocardial damage and start to rise in the blood within 4-6 hours of the injury. This may remain elevated for up to 2 weeks.

CK and CKMB start to rise 12 hours after the injury and taper off within 48-72 hours. CK is not specific for cardiac muscle and may be elevated following defibrillation or intramuscular injections. CKMB and troponins are specific.

When troponins are available, this is the better investigation.

5. Maud suddenly develops acute cardiopulmonary arrest in the emergency department. Describe your immediate series of responses?

- Call for assistance

Precordial thump if witnessed arrest

(The precordial thump may be considered for patients with monitored, pulseless ventricular tachycardia if a defibrillator is not immediately available.

The precordial thump is relatively ineffective for ventricular fibrillation, and it is no longer recommended for this rhythm.

There is insufficient evidence to recommend for or against the use of the precordial thump for witnessed onset of asystole caused by AV-conduction disturbance. The precordial thump should not be used for unwitnessed cardiac arrest. (Australian Resuscitation council 2012))

- Attach defibrillator and check the monitor is in place
- Check rhythm and pulse
- Prompt assessment and restoration of airway, administration of oxygen via mask and restoration of circulation using CPR

6. What is the evidence base behind the use of thrombolytic and antiplatelet drugs in the emergency department in the setting of acute myocardial infarction?

Thrombolytic therapy has been shown to improve survival rates in patients with acute myocardial ischemia if administered in a timely fashion in the appropriate group of patients. If coronary angiography is not available or will cause a delay greater than 90 minutes, then the optimal approach is to administer thrombolytics within 12 hours of onset of symptoms in patients with ST-segment elevation greater than 0.1 mV in 2 or more contiguous ECG leads, new left bundle-branch block (LBBB), or anterior ST depression consistent with posterior infarction. Tissue plasminogen activator (t-PA) is superior to streptokinase in achieving a higher rate of coronary artery patency; however, the key to efficacy lies in the speed of the delivery of therapy. More recently, the addition of a IIb/IIIa receptor antagonist is combined with a half dose of a thrombolytic agent as the initial reperfusion strategy to increase patency. The reduced dose of a thrombolytic agent combined with a potent platelet inhibitor may prove to be the preferred method for medical reperfusion, but larger trials are pending.

Percutaneous coronary angiography is the treatment of choice in most patients with STEMI, assuming the intervention can be initiated in less than 90 minutes of the arrival in hospital. This provides greater coronary patency, lower risk of bleeding, and instant knowledge about the

extent of the underlying disease. There may be a mortality benefit over thrombolytic therapy. The widespread use of stenting and adjunctive IIb/IIIa therapy are improving the results of primary PCI. Recently, it has been shown that patients with acute MI, coronary stenting and abciximab lead to a greater degree of myocardial salvage and a better clinical outcome than fibrinolysis with thrombolytic therapy. Improvement of long- and short-term outcomes, however, depends highly on the speed with which reperfusion is achieved. Primary PCI is also the treatment of choice in patients with cardiogenic shock, patients in whom thrombolysis failed, and those with high risk of bleeding or contraindications to thrombolytic therapy. However, the procedure should be limited to experienced operators and restricted to sites where appropriate facilities are available. Operators should have at least 75 cases per year, while the centre should perform at least 200 cases per year as per the recommendations according to recommendations laid down by the American College of Cardiologists.