

**MYOCARDIAL INFARCTION** (MI), commonly known as a heart attack, is the irreversible necrosis of heart muscle resulting from a period of prolonged ischemia (restriction in blood supply). It results from the rupture of an atherosclerotic plaque within a coronary artery, followed by thrombus formation. Thus, whichever portion of the myocardium receives its blood supply from that artery will be directly affected.

**RISK FACTORS** for atherosclerosis / coronary artery disease:

AGE > 45 : *Patient is 52 years old*

Smoking : *Smokes 40 cigarettes a day*

Air pollution : *He worked as miner for 24 years*

Hypertension

Diabetes Mellitus

Hypercholesterolemia

Obesity

Family history

Chronic psychological / social stress : *Patient is from one of the most deprived areas in the UK and has been unemployed for 9 years.*

[http://www.medicine.gu.se/digitalAssets/1120/1120543\\_WLabBergstomNOPIC.pdf](http://www.medicine.gu.se/digitalAssets/1120/1120543_WLabBergstomNOPIC.pdf)

### CLINICAL OBSERVATIONS – INITIAL PRESENTATION

- *Barely conscious* – due to inadequate cerebral perfusion and cardiogenic shock.
- *Sweating profusely* – also referred to as ‘diaphoresis’ – occurs in an acute MI due to an increased output of the sympathetic nervous system. This also accounts for *cold and clammy* skin.
- *Sudden attack of chest pain* caused by lack of blood / oxygen supply to required area.
- *Shortness of breath* – also referred to as ‘dyspnoea’ – caused by reduced cardiac output leading to left ventricular failure. Heart can no longer adequately pump blood around the body, causing a ‘back-up’ of deoxygenated blood in the pulmonary circulation.
- *Nausea and vomiting* – occurs due to pain, redirection of blood away from the gastrointestinal system, and increased output of the sympathetic nervous system – particularly due to stimulation of abdominal splanchnic and vagal afferent nerves.

- *His pulse is weak... and he is markedly hypotensive – CARDIOGENIC SHOCK.* Acute damage to the myocardium results in reduced cardiac output. This leads to acute hypoperfusion and hypoxia of the tissues and organs, despite the presence of an adequate intravascular volume. This is a medical emergency requiring immediate resuscitation by treating the underlying cause: patient is therefore admitted to *Intensive Care Unit*.

***In spite of all these classical manifestations, it is important to note that approximately a quarter of all myocardial infarctions are silent, without chest pain or other obvious symptoms.***

- *Occasional chest pain radiating into his right arm – angina*, which in turn is a sign of coronary artery disease. The coronary arteries are narrowed by fatty, fibrous deposits. During physical exertion, increased cardiac output requires more oxygen than the coronary arteries can supply, causing myocardial ischemia – which manifests as chest pain.
- *Why did patient presume indigestion?* Occasional nature of chest pain (did not initially cause enough problems for patient to require medical attention); radiating into right arm (angina typically presents with pain radiating to the left side).

#### **PATIENT MANAGEMENT**

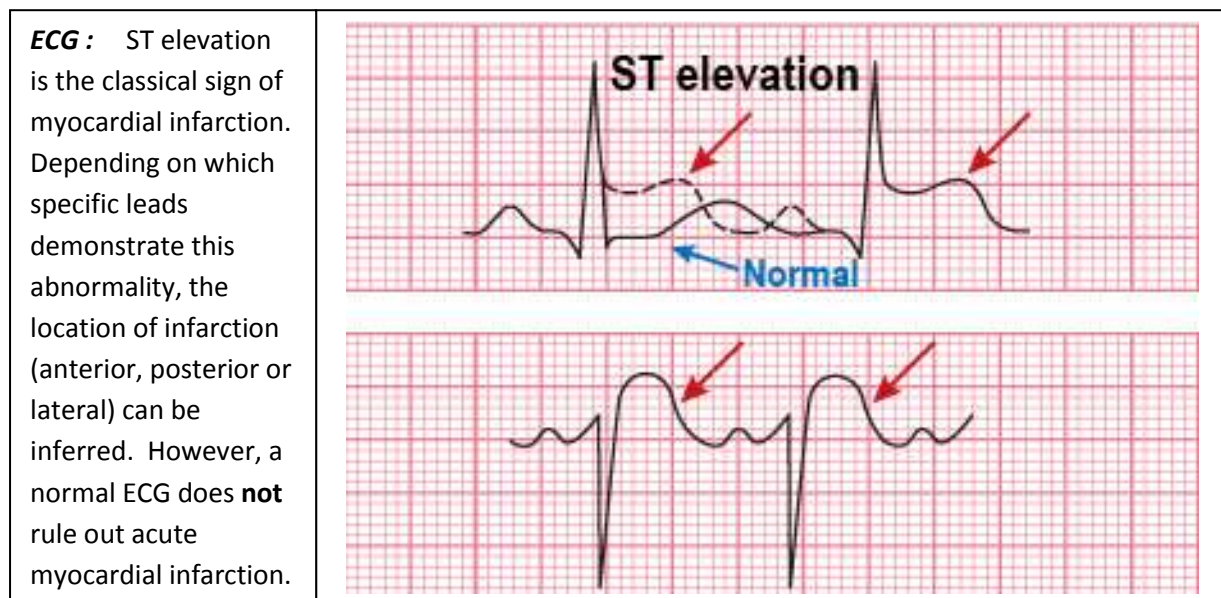
- Aspirin 300 mg orally (dispersible or chewed).
- Monitor oxygen saturation using pulse oximetry: offer supplemental O<sub>2</sub> to patient if saturation is less than 94% (making sure he is not at risk of hypercapnic respiratory failure), aiming for a target O<sub>2</sub> saturation of 94–98%.
- Pain relief with GTN spray/sublingual and/or an intravenous opioid 2.5-5 mg diamorphine, or 5-10 mg morphine intravenously with an anti-emetic (anti-sickness medication).
- Patency of the occluded artery can be restored by percutaneous coronary intervention (PCI) or by administering a thrombolytic drug. PCI is the preferred method, due to fewer long-term risks. It involves non-surgical widening of the coronary artery, using a balloon catheter to dilate the artery from within. A metallic stent is usually placed in the artery after dilatation.

**OTHER MEDICATIONS:**      Anti-platelets (e.g. aspirin, clopidogrel)  
    Beta-blockers (e.g. propranolol, atenolol)  
    Angiotensin-converting enzyme (ACE) inhibitors (e.g. ramipril)

## INVESTIGATIONS

*Cardiac troponins T and I* are highly sensitive and specific markers for cardiac damage. The risk of death from a myocardial infarction is directly related to troponin level. Patients with no detectable troponins have a good short-term prognosis. Serum levels increase within 3-12 hours from the onset of chest pain, peak at 24-48 hours, and return to baseline over 5-14 days. Troponin levels may therefore be initially normal, and should be repeated.

*Myocardial muscle creatine kinase (CK-MB)* levels increase within 3-12 hours of onset of chest pain, reach peak values within 24 hours, and return to baseline after 48-72 hours.



## CLINICAL OBSERVATIONS – SECOND ADMISSION

*Tachycardia & hypotension* – CARDIOGENIC SHOCK

*Pulsus paradoxus* – Systemic arterial pressure normally falls by less than 10 mmHg during inspiration in healthy individuals, but this decline is not palpable at the peripheral pulse. However, moderate to severe cardiac tamponade induce hemodynamic changes that exaggerate the inspiratory fall in systolic blood pressure. Heart sounds can be auscultated, but the peripheral pulse at points such the radial artery is barely detectable, if at all.

*Acute cardiac failure* – Mechanical complication of MI, exacerbated in this patient due to...

**MYOCARDIAL RUPTURE** (not explicitly mentioned in notes but an almost definite complication):

The most common cause of a free wall rupture – of the left or right ventricles – is a myocardial infarction. This causes bleeding into the pericardium, leading to cardiac tamponade, with rapidly progressive deterioration in cardiac function (haemodynamic collapse).

Emergency pericardiocentesis (draining the pericardial effusion by fine needle aspiration) and surgery (to repair the ruptured ventricle wall) are essential for any hope of survival, but cardiac arrest and death is unfortunately the more likely outcome.

**CARDIAC TAMPONADE** is a clinical syndrome of caused by the accumulation of fluid in the pericardial space (pericardial effusion), resulting in reduced ventricular filling and subsequent hemodynamic compromise. It should be noted that myocardial rupture is not the only cause of cardiac tamponade – others include aortic dissection, pericarditis etc...

Pericardium surrounds the heart, and is composed of 2 layers. The thicker parietal pericardium is the outer fibrous layer; the thinner visceral pericardium is the inner serous layer. Between these layers is the pericardial space, which usually contains 20-50 ml of pericardial fluid.

