Case outline

Peter Brown is a 58-year-old gentleman who has experienced an episode of crushing central chest pain while at work. Peter works as a taxi driver and a colleague has taken him to the Accident and Emergency Department. On admission, Peter is sweaty, clammy, nauseated and short of breath. He is complaining of chest pain radiating to his left arm. This is Peter’s first presentation to hospital and he has no relevant past medical history. Peter smokes approximately 20–30 cigarettes per day and takes alcohol at weekends only. Peter is anxious and is concerned that his wife and children are informed. He also states his father died suddenly following a heart attack a number of years ago. Peter is immediately triaged and taken to the resuscitation room. You are the receiving nurse.

Observations on admission include:
- Respiratory rate: 18 breaths per minute
- Oxygen saturations: 95%
- Blood pressure: 150/90 mmHg
- Pulse: 94 beats per minute
- Temperature: 37°C.

On admission to hospital an electrocardiograph (ECG) has been undertaken. Peter has been diagnosed with an anterior ST segment elevation myocardial infarction (anterior STEMI). Blood samples have also been drawn for urea and electrolytes (U&E), full blood picture (FBP) and highly sensitive troponin T.

1. Discuss Peter’s immediate problems and explain these using your knowledge of pathophysiology.

A. On admission to Accident and Emergency (A&E), Peter will be assessed using the Manchester Triage system (Cooke and Jinks 1999) incorporating the ABCDE approach as per the Resuscitation Council (UK) (2011). The ABCDE assessment and management tool can be applied to all deteriorating or critically ill patients. It is recognized that approximately 30 per cent of people developing a myocardial infarction die before reaching hospital (Resuscitation Council (UK) 2011). Furthermore, Henderson (2010) states that in Europe the estimated incidence of acute coronary syndrome (ACS) lies between 1 in 80 and 1...
in 170 of the population. While awareness of risk factors may reduce these figures, it is imperative that nursing staff are aware of the seriousness of ACS and the risk it poses to life. Effective and efficient assessment together with early treatment is at the heart of improving survival.

An ST elevation myocardial infarction is characterized by fissures or cracks in the atherosclerotic plaques of coronary arteries. As Henderson (2010) explains, plaque rupture triggers platelet aggregation and activation of the coagulation cascade. This results in coronary thrombosis and a resulting occlusion in the coronary artery. The size and pattern of the myocardial infarction are dependent on the location and extent of the occlusion.

**Central crushing chest pain**

When assessing the patient with a probable ACS, it is imperative to undertake a good history in relation to the patient’s chest pain and presenting symptoms. It is notable that while chest pain is one of the main symptoms of ACS, it does not occur in 25 per cent of all cases (Naik et al. 2007). This is particularly common among patients with diabetes who may not sense pain due to peripheral neuropathy.

Chest pain arises due to myocardial ischaemia, and the coronary occlusion. As Woods et al. (2010) state, chest discomfort is related to an imbalance between oxygen supply and demand. Blood is essentially trying to ‘push’ its way past an obstruction. A myocardial infarction results when there is prolonged ischaemia, causing irreversible damage to the heart muscle. Part of the heart muscle is not receiving oxygenated blood due to an obstructed coronary artery. The lack of oxygenated blood contributes to the pain felt by Peter. Furthermore, the triggering of the inflammatory process, when there is myocardial injury together with the cellular change from aerobic to anaerobic metabolism, causes an increase in the production of lactic acid. The inflammatory process together with the release of lactic acid causes swelling, oedema and increased pressure on nerve endings, resulting in chest pain.

**Sweaty and clammy sensation**

Significant coronary arterial occlusion will stimulate the sympathetic nervous system (SNS). This stimulation of the SNS will signal the release of adrenaline and noradrenaline from the adrenal medulla. Adrenaline release is rapid and will cause a tachycardia in an effort to maintain homeostasis. The release of adrenaline and noradrenaline also causes peripheral vasoconstriction due to their effects on alpha- and beta-adrenergic receptors (Karch 2008). The effect of these naturally occurring catecholamines is to make the skin feel sweaty, slightly cold and clammy. Furthermore, the release of catecholamines also heightens the state of anxiety by increasing the heart rate and blood pressure. Porth (2011) explains that the developing sympathetic stimulation gives rise to restlessness, tachycardia and a feeling of impending doom.

As the heart rate increases in an attempt to compensate for a reduction in contractility, there will be a corresponding rise in blood pressure. Peter’s blood pressure rise can also be explained by the increase in catecholamine activity together with the pain experienced and the increased sympathetic activity. These compensatory activities cause a rise in blood pressure
due to increased vasoconstriction, increased contractility and tachycardia. However, as the infarction develops, if left untreated, these compensatory activities will fail and may exacerbate the infarcted area of heart muscle. This is due to the increase in myocardial oxygen demand caused by the increase in heart rate. In other words, there is a tipping of the scale towards oxygen demand with an inability to supply the damaged myocardium as a result of coronary thrombosis.

**Nausea**

Nausea results from the stimulation of the medullary vomiting centre and often precedes vomiting (Porth 2007). The nausea a patient experiences with the development of an ACS may range from mild to severe and may indeed be accompanied with vomiting. Hypoxia can exert a direct effect on the vomiting centre and may account for the nausea and vomiting that occur in decreased cardiac output (Porth 2007). Nausea is frequently accompanied by autonomic nervous system manifestations such as excess salivation, vasoconstriction, sweating and pallor. The nausea Peter is experiencing seems to be explained by both the severity of the chest pain and the autonomic nervous system changes that take place due to the ACS.

**Shortness of breath**

Due to decreased blood flow to the affected myocardium, oxygen reserves are rapidly utilized. As a result, there is a change from aerobic to anaerobic metabolism, and, as Porth (2011) explains, this results in a striking loss of myocardial contractile function. The shortness of breath may be manifested by an increase in the respiratory rate together with a reduction in oxygen saturations. This increase in respirations may be due to the possible development of pulmonary oedema as the contractile function of the left ventricle deteriorates. Furthermore, due to the oxygen requirements of the myocardium, there is a need to increase the respiratory rate. While Peter is complaining of shortness of breath, his resulting observations dictate a patient who is compensating reasonably well with oxygen saturations of 95 per cent.

**Outline the nursing care that Peter should receive in relation to the problems identified.**

Relief of pain may reduce the patient’s anxiety and also halt the body’s autonomic nervous system requirements. This in turn will have the effect of reducing cardiac workload (Antman 2008). The nurse should work in a calm, confident manner and explain that pain relief will be provided.

Obtaining a complete and detailed clinical history is essential when evaluating patients with suspected ischaemic coronary syndromes (Scirica 2010). Henderson (2010) explains that most patients with suspected ACS present with prolonged episodes of chest pain. Careful assessment of the patient’s chest pain is essential. The pain could be assessed using the PQRST (Position, Quality, Radiation, Severity and Time) mnemonic as described by Cole et al. (2006). The position or location of cardiac pain is usually diffuse rather than localized to a single point. Pain with ACS could be precipitated by exercise and, as Henderson (2010) suggests,
usually lasts longer than 20 minutes. The pain may radiate to the back, arms (particularly the left), neck, lower face and even upper abdomen (Naik et al. 2007). Furthermore, Naik et al. suggest the pain may be described as ‘discomfort’, ‘tightness’, ‘heaviness’ or ‘gripping’.

Patients with suspected ACS complaining of chest pain should be given sublingual glyceryl trinitrate (GTN) and opioid analgesia to relieve symptoms of chest pain (Henderson 2010). GTN is a vasodilator and acts to increase blood supply to the heart muscle. Nitrates can also be given via the buccal route (Katzung 2004). When given sublingually, the nurse should continue to assess the patient’s pain level, and as nitrates can lead to hypotension, the blood pressure should also be monitored. Porth (2011) explains that the vasodilating effects of nitrates decrease both preload (venous return) and afterload (arterial blood pressure). The net result of nitrates is to reduce myocardial oxygen consumption. Caution should be taken with patients who are hypotensive, or have any condition that limits cardiac output (Karch 2008). Furthermore, Karch (2008) explains that nitrates should be used with caution in patients with suspected cerebral haemorrhage (due to the possibility of increasing intracranial bleeding following relaxation of cerebral vessels). Aspirin 300 mg (dispersible) may also be given in the initial stage as it helps to reduce platelet aggregation and adhesion. Porth (2011) explains that aspirin is thought to promote reperfusion and reduce the likelihood of further thrombosis.

Although several analgesics can be used to treat the pain of ACS, the opiates, in particular morphine (or diamorphine), are usually the drug of choice (Smith and Whitwam 2006). Intravenous morphine (or diamorphine) should be titrated to control the patient’s symptoms. However, as the Resuscitation Council (UK) (2011) explains, sedation and respiratory depression should be avoided. An antiemetic should also be given with the opiate analgesic in order to decrease levels of nausea. Tissues, mouthwash and an emesis bowl should also be provided.

Oxygen may also be used but, as the Resuscitation Council (UK) (2011) explains, nurses must remember to stay within the current guidelines, i.e. to keep the oxygen saturations between 94–98 per cent in patients without chronic obstructive pulmonary disease (COPD). For patients with COPD, the recommended level is between 88 and 92 per cent. However, the patient should be encouraged to sit in an upright position to help lung fields expand.

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**Check point: treatment memory aids**

**MOVE**
- M = Cardiac monitoring and regular monitoring of observations
- O = Oxygen (if necessary)
- V = Venous access and remember to take bloods
- E = 12-lead ECG

**MONA**
- M = Morphine or diamorphine
- O = Oxygen (if necessary)
- N = Nitrates
- A = Aspirin
Discuss the ECG that has been recorded with reference to the diagnosis.

A 12-lead ECG should be recorded as a matter of urgency during the initial assessment (Figure 1.1). Porth (2011) recommends that as well as a 12-lead ECG, cardiac monitoring should be instigated immediately. While the importance of recording an ECG should not be underestimated, Morris and Whiteside (2008) suggest that it should be interpreted in the light of the clinical information. These authors explain that the ECG is not perfect and can be normal in the early stages of ST segment elevation myocardial infarction (STEMI).

![Figure 1.1 Acute anterior STEMI](http://library.med.utah.edu/kw/ecg/mml/ecg_12lead028.html)

Peter’s ECG shows ST segment elevation in leads V2, V3, V4 and to a lesser extent in V5. This is typical of an anterior ST elevation myocardial infarction (Hatchett and Thompson 2007). This ECG indicates that Peter should be treated urgently with a view to reperfusion therapy. Reperfusion therapy involves either thrombolytic therapy or being taken to the cardiac catheterization laboratory in order that a stent may be inserted in the culprit coronary artery.

Discuss the relevance of taking a high-sensitivity troponin T

A number of isoforms of the troponin complex exist. These are troponin I, T and C and they exist on the thin filament of myofibrils and regulate skeletal and cardiac muscle contraction (Henderson 2010). Troponins are sensitive and are markers of myocardial injury. Cardiac troponin begins to rise within 4 hours of the myocardial infarction and peaks between 24–48 hours. However, care should be taken with the interpretation of troponins. While a troponin rise does signify myocardial damage, it is not specific to ACS. Scirica (2010) explains that
troponins can be elevated in patients with sepsis or pulmonary emboli, and while there is myocardial damage, it is not caused by ACS.

High-sensitivity troponins now offer greater sensitivity and earlier detection of myocardial injury. Melanson et al. (2007) report that 64 per cent of patients with initial negative troponin results actually had raised levels when using the high-sensitivity assay. While the use of highly sensitive troponins may lead to better diagnosis, caution should be exercised as Sabatine et al. (2009), in a small-scale study, found that exercise testing and transient stress-induced ischaemia were also associated with an elevated highly sensitive troponin rise. Troponin should therefore be used as an adjunct to both clinical assessment and the ECG interpretation in assisting the physician make a diagnosis.

Other bloods in particular potassium levels should also be observed. This is assayed on the urea and electrolytes (U&E) sample. The potassium should be kept within normal limits (usually 3.5–5.0 mmol/L). A potassium level outside these normal limits should be corrected in order to reduce the possibility of triggering cardiac arrhythmias (Resuscitation Council (UK) 2011). The full blood picture is taken to check for any signs such as a drop in haemoglobin, abnormal levels of platelets or white cells. A low level of haemoglobin can trigger chest pain and may warrant further investigation. Low levels of platelets may have implications for administering antiplatelet drugs and again warrants further investigation.

The cardiologist has decided to take Peter to the cardiac catheterization laboratory (cath lab) to undergo a percutaneous coronary intervention (PCI) as opposed to giving him thrombolytic therapy. The cardiologist wants Peter to have clopidogrel 600 mg and aspirin 300 mg prior to transfer to the catheterization laboratory.

Briefly discuss the benefits of PCI in comparison to thrombolytic therapy.

Randomized controlled trials suggest a significant benefit in favour of early invasive angiography together with the deployment of a stent. These trials (Mehta et al. 2005; NICE 2010) suggest an early invasive strategy is associated with a lower mortality and risk of recurrent ischaemia. The optimal timing for coronary angiography has not been elicited but for patients in a high risk group (early STEMI), then the benefits are greatest with significant reductions in risk of death if undertaken early.

Discuss the modes of action of clopidogrel and aspirin and their use prior to PCI.

Both clopidogrel and aspirin are antiplatelet drugs and decrease the formation of the platelet plug. These drugs are used effectively in the treatment of cardiovascular diseases. Clopidogrel is known as an adenosine diphosphate (ADP) inhibitor and, as such, limits platelet adhesion and aggregation by stopping one pathway to platelet adhesion (Rang et al. 2003). Aspirin works by inhibiting the synthesis of a substance known as thromboxane A2 (Karch 2011). Patients requiring PCI are effectively ‘loaded’ with both drugs prior to the procedure. The dosage is determined by the cardiology consultant but is normally 300 mg for both drugs followed by aspirin 75 mg and clopidogrel 75 mg (Royal Pharmaceutical Society of Great Britain 2008).
Following Peter’s transfer to the cath lab, he had a stent deployed to his left anterior descending coronary artery. Subsequently Peter made a full uncomplicated recovery and was discharged four days later.

Key points

- A 12-lead ECG should be recorded as a matter of urgency during the initial assessment. Troponins are sensitive and are markers of myocardial injury.
- Randomized controlled trials suggest a significant benefit in favour of early invasive angiography together with the deployment of a stent.

REFERENCES


