UPDATE
Module 1647

This module covers:
- The key risk factors for myocardial infarction, including ethnicity and gender, and its incidence in the UK
- The possible presentations of MI and how a diagnosis is confirmed
- The initial treatment and management in secondary care

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Myocardial infarction: part 1

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Coronary heart disease (CHD) is the most common cause of death in the UK, and the average incidence of myocardial infarction (MI) for those aged between 30 and 69 years is about 600 per 100,000 for men (0.6 per cent), and 200 per 100,000 for women (0.2 per cent). The incidence increases with age and elderly people also tend to have higher rates of morbidity and mortality from their infarcts. The cost of prescriptions for treating cardiovascular diseases is £1.6 billion per year.

Risk factors for MI
A variety of genetic and potentially modifiable lifestyle factors increase the likelihood that a person will develop atheromatous plaques and, later, heart attack. The most easily recognised of these risk factors include high cholesterol, diabetes, hypertension, a family history of premature coronary disease, a sedentary lifestyle with limited physical exercise and cigarette smoking.

In the UK, the highest rates of CHD disease mortality are in people born in India, Pakistan and Bangladesh. South Asians are thought to have a 40 to 60 per cent higher risk of CHD-related mortality compared with other populations.

Results of studies involving patients living in 52 countries identified nine factors that accounted for 90 per cent of the population-attributable risk for MI in men and 94 per cent in women. These factors were:

- smoking – any tobacco in last 12 months
- diabetes
- hypertension
- abdominal obesity
- high Apo-B: Apo-A1 ratio (ratio of apolipoprotein-B, a constituent of atherogenic lipoproteins to apolipoprotein-A1, a component of anti-atherogenic high-density lipoprotein (HDL) cholesterol)
- inadequate daily consumption of fruit and vegetables
- psychosocial: a combination of depression, work or home stress, financial stress or one or more life events
- exercise – less than four hours per week
- low alcohol consumption – less than three times per week (moderate intake is protective).

Lifestyle changes implemented early in life have the potential to substantially reduce the risk of acute MI. Five of the modifiable risk factors – smoking, poor diet, physical inactivity, alcohol consumption and being overweight/obese – together accounted for 80 per cent of the risk.

Pathology of MI
Myocardial infarction is part of a spectrum referred to as acute coronary syndrome (ACS). This refers to a range of acute myocardial ischaemia that includes unstable angina, NSTEMI (non-ST segment elevation myocardial infarction), where a coronary artery is partially blocked, and STEMI (ST segment elevation myocardial infarction), where an artery is completely blocked.

An acute MI is caused by necrosis of myocardial tissue due to ischaemia, usually due to blockage of a coronary artery by a thrombus. The rupture of an atheromatous plaque causes adhesion and aggregation of platelets, which initiates the clotting cascade leading to thrombus formation.

The patient’s description of the nature of the pain, whether it is intermittent or prolonged, the quality of the pain, any exacerbating factors and radiation to other areas, needs to be considered.

Three quarters of patients who develop acute MI present with characteristic central or epigastric chest pain radiating to the arms, shoulders, neck or jaw. The pain, which lasts for more than 15 minutes and may last for many hours, usually feels like a heaviness or tightness and may also spread to the arms, neck, jaw, face, back or stomach. Other symptoms include sweating, light-headedness, nausea or shortness of breath.
Sometimes a heart attack may be silent and produce little discomfort. Atypical presentations are also common and symptoms may include abdominal discomfort or jaw pain. They are often seen in women, older men, people with diabetes and people from ethnic minorities. Elderly patients may present with an altered mental state.

**Diagnosis of MI**

The criteria currently used for diagnosing myocardial infarction are detection of rise and/or fall of cardiac biomarkers, together with evidence of myocardial ischaemia from an electrocardiogram (ECG) and/or imaging evidence. Cardiac biomarkers include myocardial muscle creatine kinase (CK-MB) and troponin.

CK-MB is the most specific cardiac enzyme, and its levels rise and fall within the first 24 hours of an MI, with a peak concentration 24 hours post-infarction. Cardiac troponins T and I are the preferred markers for myocardial injury as they have the highest sensitivities and specificities for the diagnosis of acute MI. Troponin serum levels increase within three to 12 hours from the diagnosis of acute MI. Troponin levels rise and fall within three to 12 hours from the onset of chest pain, peak at 24 to 48 hours and return to baseline over five to 14 days. Levels may not be detectable for six hours after the onset of myocardial cell injury.

The diagnosis of acute MI requires documentation of changing troponin concentrations in the first 24 hours, with at least one value being above the diagnostic threshold. Troponins are often used in stratifying risk in patients with an acute MI as they indicate the patient’s progress.

Other biochemical and blood tests contribute to the assessment and prognostic diagnosis. An ECG gives valuable clues to identify the site of myocardial damage, while a coronary angiogram allows visualisation of narrowing or obstructions in the heart vessels.

**Treatment of MI**

The longer the blood supply to the myocardium is occluded, the greater the amount of heart muscle lost. Nearly half of potentially salvageable myocardium is lost within one hour of the coronary artery being occluded and two thirds is lost within three hours.

Patients experiencing symptoms of suspected MI should call the emergency services immediately. A large national advertising campaign has been used to increase awareness among the public and healthcare professionals of the common symptoms of acute MI. Mobile coronary care units are successful in providing immediate care.

The main focus of treatment is to salvage myocardial tissue and prevent further complications; oxygen, aspirin and GTN are all used. Morphine and diamorphine can be used if GTN is not effective, but these may increase mortality in patients with NSTEMI.

In the acute stages of MI the medicines administered are:
- Oral aspirin 300mg (and 75mg daily thereafter if not contraindicated)
- Oral clopidogrel
- Oxygen 40 per cent, at a rate of 5l/min
- Intravenous diamorphine 2.5 mg to 5 mg and repeated as necessary
- Intravenous metoclopramide (or other antiemetic)
- GTN spray, if not hypotensive
- Thrombolytic therapy: if administered, this should be given within 30 minutes of attendance.

Primary percutaneous coronary intervention (PCI) 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; this has emerged as the reperfusion strategy of choice for people with STEMI provided it can be delivered in a timely fashion, and is becoming increasingly available for initial patient care.

For patients who cannot be offered PCI within 90 minutes of diagnosis, a thrombolytic drug is administered. For pre-hospital thrombolysis NICE recommends retapase or tenecteplase, administered by bolus injection, rather than infusion.

Streptokinase and alteplase are given by intravenous infusion. Alteplase, reteplase and streptokinase need to be given within 12 hours of symptom onset; tenecteplase should be given as early as possible, usually within six hours of symptom onset.

The main risks associated with thrombolysis are bleeding complications. Streptokinase should only be administered once, as antibodies can reduce the effectiveness of subsequent doses.

On admission to the coronary or intensive care unit, a beta-blocker is administered intravenously and continued orally thereafter. Beta-blockers are contraindicated in patients with asthma, peripheral ischaemia and pulmonary oedema.

Unfractionated heparin (UFH) is started to help reduce risk from deep venous thrombosis. A low-molecular-weight heparin, fondaparinux, or the glycoproteins 11b/11a (abciximab/epitifibatide) are alternatives if UFH is contraindicated, and are used to prevent re-infarction and to help maintain coronary artery patency.

An angiotensin-converting enzyme (ACE) inhibitor is started in patients with abnormal left ventricular function to help reduce long-term mortality, recurrent heart failure and re-infarction.

Therapy with an ACE inhibitor is usually initiated three to 16 days after infarction and dosing is titrated upwards to the maximum tolerated or target dose. Renal function, electrolytes and blood pressure must be measured before commencing therapy, and again within one to two weeks.

Intravenous nitrates and calcium antagonists are not routinely recommended in the acute phase of treatment of MI. Patients with a significantly reduced cardiac ejection fraction (less than or equal to 40 per cent) and either diabetes or clinical signs of heart failure, should receive the aldosterone antagonist eplerenone (started within three to 14 days of the myocardial infarction, ideally after ACE inhibitor therapy) unless contraindicated.

Arrhythmias must be diagnosed and managed, and any cases of undiagnosed diabetes need to be identified and treated.

A statin or other cholesterol-lowering agent should be started as soon as possible for all patients. Advice on healthy lifestyles, healthy eating, stopping smoking and cardiac rehabilitation are also essential. Social and psychological support is important given the high incidence of anxiety and depression among patients post-infarction and its association with poor outcomes.

Hospital length of stay after acute MI has steadily decreased because of both improved treatments and cost considerations. In most cases, patients are encouraged to return to work within three months. Patients who have suffered a cardiac arrest or undergone a coronary artery bypass graft procedure generally take longer to recover and may require up to six months off work.

**References**

2. Patient.co.uk website, accessed March 2013
CPD Zone Update

5 minute test

Sign up to take the 5 Minute Test and get your answers marked online: www.chemistanddruggist.co.uk/update

Take the 5 Minute Test

1. Coronary heart disease is the second most common cause of death in the UK.
   True or false?
2. The average incidence of MI in men aged 30 to 69 years is about 0.6 per cent.
   True or false?
3. People born in India, Pakistan and Bangladesh have the highest recorded rates of CHD disease mortality in the UK.
   True or false?
4. Three quarters of patients who develop acute MI present with central or epigastric chest pain radiating to the arms, shoulders, neck or jaw.
   True or false?
5. Within an hour of a coronary artery being occluded, nearly half of potentially salvageable myocardium is lost.
   True or false?
6. For pre-hospital thrombolysis, Nice recommends intravenous infusion of reteplase or tenecteplase.
   True or false?
7. Streptokinase needs to be given within six hours of symptom onset.
   True or false?
8. If ACE inhibitor therapy is required, it is usually initiated three to 16 days after infarction.
   True or false?
9. Intravenous nitrates and calcium antagonists are not routinely recommended in the acute phase of treatment of MI.
   True or false?
10. All patients with MI should be started on a statin or another cholesterol-lowering agent as soon as possible.
    True or false?

Tips for your CPD entry on myocardial infarction

Reflect What are the main risk factors for coronary heart disease and myocardial infarction (MI)? Which cardiac biomarkers are used for MI diagnosis? How soon after symptom onset should thrombolytic agents be given?

Plan This article provides information for pharmacists about myocardial infarction. Incidence and risk factors are discussed as well as symptoms, criteria for diagnosis and the drugs used in immediate treatment.

Act Read the article and the suggested reading (below), then take the 5 Minute Test (left). Update subscribers can then access their answers and a pre-filled CPD logsheet.

Read more about myocardial infarction on the NHS Choices website
http://tinyurl.com/coronary1
Find out more about cardiac enzymes from the Patient UK website
http://tinyurl.com/coronary2
Read more about the management of myocardial infarction on the Patient UK website
http://tinyurl.com/coronary3
Think about how you could raise patient awareness of what to do if someone has a myocardial infarction; useful information can be found on the British Heart Foundation website
http://tinyurl.com/coronary4

Evaluate Are you now confident in your knowledge of the symptoms and risk factors for myocardial infarction? Are you familiar with its management and could you give advice about this to patients?

Ask your questions on cardiovascular disease

Do you have a question on MI or any other cardiovascular topic? From therapy choices to risk factors, our specialist is on hand to help. Submit your questions now via chris.chapman@ubm.com

Update 2013

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