Acute Myocardial Infarction

An acute myocardial infarction is caused by necrosis of myocardial tissue due to ischaemia, usually due to blockage of a coronary artery by a thrombus. Most myocardial infarctions are anterior or inferior but may affect the posterior wall of the left ventricle to cause a posterior myocardial infarction. Nearly half of potentially salvageable myocardium is lost within one hour of the coronary artery being occluded, and two thirds are lost within three hours. [1]

Definition

Myocardial infarction is now considered part of a spectrum referred to as acute coronary syndrome (ACS). This refers to a spectrum of acute myocardial ischaemia that also includes unstable angina and non-ST-segment elevation myocardial infarction (NSTEMI). [2]

The new criteria for diagnosing myocardial infarction are detection of rise and/or fall of cardiac biomarkers (preferably troponin) with at least one value above the 99th percentile of the upper reference limit, together with evidence of myocardial ischaemia with at least one of the following: [3, 4]

- Symptoms of ischaemia.
- ECG changes indicative of new ischaemia (new ST-T changes or new left bundle branch block (LBBB)).
- Development of pathological Q-wave changes in the ECG.
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.
- Identification of an intracoronary thrombus by angiography or autopsy.

Epidemiology [5]

- Coronary heart disease (CHD) is the most common cause of death in the UK. CHD is responsible for the deaths of approximately one in five men and one in ten women.
- It is estimated that about 103,000 people in the UK have a myocardial infarction each year. About 60% occur in men.
- Mortality rates after ST-elevation myocardial infarction (STEMI) are equal at 30 days, if both sexes receive equivalent care. Mortality rates become significantly higher for women three years after discharge. [6]
- Premenopausal women appear to be protected from atherosclerosis.
- Incidence increases with age and elderly people also tend to have higher rates of morbidity and mortality from their infarcts.
- Incidence rates of myocardial infarction are lower in the South of England compared with the North of England, Scotland and Northern Ireland.
- Incidence rates tend to be higher in urban areas than in rural areas.

Risk factors

- Non-modifiable risk factors for atherosclerosis include increasing age, being male, family history of premature CHD, premature menopause.
- Modifiable risk factors for atherosclerosis include smoking, diabetes mellitus (and impaired glucose tolerance), metabolic syndrome, hypertension, hyperlipidaemia, obesity and physical inactivity. [7]
- Certain ethnic groups have higher risk of CHD. In the UK, the highest recorded rates of coronary artery disease mortality are in people born in India, Pakistan and Bangladesh. [8] South Asians are thought to have a 40-60% higher risk of CHD-related mortality compared to other populations.
Presentation

- Chest pain (central chest pain may not be the main symptom):
  - Three quarters of patients present with characteristic central or epigastric chest pain radiating to the arms, shoulders, neck, or jaw.
  - The pain is described as substernal pressure, squeezing, aching, burning, or even sharp pain.
  - Radiation to the left arm or neck is common.
  - Chest pain may be associated with sweating, nausea, vomiting, dyspnoea, fatigue and/or palpitations.

- Shortness of breath: may be the patient’s anginal equivalent or a symptom of heart failure.
- Atypical presentations are common and tend to be seen in women, older men, people with diabetes and people from ethnic minorities. Atypical symptoms include abdominal discomfort or jaw pain; elderly patients may present with altered mental state.

Signs

Cardiovascular examination findings can vary enormously:

- Low-grade fever, pale and cool, clammy skin.
- Hypotension or hypertension can be observed depending on the extent of the myocardial infarction.
- Dyskinetic cardiac impulse (in anterior wall myocardial infarction) can be palpated occasionally.
- Third and fourth heart sound, systolic murmur if mitral regurgitation or ventricular septal defect develops, pericardial rub.
- There may be signs of congestive heart failure, including pulmonary rales, peripheral oedema, elevated jugular venous pressure.

Assessment for possible acute coronary syndrome[^4]

- Consider the history of the pain, any cardiovascular risk factors, history of CHD and any previous treatment, and previous investigations for chest pain.
- Symptoms that may indicate ACS include:
  - Pain in the chest and/or other areas (e.g., the arms, back or jaw) lasting for longer than 15 minutes.
  - Chest pain with nausea and vomiting, marked sweating and/or breathlessness, or haemodynamic instability.
  - New-onset chest pain, or abrupt deterioration in stable angina, with recurrent pain occurring frequently with little or no exertion and often lasting longer than 15 minutes.

- The response to glyceryl trinitrate (GTN) should not be used to make a diagnosis and symptoms should not be assessed differently in men and women or among different ethnic groups.
- Patients with pre-existing angina should be advised that when an attack of angina occurs, they should:[^9]
  - Stop what they are doing and rest.
  - Use GTN spray or tablets as instructed.
  - Take a second dose of GTN after five minutes if the pain has not eased.
  - Take a third dose of GTN after a further five minutes if the pain has still not eased.
  - Call 999/112/911 for an ambulance if the pain has not eased after another five minutes (i.e., 15 minutes after onset of pain), or earlier if the pain is intensifying or the person is unwell.

Differential diagnosis

see also the separate Chest Pain and Cardiac-type Chest Pain Presenting in Primary Care articles.

- Cardiovascular: stable angina, another form of ACS (unstable angina or NSTEMI), acute pericarditis, myocarditis, aortic stenosis, aortic dissection, pulmonary embolism.
- Respiratory: pneumonia, pneumothorax.
- Gastrointestinal: oesophageal spasm, gastro-oesophageal reflux disease, acute gastritis, cholecystitis, acute pancreatitis.
- Musculoskeletal chest pain.

Consider non-atherosclerotic causes of myocardial infarction in younger patients or if there is no evidence of atherosclerosis: coronary emboli from sources such as an infected cardiac valve, coronary occlusion secondary to vasculitis, coronary artery spasm, cocaine use, congenital coronary anomalies, coronary trauma, increased oxygen requirement (e.g., hyperthyroidism) or decreased oxygen delivery (e.g., severe anaemia).

Investigations

- If diagnosis is suspected, immediately arrange urgent hospital assessment and admission. Call 999/112/911 ambulance.
- ECG:
  - May be helpful in a pre-hospital setting if the diagnosis is uncertain or in a remote area in the assessment for pre-hospital thrombolysis but otherwise should not delay getting the patient to hospital.
  - Features may initially be normal but abnormalities include new ST-segment elevation; initially peaked T waves and then T-wave inversion; new Q waves; new conduction defects.
  - Do not exclude an ACS when people have a normal resting 12-lead ECG.
In hospital

- FBC to rule out anaemia; leukocytosis is common; monitor potassium levels (electrolyte disturbances may cause arrhythmias, especially potassium and magnesium); renal function - estimated glomerular filtration rate (eGFR) - should be measured prior to starting an angiotensin-converting enzyme (ACE) inhibitor. Lipid profile needs to be obtained at presentation because levels can change after 12-24 hours of an acute illness. Measure C-reactive protein (CRP) and other markers of inflammation.
- Cardiac enzymes:
  - See also separate Cardiac Enzymes and Markers for Myocardial Infarction article.
  - Cardiac troponins T and I are highly sensitive and specific for cardiac damage. The risk of death from an ACS is directly related to troponin level and 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 normal initially and should be repeated.
  - Myocardial muscle creatine kinase (CK-MB) is found mainly in the heart. 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. Sensitivity and specificity are not as high as for troponin levels.
- Serial ECGs and continuous ECG monitoring in a coronary care unit (CCU).
- CXR: to assess the patient's heart size and the presence or absence of heart failure and pulmonary oedema. This may also assist in differential diagnosis.
- Pulse oximetry and blood gases: monitor oxygen saturation.
- Cardiac catheterisation and angiography: cardiac angiography defines the patient's coronary anatomy and the extent of the disease.
- Echocardiography can define the extent of the infarction and assess overall ventricular function and can identify complications, such as acute mitral regurgitation, left ventricular rupture or pericardial effusion.
- Myocardial perfusion scintigraphy using single photon emission computed tomography (SPECT): the National Institute for Health and Care Excellence (NICE) recommends that myocardial perfusion scintigraphy using SPECT should be the first test used for:

  - People where stress ECG may not give accurate or clear results - eg, women, people who have certain unusual patterns in the electrical activity of their heart, people with diabetes or people for whom exercise is difficult or impossible.
  - The diagnosis of people who are less likely to have coronary artery disease and who are at lower risk of having heart problems in the future. The likelihood of a person having coronary artery disease can be assessed by considering a number of factors - eg, age, sex, ethnic background and family history as well as the results of physical examination and investigations.
  - As an investigation in people who still have symptoms following a myocardial infarction or despite having had treatment to improve coronary artery blood flow.

Management

See separate Acute Myocardial Infarction Management, Cardiovascular Risk Assessment and Cardiac Rehabilitation articles.

Complications

See separate Complications of Acute Myocardial Infarction article.

Prognosis[5]

- Mortality rates from CHD have been falling in the UK since the late 1970s. Deaths caused by acute myocardial infarction roughly halved in the UK between 2002 and 2012. The mortality of ACS with clinical myocardial infarction treated with modern treatments including thrombolysis has been estimated to be 12-15% within six months of the ACS.[12]
- However, up to 50% of people who have an acute myocardial infarction die within 30 days of the event, and over half of these deaths occur before medical assistance arrives or the patient reaches hospital. About one third of all deaths occur within the first hour, usually as the result of an acute fatal arrhythmia.
- Prognosis correlates with the degree of myocardial necrosis. Greater degrees of myocardial necrosis are associated with a worse prognosis. The degree of myocardial necrosis can be estimated by various factors - for example:
  - The rise in serum troponin T.
  - Degree and extent of ECG changes.
  - Degree of left ventricular dysfunction on echocardiography.
- The prognosis also depends on the timing and nature of intervention; the prognosis is improved with successful early reperfusion, preserved left ventricular function and short-term and long-term treatment with beta-blockers, aspirin, statins and ACE inhibitors.[2]
- Other factors that may adversely affect prognosis following an acute myocardial infarction include:
  - Comorbidities - eg, hypertension, chronic kidney disease, anaemia and diabetes.
  - Site of the infarction - eg, anterior myocardial infarction generally has a less favourable prognosis than inferior myocardial infarction.
  - Depression, particularly treatment-resistant and insufficiently treated depression.
  - Older age and female sex.

Prevention
See separate Prevention of Cardiovascular Disease article.

Further reading & references

- Unstable angina and NSTEMI; NICE Clinical Guideline (March 2010 - last updated November 2013)
- Myocardial infarction (acute): Early rule out using high-sensitivity troponin tests (Elecsys Troponin T high-sensitive, ARCHITECT STAT High Sensitive Troponin-I and AccuTnI+3 assays); NICE Diagnostics Guidance (October 2014)

1. Myocardial infarction with ST-segment elevation: The acute management of myocardial infarction with ST-segment elevation; NICE Clinical Guideline (July 2013)
2. 2015 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation; European Society of Cardiology (August 2015)
3. Third Universal Definition of Myocardial Infarction; European Society of Cardiology (2012)
5. MI - secondary prevention; NICE CKS, October 2015 (UK access only)
9. Angina; NICE CKS, October 2015 (UK access only)
11. Myocardial perfusion scintigraphy for the diagnosis and management of angina and myocardial infarction; NICE Technology Appraisal Guidance, November 2003
12. Management of Acute Myocardial Infarction in patients presenting with ST-segment elevation; European Society of Cardiology (2012)

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