Acute Chest Pain

Objectives

By reading this material you should be able to

- Appreciate the importance of chest pain as a presenting symptom
- Feel more confident about the recognition and early management of chest pain
- Recognise common clinical symptoms associated with cardiac disease

Background

Chest pain accounts for up to 50% of all acute medical admissions and a significant proportion of these will be due to cardiac causes. Despite advances in both pharmacological and interventional strategies, ischaemic heart disease still remains the biggest cause of death in the UK. The early and correct diagnosis of cardiac chest pain and the subsequent initiation of appropriate therapies have been shown to significantly reduce both morbidity and mortality.

It is important to be aware of the differential diagnosis, to help to differentiate cardiac pain from other causes of chest pain. The more common are listed below, in table 1.

Table 1 - Causes of chest pain

<table>
<thead>
<tr>
<th>Cardiac</th>
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<tbody>
<tr>
<td>Acute coronary syndrome</td>
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<tr>
<td>• ST elevation MI</td>
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<tr>
<td>• Non ST elevation MI</td>
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<tr>
<td>• Unstable angina</td>
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<tr>
<td>Aortic dissection</td>
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<tr>
<td>Pericarditis</td>
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<tr>
<td>Myocarditis</td>
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<table>
<thead>
<tr>
<th>Respiratory</th>
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<tr>
<td>Pulmonary embolism</td>
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<tr>
<td>Pneumothorax</td>
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<table>
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<tr>
<th>Gastrointestinal</th>
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<tr>
<td>Peptic ulcer disease</td>
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<tr>
<td>Oesophageal perforation</td>
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<tr>
<th>Musculoskeletal</th>
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<td>Costochondritis</td>
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The initial management of all patients presenting with chest pain should always start with ABCDE assessment. You will be able to carry this out on an actor on your study day.

Remember: if the patient is presenting acutely unwell, there is sometimes not time to elicit a full history prior to commencing treatment. Under these circumstances

A. The airway should be checked and oxygen administered
B. The breathing and ventilation status of the patient should be assessed. Apply pulse oximetry.
C. The circulation should be assessed by feeling the peripheries, instituting i.v. access and measuring pulse and blood pressure.
D. Assess the patients disability – in this case their conscious level
E. Make sure the patient is undressed to facilitate examination. Make sure the patient is neither too hot or too cold.

The cause of the pain should then be quickly sought so as to commence the appropriate treatment, such as thrombolysis, rapidly.

**History**
After assessing the patients in the ABCDE approach, specific points in the history should be sought in order to elicit the likely cause. These include:

- nature - type, location, radiation, severity, aggravating and relieving factors
- time of onset
- duration
- risk factors – eg. Diabetes, smoking, hypertension, family history
- prior history of ischaemic heart disease
- previous treatment with and contraindications to angioplasty and thrombolysis

Myocardial ischaemia is typically associated with a heavy or crushing sensation that may radiate to the neck and jaw. There may also be heaviness or tingling in the left arm and patients may complain of nausea, vomiting, breathlessness or sweating. Atypical pain should not exclude ischaemia but alternate diagnoses, for example pericarditis, should also be considered. Heartburn is a common presentation of an inferior myocardial infarction.

**Examination**
Clinical examination can often be normal in patients presenting with an acute coronary syndrome (ACS). Examination is therefore directed towards identifying complications of the ACS (eg. Arrhythmias, acute heart failure), or establishing an alternative diagnosis.

Signs of an arrhythmia:
- irregular pulse
- fast or slow heart rate
- low blood pressure
- Reduced conscious level if blood pressure is very low
Signs of heart failure:

Right heart
- peripheral oedema
- pulsatile liver edge
- raised JVP

Left heart
- reduced air entry or crackles in the lung bases.
- tachycardia (because the heart is unable to pump much blood out with each beat, so it beats faster to compensate)
- cool peripheries with a prolonged capillary refill time.
- In severe heart failure, the blood pressure may be low.

Alternative diagnosis

Aortic dissection
- differential pulses (depends on level of dissection, but may be difference between left and right radial, or radial and femoral pulses)
- differential blood pressure between left and right arms
- acute aortic regurgitation
- signs of cardiac tamponade (muffled heart sounds, raised JVP)

Pericarditis
- audible pericardial friction rub

Pneumothorax
- hyper-resonance to percussion on affected side
- reduced or absent breath sounds on affected side
- tracheal deviation in a tension pneumothorax

Pulmonary embolism
- signs of a DVT (swollen, oedematous leg)
- occasionally raised JVP (in massive PE)

Perforated oesophagus
- surgical emphysema
- signs of sepsis

Musculo-skeletal
- tenderness to palpation or movement (the presence of this does not rule out a more serious pathology – be very careful when attributing chest pain to the musculoskeletal system)

Investigations

ECG
A 12 lead ECG should be performed on all patients immediately. If the initial ECG is normal and the pain is ongoing then repeat ECGs should be performed at 10-minute intervals to quickly identify any evolving changes. Treatment options dichotomise to those with ST elevation / new Left bundle branch block (LBBB) versus all other changes (normal, ST depression, T wave inversion). The patient should be continuously monitored for arrhythmias.

Bloods
Blood tests should include full blood count, urea and electrolytes, glucose, lipids and appropriately timed cardiac enzymes. Troponin T or I are sensitive and specific markers of myocardial damage and allow the detection of more subtle volumes of myocyte damage than creatine kinase (CK). Current SIGN guidelines recommend checking troponin on arrival at hospital as a positive result can help guide treatment.
A troponin level should also be taken 12 hours after onset of pain, as this is when the troponin level should be at its highest, therefore most likely to be detected.

Arterial blood gases are rarely required, only being indicated in severely unwell patients (eg. following cardiac arrest, cardiogenic shock, severe pulmonary oedema). It should be remembered that access to the arterial system for percutaneous coronary intervention is often via the radial artery, so avoiding damaging the vessel by repeated punctures is advisable. Additionally, if a patient is thrombolysed they are liable to bleed from sites of previous arterial puncture.

**X-ray**
The chest x-ray is likely to be normal in an acute coronary syndrome, so is not generally essential. However, it can be useful where diagnosis is in doubt - eg. if aortic dissection or respiratory/oesophageal pathology is suspected. It is also useful if pulmonary oedema is thought to be present.

**Classification of Acute Coronary Syndromes**
Acute coronary syndrome (ACS) is used to describe a range of clinical conditions including acute ST elevation myocardial infarction, non-ST elevation myocardial infarction and unstable angina.

There is often confusion into the definition of a myocardial infarction. Here the definition proposed by the European Society of Cardiology, the American College of Cardiology and the American Heart Association is used. This is a more sensitive definition than those used previously and requires a typical clinical syndrome plus a rise and fall in troponin (or CK-MB if troponin not available).

Table 2: comparison of definition and prognosis by Troponin T concentration (from SIGN guideline 93)

<table>
<thead>
<tr>
<th>12 hr serum troponin T concentration (mg/l)</th>
<th>&lt; 0.01</th>
<th>≥0.01 and &lt; 1.0</th>
<th>≥1.0</th>
</tr>
</thead>
<tbody>
<tr>
<td>BCS definition</td>
<td>ACS with unstable angina</td>
<td>ACS with myocyte necrosis</td>
<td>ACS with clinical myocardial infarction</td>
</tr>
<tr>
<td>ESC/ACC definition</td>
<td>unstable angina</td>
<td>myocardial infarction</td>
<td>myocardial infarction</td>
</tr>
<tr>
<td>WHO definition</td>
<td>unstable angina</td>
<td>unstable angina</td>
<td>myocardial infarction</td>
</tr>
<tr>
<td>30-day mortality*</td>
<td>4.5%</td>
<td>10.4%</td>
<td>12.9%</td>
</tr>
<tr>
<td>6-month mortality*</td>
<td>8.6%</td>
<td>18.7%</td>
<td>19.2%</td>
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(BCS – British Cardiac Society; ESC – European Society of Cardiology; ACC – American College of Cardiology; WHO – World Health Organisation)

**Management of Acute Coronary Syndromes**
The aims of the acute management of ACS are:

1. Rapidly establish a diagnosis
2. Treat any haemodynamic and acute arrhythmic complications
3. Provide prompt pain relief and adequate arterial oxygen concentrations
4. Initiate rapid reperfusion to limit infarct size and minimise the risk of pump failure and arrhythmias
5. Treat any early complications
6. Risk assessment for longer term management and commence secondary prevention

Patients should be rapidly assessed on arrival to hospital as previously described. All patients (unless strongly contra-indicated) should be treated with aspirin 300mg, followed by 75mg daily.
Sublingual GTN spray can be used for pain control. The effects of this are short lived.
Patients should be placed on cardiac monitoring to allow prompt identification of arrhythmias.

Any patient with any ECG changes consistent with ischaemia (ST changes, T-wave inversion) or a raised troponin should also be treated with clopidogrel 300mg (ticagrelor in GG&C)
The ECG findings are used to further divide patients into the STEMI or NSTEMI/Unstable angina groups, which dictates further treatment.

**ST Elevation Myocardial Infarction (STEMI)**

STEMI occurs when a coronary artery becomes acutely occluded with thrombus following the rupture of an atheromatous plaque.

The diagnosis of a STEMI is confirmed by:
- history of cardiac sounding chest pain
- a limited examination for shock / murmurs / complications
- 12 lead ECG criteria:
  - 1mm ST elevation in 2 contiguous limb leads
  - 2mm ST elevation in 2 contiguous chest leads
  - New left bundle branch block
  - True posterior infarct (dominant R wave and ST depression in V1 + V2).

**Primary percutaneous coronary intervention**

Reperfusion by primary percutaneous coronary intervention (PPCI), if performed promptly and by an experienced team, has been shown to be superior to thrombolysis in terms of:
- overall mortality
- reinfarction rates
- improved left ventricular systolic function
- stroke.³

PPCI also has the advantage of giving visual evidence that the occluded artery has been reopened and normal flow has been restored, as well as information about disease in other coronary arteries.

**Thrombolysis**

The greatest benefit is gained within the first 2 hours of onset of pain with a subsequent progressive decrease of 1.6 deaths per hour of delay, per thousand patients treated.¹ The benefit does persist up to 6 hours though with some evidence for giving it up to 12 hours in the presence of persistent pain and ECG changes. Beyond 12 hours there is no clear evidence whether the benefit outweighs the risk of major bleeding.
Table 2 – Contraindications to Thrombolysis

<table>
<thead>
<tr>
<th>Absolute</th>
<th>Relative</th>
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<tbody>
<tr>
<td>• Haemorrhagic CVA at any time</td>
<td>• Severe, uncontrolled hypertension</td>
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<tr>
<td>• Any CVA within 1 year</td>
<td>• Current use of anticoagulants</td>
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<tr>
<td>• Known intracerebral neoplasm</td>
<td>• Known bleeding diathesis</td>
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<tr>
<td>• Active internal bleeding</td>
<td>• Recent trauma e.g. head trauma, prolonged CPR</td>
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<tr>
<td>• Suspected aortic dissection</td>
<td>• Major surgery within 3 weeks</td>
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<tr>
<td></td>
<td>• Non-compressible vascular punctures</td>
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<tr>
<td></td>
<td>• Recent internal bleeding</td>
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<tr>
<td></td>
<td>• Pregnancy</td>
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<td></td>
<td>• Active peptic ulcer</td>
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<td></td>
<td>• History of chronic severe hypertension</td>
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</table>

Figure 2 – Acute ST elevation myocardial infarction (inferior)

Optimal Reperfusion for STEMI – West of Scotland

The aim of this service is to restore myocardial perfusion quickly and effectively in all STEMI patients. PPCI is more effective and associated with less complications assuming it can be delivered quickly.

If the patient presents within 6 hours of pain (i.e. they are a candidate for PPCI or thrombolytic):

PPCI is the preferred strategy if the patient can get to the interventional centre within 60mins (assumes total 90 mins to open vessel)

Otherwise thrombolysie and transfer
If the patient presents outwith 6 hours they are not a candidate for thrombolytic and should be transferred immediately for PPCI

All patients with cardiogenic shock should be transferred immediately for PPCI and not thrombolysed

Some contraindications to thrombolytic may also be relative contraindications to PPCI (esp. active bleeding as aspirin, clopidogrel and heparin are the minimum – some patients may also require GPIIbIIIa depending on the clot burden / lesion)

All patients with STEMI should be discussed with the interventional centre
   GJNH for most of the West of Scotland
   Hairmyres for Lanarkshire / Ayrshire + Arran

**Failed Reperfusion**

The REACT study has clarified the management of patients who fail to respond to thrombolysis (as evidenced by continuing chest pain and failure of ECG resolution). In patients where the ECG at 90 minutes after thrombolysis demonstrates that the ST segment in the worst lead has failed to reduce by 50%, “rescue” angioplasty should be considered.

This means it is important that all thrombolysed patients are transferred to the interventional centre as soon as possible, such that rescue PCI can be carried out should they fail to reperfuse.

**Other Acute Coronary Syndromes**

This includes both non-ST elevation myocardial infarctions (NSTEMI) and unstable angina (troponin negative ACS). These are usually mediated by platelet activation rather than thrombin formation. More often they are due to an incomplete occlusion of a coronary artery.

**Diagnosis**

The first priority is to establish a working diagnosis based upon:
- The clinical picture
- ECG
- Cardiac biomarkers including troponin.

The ECG may be normal but typical ischaemic changes are ST depression, dynamic ST elevation and T wave inversion.

**Management**

The risks of a NSTEMI or unstable angina continue over time, with the maximum risk being in the first 3 months, as a result of further thrombotic events. Whilst in hospital it is important to regularly check for further pain. Any further pain should prompt further ECG and repeat cardiac enzymes. The patient should be continuously monitored for arrhythmias and any haemodynamic compromise.

**Pharmacological Treatment**

1. Reduce platelet aggregation – as for all acute coronary syndromes.
- Aspirin 300mg once then 75mg daily
- Clopidogrel 300mg once then 75mg daily: guidelines now recommend use of this in addition to aspirin in all NSTEMIs for at least 3 months.

2. Reduce thrombin formation
   - Either low molecular weight heparin (eg. enoxaparin, dalteparin) or fondaparinux.

3. Increase oxygen delivery to the heart
   - IV GTN (dilates coronary vessels)
   - Oxygen (if saturations low)

4. Reduce the work of the heart
   - Beta-blockers (only if no evidence of heart failure, bradycardia or hypotension)
   - If blood pressure is high, reducing this will reduce the work the heart needs to do (again IV GTN works well)

5. Analgesia
   - Morphine/Diamorphine

6. Secondary prevention
   - Statins
   - ACE inhibitors

There is debate over the timing of the use of glycoprotein IIb/IIIa receptor blockers in acute coronary syndromes. Most of their benefit occurs when they are used during percutaneous coronary intervention, rather than when they are administered as part of medical therapy before possible angioplasty (so called “upstream” use)

Figure 3 – Acute management of NSTEMI / unstable angina

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Suspected ACS
No ST elevation

Serial ECGs
Oxygen & analgesia
Aspirin/clopidogrel/heparin/β blocker
Secondary prevention

Presence of high risk features*
Refer to cardiology as in patient
Coronary angiogram +/- PCI

Absence of high risk features
Exercise tolerance test

Intermediate / high risk result
Low risk result

Unlikely to be cardiac pain
Discharge home
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High risk features

- Prolonged pain
- Raised troponin
- Significant ECG changes
- Haemodynamic instability
- Rhythm instability
**Aortic Dissection**
Aortic dissection occurs because of a tear in the intimal lining, which then propagates along the media of the aorta and its branches. This should be suspected especially in patients with known hypertension or connective tissue disease.

**Presentation**
Symptoms are usually of severe, tearing retrosternal chest pain, often radiating through to the back and intrascapular region. More rarely it can present with dyspnoea, sudden paralysis if the cerebral or spinal arteries are affected, or limb ischaemia. The patient is usually tachycardic and the pulse may be of low volume. Blood pressure may or may not be raised and there may be a pulse and blood pressure deficit between the arms. If the dissection extends to the aortic root an early diastolic murmur of aortic regurgitation may be heard, or there may be evidence of tamponade or ST elevation on the ECG - NB thrombolysing this is likely to be fatal!

**Investigations**
Diagnostic investigations – choice depends on clinical condition, and availability:
- Contrast CT
- Transoesophageal echo

Other investigations
- Chest X-ray – look for widened mediastinum (present in 60% cases)
- Transthoracic echo – only proximal aorta seen, therefore lacks sensitivity & specificity in the detection of a flap, but will identify pericardial fluid and aortic regurgitation if present

**Management**
If the dissection affects the ASCENDING aorta urgent referral to the cardiothoracic surgeons is required.

If only the DESCENDING aorta is affected the mainstay of treatment is analgesia and maintenance of systolic blood pressure 100 – 110mmHg (e.g. with IV labetalol). The patient should be continuously monitored either on a coronary care or high dependency unit. It is important to watch for signs of haemodynamic compromise or extension of dissection (often heralded by more pain), that may lead to loss of peripheral pulses, renal or cerebral infarction etc.

**Acute Pericarditis**
The commonest causes of pericarditis are viral (most frequently parvovirus B19 or coxsackie) and post myocardial infarction.

The presentation is typically of sharp, substernal chest pain that is relieved by sitting forward and made worse by lying down. The pain may be aggravated by movement and inspiration. On examination the patient may be pyrexial and there may be a friction rub audible.

The ECG is diagnostic in pericarditis. In the acute phase there is concave ST elevation (so called “saddle shaped”) in a number of leads (those facing the
epicardial surface). This elevation later normalises and T wave inversion may develop. Cardiac enzymes may be elevated if there is associated myocarditis.

Treatment is anti-inflammatory drugs e.g. indomethacin 50mg tds.

**Myocarditis**

Myocarditis may present in association with pericarditis or as an acute disease characterised by a febrile illness and heart failure. The cause is frequently unidentified and can be idiopathic, infective (viral, bacterial or protozoal) or autoimmune.

An ECG may demonstrate ST and T wave changes, again affecting a number of leads. There may also be arrhythmias and occasionally heart block. Cardiac enzymes will be elevated due to inflammation of the myocardium. Investigations include viral antibody titres, transthoracic echo which may show a regional wall motion abnormality and rarely, endomyocardial biopsy.

The mainstay of treatment is the management of heart failure and arrhythmias.

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**SUMMARY**

Chest pain is a very common symptom in medical patients

Don’t forget airway, breathing and circulation!

In the acute ST elevation MI - TIME IS MUSCLE. The goal is to open the artery as quickly as possible, with PPCI or thrombolysis if there is a delay to PPCI.

Primary angioplasty is a more effective treatment for acute MI.

**References**


**Further Reading**

