THE PATIENT PRESENTS WITH

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**Objectives**

By the end of this chapter you should:
- be able to take a clear history from a patient presenting with chest pain
- be aware of the differential diagnosis of chest pain
- be able to examine the patient with chest pain
- understand the appropriate investigations for a patient presenting with chest pain.

**Differential Diagnosis of Chest Pain**

Chest pain is one of the most common presenting complaints seen by cardiologists. It is important to remember that:

- there are many causes of chest pain
- some causes of chest pain are life-threatening and require prompt diagnosis and treatment; other causes are more benign.

The first differentiation to be made is between cardiac and non-cardiac chest pain (Fig. 1.1).

**History to Focus on the Differential Diagnosis of Chest Pain**

The differential diagnosis of chest pain is very diverse; a thorough history is, therefore, very important.

**Presenting Complaint**

Differentiation depends on a detailed history of the pain, with particular emphasis on the following characteristics of the pain (Fig. 1.2):

- Whether the pain is continuous or intermittent
- Duration of the pain

- Position of the pain – central or lateral/posterior
- Exacerbating factors – exertion, emotion, food, posture, movement, breathing
- Radiation of the pain – to neck, arms, head
- Quality of pain – crushing, burning, stabbing.

To get an idea of the severity of a patient's symptoms ask them about their ‘effort tolerance’ – how far can they walk on the flat? Do they have stairs at home, and does walking up them cause chest pain?

**Past Medical History**

This can provide important clues:

- A history of ischaemic heart disease
- A history of peptic ulcer disease, or of frequent ingestion of non-steroidal anti-inflammatory drugs
- Recent operations – cardiothoracic surgery can be complicated by Dressler’s syndrome, mediastinitis, ischaemic heart disease or pulmonary embolus (PE)
- Pericarditis might be preceded by a prodromal viral illness
- PE can be preceded by a period of inactivity (e.g. a recent operation, illness or long journey)
Chest pain

EXAMINATION OF PATIENTS WHO HAVE CHEST PAIN

Points to note on examination of the patient who has chest pain are shown in Fig. 1.3.

Inspection
On inspection, look for:

- signs of shock (e.g. pallor, sweating) – may indicate myocardial infarction (MI), dissection aorta, PE
- laboured breathing – may indicate MI leading to left ventricular failure (LVF) or a pulmonary cause
- signs of vomiting – suggests MI or an oesophageal cause
- coughing – suggests LVF, pneumonia.

Cardiovascular system
Note the following:

- Pulse and blood pressure – is there any abnormal rhythm, tachycardia, bradycardia, hypotension, hypertension? Inequalities in the pulses or blood pressure between different extremities are seen in aortic dissection.
- Mucous membranes – pallor could suggest angina due to anaemia; cyanosis suggests hypoxia.
- Any increase in jugular venous pressure – a sign of right ventricular infarction or pulmonary embolus.
- Carotid pulse waveform – a collapsing pulse is seen with aortic regurgitation, which can complicate aortic dissection. It is slow rising if angina is due to aortic stenosis.
- Displaced apex beat, abnormal cardiac impulses (e.g. paradoxical movement in anterior MI).
- Auscultation – listen for a pericardial rub, third heart sound (a feature of LVF), mitral or aortic regurgitation (features of MI or dissection respectively), aortic stenosis (causes angina).

Respiratory system
Note the following signs:

- Breathlessness or cyanosis
- Unequal hemithorax expansion – a sign of pneumonia and pneumothorax
- Abnormal dullness over lung fields – a sign of pneumonia

Drug history, family history and social history
Other risk factors for ischaemic heart disease, such as a positive family history and smoking, should be excluded. A history of heavy alcohol intake is a risk factor for gastritis and peptic ulcer disease.

Hypertension is a risk factor for both ischaemic heart disease and dissection of the thoracic aorta.
Any bronchial breathing or pleural rub – signs of pneumonia and pleurisy.

Gastrointestinal system
Specifically look for:
- abdominal tenderness or guarding
- scanty or absent bowel sounds – suggests an ileus (e.g. due to perforated peptic ulcer and peritonitis).

### INVESTIGATION OF PATIENTS WHO HAVE CHEST PAIN

A summary of tests used to investigate chest pain is shown in Fig. 1.4; an algorithm is given in Fig. 1.5.

### Blood tests
These include:
- cardiac biomarkers including cardiac troponin and creatine kinase – cardiac troponin T and I are now commonly used to risk stratify patients presenting with acute coronary syndrome (Fig. 1.6)
- full blood count – anaemia may exacerbate angina
- renal function and electrolytes – may be abnormal if the patient has been vomiting, leading to dehydration and hypokalaemia, or due to diuretic therapy
- arterial blood gases – hypoxia is a sign of PE and LVF; hypcapnoea is seen with hyperventilation
- liver function tests and serum amylase – deranged in cholecystitis and peptic ulcer disease.
Chest pain

Electrocardiography

Findings may include:

- ST elevation in absence of bundle branch block (BBB) – indicates acute MI (occasionally it is due to Prinzmetal’s angina)

Chest radiography

The following signs may be seen:

- Cardiomegaly
- Widening of the mediastinum in aortic dissection

Fig. 1.3 Points to note when examining a patient who has chest pain. AR, aortic regurgitation; AS, aortic stenosis; BP, blood pressure; JVP, jugular venous pressure; LVF, left ventricular failure; MI, myocardial infarction; MR, mitral regurgitation; P2, pulmonary component of the second heart sound.

Fig. 1.4 First-line tests to exclude a chest pain emergency

<table>
<thead>
<tr>
<th>Test</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>ECG</td>
<td>If normal excludes MI, although evidence for this may emerge upon observation</td>
</tr>
<tr>
<td>CXR</td>
<td>Widened mediastinum suggests aortic dissection; may show pleural effusion or pulmonary consolidation</td>
</tr>
<tr>
<td>Biochemical markers</td>
<td>May be normal in first 4 h after MI, but CK-MB, cardiac troponins will then increase</td>
</tr>
<tr>
<td>Arterial blood gases</td>
<td>In the dyspnoeic patient severe hypoxaemia suggests pulmonary embolus, LVF or pneumonia</td>
</tr>
<tr>
<td>CT scan</td>
<td>Carry out urgently for suspected aortic dissection</td>
</tr>
</tbody>
</table>

Diagnosis

- ST depression in absence of BBB – indicates myocardial ischaemia. At rest this equates with unstable angina or non-Q wave infarction; on exertion this equates with effort-induced angina pectoris or tachyaryrhythmias
- BBB – if new this may be due to MI; if it is old, MI cannot be diagnosed from the electrocardiogram (ECG)
- fully developed Q waves – indicate old MI (i.e. over 24-h old)
- atrial fibrillation secondary to any pulmonary disease or myocardial ischaemia.

In the event of a large PE the classic changes are:

- sinus tachycardia (or atrial fibrillation)
- tall P waves in lead II (right atrial dilatation)
- right axis deviation and right BBB
- S wave in lead I, Q wave in lead III, and inverted T wave in lead III (SI QIII TIII pattern seen only with very large PE).
Lung lesions
Pleural and pericardial effusions
Oligaemic lung fields in PE.

**Echocardiography**
This may reveal:
- pericardial effusion – suggests pericarditis or dissection
- regional myocardial dysfunction – a feature of MI or ischaemia
- aortic dissection with false lumen
- aortic or mitral valve abnormalities.

**Computed tomography and magnetic resonance imaging**
These are the most sensitive methods for excluding aortic dissection and should be performed urgently if this diagnosis is suspected.
It is sometimes possible to visualize PE with spiral computed tomography (CT).

**Ventilation/perfusion scan**
This excludes PE in most cases if performed promptly. If the V/Q scan is negative and PE is strongly suspected, a more sensitive test is a pulmonary angiogram.
Chest pain

**Exercise tolerance test or myocardial perfusion scan**
This may be performed at a later date if angina is suspected.

**CENTRAL CHEST PAIN AT REST OF RECENT ONSET IN AN ILL PATIENT**
The importance of this subject is that this situation represents a medical emergency requiring rapid diagnosis and treatment.

**Contraindications to fibrinolytic therapy**

**Absolute contraindications**
- Haemorrhagic stroke or stroke of unknown origin at any time
- Ischaemic stroke in preceding 6 months
- Central nervous system damage or neoplasms
- Recent major trauma/surgery/head injury (within preceding 3 weeks)
- Gastrointestinal bleeding within the last month
- Known bleeding disorder
- Aortic dissection.

**Relative contraindications**
- Transient ischaemic attack in preceding 6 months
- Oral anticoagulant therapy
- Pregnancy or within 1 week postpartum
- Non-compressible punctures
- Traumatic resuscitation
- Refractory hypertension (systolic blood pressure >180 mmHg)
- Advanced liver disease
- Infective endocarditis
- Active peptic ulcer.

It is necessary in this situation to distinguish between the following:
- MI (see Ch. 10)
- Unstable angina (see Ch. 9)
- Pericarditis (see Ch. 17)
- Dissection of thoracic aorta
- PE
- Mediastinitis secondary to oesophageal tear
- Non-cardiac chest pain.

**Thrombolysis**

**Conditions for which thrombolysis is contraindicated**
The following guidelines help differentiate MI from disorders in which thrombolysis can be fatal.

**Pericarditis**
In pericarditis:
- the patient may have a prodromal viral illness
- pain can be exacerbated by breathing movements
- there might be concomitant indications of infection.
- examination might reveal a pericardial rub – an added sound (or sounds) in the cardiac area on auscultation. This has a scratchy quality and seems close to the ears. If complicated by pericardial effusion, there could be an:
  - impalpable cardiac impulse
  - increased cardiothoracic ratio on the chest radiograph, with a globular heart shadow
- the ECG shows characteristic concave – upwards raised ST segments in all leads except AVR.

Thrombolysis is contraindicated because it causes haemopericardium.

**Dissection of the thoracic aorta**
The pain is sharp and tearing. There is often radiation of the pain to the back. There might be a previous history of hypertension.

On examination, the patient might be shocked and there could be delays between the major pulses (e.g. right brachial versus left brachial, brachial versus femoral).

Chest radiography might show a widened mediastinum. The ECG will not show ST elevation unless the coronary ostia are dissected. Confirmation might require
high-resolution spiral CT, echocardiography, or magnetic resonance imaging (which is the best investigation when available; Fig. 1.7).

Thrombolysis is contraindicated because it causes massive bleeding from the aorta (Fig. 1.8).

**Mediastinitis**

This is unusual and need not usually be considered unless there is a possibility of an oesophageal leak (e.g. after endoscopy or oesophageal surgery).

### Pulmonary embolus

Pulmonary emboli can present as acute chest pain in an ill patient or as intermittent chest pain in a relatively well patient. For this reason it is crucial to suspect PE in all patients who have chest pain that is not typically anginal.

The pain of a PE can be pleuritic or tight in nature and might be located anywhere in the chest. It can be accompanied by the following symptoms and signs:

**Fig. 1.7 Overview of dissection of the thoracic aorta**

<table>
<thead>
<tr>
<th>Predisposing factors</th>
<th>Hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bicuspid aortic valve</td>
<td></td>
</tr>
<tr>
<td>Pregnancy</td>
<td></td>
</tr>
<tr>
<td>Marfan’s, Turner’s, Noonan’s syndromes</td>
<td></td>
</tr>
<tr>
<td>Connective tissue diseases – SLE, Ehlers–Danlos syndrome</td>
<td></td>
</tr>
<tr>
<td>Men &gt; women</td>
<td></td>
</tr>
<tr>
<td>Middle age</td>
<td></td>
</tr>
</tbody>
</table>

| Pathophysiology | Damage to the media and high intraluminal pressure causing an intimal tear |
| Blood enters and dissects the luminal plane of the media creating a false lumen |

| Classification | Stanford classification: type A – all dissections involving the ascending aorta; type B – all dissections not involving the ascending aorta |

| Symptoms | Central tearing chest pain radiating to the back |
| Further complications as the dissection involves branches of the aorta: coronary ostia – myocardial infarction; carotid or spinal arteries – hemiplegia, dysphasia, or paraplegia; mesenteric arteries – abdominal pain |

| Signs | Shocked, cyanosed, sweating |
| Blood pressure and pulses differ between extremities |
| Aortic regurgitation |
| Cardiac tamponade |
| Cardiac failure |

| Investigation | CXR – widened mediastinum ± fluid in costophrenic angle |
| ECG – may be ST elevation |
| CT/MRI – best investigations, show aortic false lumen |
| Transoesophageal echo if available is also very sensitive |
| Echocardiography – may show pericardial effusion if dissection extends proximally; tamponade may occur |

| Management | Pain relief – diamorphine |
| Intravenous access – central and arterial line |
| Fluid replacement – initially colloid then blood when available – crossmatch at least 10 units |
| Blood pressure control – intravenous nitroprusside infusion or labetolol infusion if no cardiac failure – keep blood pressure 120/80 mmHg |
| Surgery for all type A dissections |
| Medical management and possibly surgery or percutaneous treatment for type B |

*CT, computed tomography; CXR, chest radiography; ECG, electrocardiography; MRI, magnetic resonance imaging; SLE, systemic lupus erythematosus.*
Chest pain

Fig. 1.8 Classification of aortic dissections.

- Dyspnoea
- Dry cough or haemoptysis
- Hypotension and sweating
- Sudden collapse with syncope.

Massive PE will cause collapse with cardiac arrest. The ECG will show ventricular tachyarrhythmias or sinus rhythm with electromechanical dissociation. Patients will often experience a sense of ‘impending doom’ or profound anxiety.

Conditions predisposing to clot formation in the deep veins of the leg are associated with a high incidence of PE (Fig. 1.9).

As the mortality rate resulting from PE is approximately 10%, appropriate investigations to exclude PE should be carried out promptly and anticoagulation commenced using either intravenous heparin as an infusion or an appropriate low-molecular-weight heparin preparation subcutaneously. Warfarin therapy should be commenced if PE is confirmed.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Immobility</td>
<td>Prolonged bed rest for any reason, long air journeys</td>
</tr>
<tr>
<td>Postoperative</td>
<td>Abdominal and pelvic surgery, leg and hip surgery</td>
</tr>
<tr>
<td>Haemoconcentration</td>
<td>Diuretic therapy, polycythaemia</td>
</tr>
<tr>
<td>Hypercoagulable states</td>
<td>Malignancy, oral contraceptive pill, protein C/protein S deficiency, etc.</td>
</tr>
<tr>
<td>Venous stasis (poor flow of venous blood)</td>
<td>Congestive cardiac failure, atrial fibrillation (formation of thrombus in the right ventricle can result in PE)</td>
</tr>
</tbody>
</table>