# Chest Pain And Acute Coronary Syndromes

## *Executive summary*

## Introduction

Cardiovascular disease represents the leading cause of morbidity and mortality worldwide. Chest pain secondary to myocardial ischaemia in the forms of stable angina and acute coronary syndromes (ACS), often referred to as a ‘heart attack’, represent perhaps the most well-known presentations of cardiovascular disease (table 1). The prevalence of cardiovascular disease and resultant ACS continues to rise in low-income settings. Despite this cardiac chest pain and ACS are understudied and have poor outcomes in Sub-Saharan Africa*.*

## Target users

* Nurses
* Doctors

## Target area of use

* Outpatient department
* Ward

## Key areas of focus / New additions / Changes

This guideline discusses how to identify cardiac chest pain, how to classify severity and the management of both chronic cardiac chest pain and acute coronary syndromes.

## Limitations

None

## Background and definitions

Chest pain is an extremely common presenting complaint, present in approximately 20% of medical admissions to hospital*.* It is a distressing symptom and can be concerning for both patients and health care professionals – often due to worries about potentially life threatening aetiologies such as ACS. The majority of chest pain is not cardiac in origin though (table 2) and can be managed as an outpatient. The wide variety of possible differential diagnoses (table 3) can make the task of identifying the underlying cause of chest pain and treating it appropriately extremely daunting. The key to the management of these patients is a thorough assessment to help identifying the underlying cause and early recognition of signs of possible life threatening aetiologies.

Table 1: Important terms in chest pain related to cardiovascular disease

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| **Term** | **Explanation** |
| Ischaemic Heart Disease | Also referred to as coronary heart disease, is present when a patient has one or more symptoms, signs, or complications from an inadequate supply of blood to the myocardium. This is most commonly due to obstruction of the epicardial coronary arteries due to atherosclerosis. |
| Angina | Occurs when myocardial oxygen demand exceeds oxygen supply; the clinical manifestation is often chest discomfort. |
| Stable Angina | Refers to chest discomfort that occurs predictably and reproducibly at a certain level of exertion and is relieved with rest or nitrates. Most patients with ischaemic heart disease will experience angina as part of the clinical manifestations of the disease. |
| Acute Coronary Syndrome (ACS) | Applied to patients in whom there is a suspicion or confirmation of acute myocardial ischaemia or infarction. Non-ST elevation myocardial infarction (NSTEMI), unstable angina (UA), and ST-elevation myocardial infarction (STEMI) are the three types of ACS. |
| STEMI | Myocardial ischaemia with raised markers of myocardial injury (troponin, CK-MB) and characteristic ECG changes (predominantly ST elevation). |
| NSTEMI | Myocardial ischemia with raised markers of myocardial injury without the characteristic ECG changes of STEMI. |
| Unstable Angina | Myocardial ischemia without raised markers of myocardial injury. |

Table 2: Aetiology of chest pain

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|  | **Percentage of Patients Presenting** |
| Musculoskeletal | 30 – 50% |
| Gastro-intestinal | 10 – 20% |
| Stable Angina | 10% |
| Respiratory Conditions | 5% |
| Acute Coronary Syndromes | 2 – 4% |

Table 3: Differential diagnoses of chest pain

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| **Cardiac** | | | |
| **Ischaemic** | Stable Angina | **Non**-**Ischaemic** | Aortic Dissection |
|  | Acute Coronary Syndrome |  | Heart Failure |
|  |  |  | Pericarditis/Myocarditis |
| **Pulmonary** | | | |
|  | Pulmonary Embolism | Asthma and COPD | |
|  | Pneumonia | Pulmonary Hypertension | |
|  | Malignancy | Pulmonary Tuberculosis | |
| **Gastro-intestinal** | | | |
|  | Oesophagitis | Gastroesophageal reflux disease | |
|  | Oesophageal rupture/perforation | | |
| **Musculoskeletal** | | | |
|  | Isolated musculoskeletal pain – i.e. costochrondritis | | |
|  | Trauma | Inflammatory Conditions | |
| **Psychiatric** |  |  | |
|  | Panic Attacks/Disorder | Factitious Disorder | |
| **Other** |  |  | |
|  | Referred Pain | Herpes Zoster | |

## Presenting symptoms and Signs

Given the variety of possible diagnoses underlying chest pain, thorough history taking is of the utmost importance.

Early assessment should focus on symptoms and signs of chest pain with possible life-threatening aetiologies. Any patients in whom there are concerns for life-threatening causes of chest pain should be referred urgently to the ward for further assessment:

* Unstable vital signs.
* Abrupt onset of chest pain and/or clinical concern for life-threatening aetiology (e.g. pulmonary embolism, aortic dissection, esophageal rupture, tension pneumothorax).
* Concern for acute coronary syndrome (ACS) based on symptoms (anginal symptoms at rest, prolonged, or progressing episodes).

Assess patients presenting with chest pain for typical signs of angina (table 4). Classic symptoms of stable angina include:

* A pressure, heaviness, tightness, or constriction in the center or left of the chest that is precipitated by exertion and relieved by rest.
* Provocation with emotional stress or cold
* Radiation (to the neck, jaw, and shoulder)
* Dyspnoea
* Nausea and vomiting
* Sweating

Severity of pain is not a useful differentiating factor and it is important to note no symptom alone or in combination identifies a group of patients in whom the diagnosis can be entirely excluded.

The clinical presentation of myocardial ischemia varies by population. Women, diabetics, and older adult patients are more likely to present without chest pain but have symptoms of dyspnoea, weakness, nausea and vomiting, palpitations, or syncope*.* Compared with older patients, younger patients are less likely to have stable angina and have a higher incidence of ACS*.*

Patients who present with ACS have:

* Anginal symptoms at rest (usually greater than 20 minutes)
* New-onset angina that is not stable and predictable
* Progressive symptoms (angina that is more frequent, longer in duration, or occurs with less exertion than previously).

These patients should be admitted urgently.

Table 4: Assessment of chest pain

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| **Stratification of Chest Pain** | |
| **Typical Angina** | Presence of three of the three features of anginal chest pain |
| **Atypical Angina** | Presence of two of the features anginal chest pain |
| **Non-Anginal Chest Pain** | Presence of one or none of the features of anginal chest pain |
| **Features of Anginal Chest Pain** | |
| Constricting discomfort in the front of the chest, or in the neck, shoulders, jaw or arm | |
| Precipitated by physical exertion | |
| Relieved by rest or GTN within about 5 minutes | |
| **Features Suggestive of Non-Anginal Chest Pain** | |
| Pleuritic pain, sharp or knife-like pain related to respiratory movements or cough | |
| Primary or sole location in the mid or lower abdominal region | |
| Any discomfort localized with one finger | |
| Any discomfort reproduced by movement or palpation | |
| Constant pain lasting for days | |
| Fleeting pains lasting for a few seconds or less | |
| Pain radiating into the lower extremities or above the mandible | |

## Examination

A focused examination of the patient with chest pain should include a cardiovascular, respiratory and abdominal examination, thereby broadly assessing the most likely underlying aetiologies. In many cases there may be few findings on examination and further investigation will be warranted.

## Investigations

Patients with typical signs of stable angina and no other concerning features should be commenced on anginal therapy, but investigations (table 5) may be warranted if there are concerns about other possible causes.

ECGs may be limited in the outpatient setting due to the cost to patients, but should be used if they will help make a diagnosis or change management plans. Concerns over ACS should prompt referral to the ward where an ECG can be performed as an in-patient.

Patients with atypical cardiac chest pain should be investigated further. If there is no other clear cause, and especially in patients with a high number of cardiac risk factors (age, smoking, hypertension, diabetes, dyslipidaemia, family history of coronary heart disease, etc) they should be commenced on anginal treatment and monitored closely for response to therapy.

Patients with non-anginal chest pain should be investigated further unless a clear diagnosis of a non-life threatening aetiology can be made clinically. For example clear musculoskeletal chest pain or a clear (low risk) pneumonia may not require any further investigation.

Patients admitted to the ward with chest pain should have urgent bloods (full blood count, urea and electrolytes, Creatine kinase MB (CK-MB) if concerns over cardiac pain), an ECG and a chest x-ray. Other investigations may also be required depending on the clinical situation.

Table 5: Investigation of patients with chest pain

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| **Investigation** | **Role and Interpretation** |
| **ECG** | A standard 12-lead electrocardiogram (ECG) should be obtained for all patients presenting with chest pain that may be from an ACS.  ECG remains the best immediately available test for detecting ACS but its sensitivity for acute myocardial infarction (AMI) is low. The initial ECG is often **NOT** diagnostic in patients with ACS and can be repeated as frequently as every 10 minutes if the initial ECG is not diagnostic but the patient remains symptomatic and there remains high clinical suspicion for AMI. Prior ECGs are important for determining whether abnormalities are new. The presence of a left bundle branch block makes it difficult to determine the presence of ischemic ECG changes.  Findings consistent with ST elevation myocardial infarction (STEMI): New ST elevation at the J point in two anatomically contiguous leads using the following diagnostic thresholds: ≥0.1 mV (1 mm) in all leads other than V2-V3, where the following diagnostic thresholds apply: ≥0.2 mV (2 mm) in men ≥ 40 years; ≥0.25 mV (2.5 mm) in men <40 years, or ≥0.15 mV (1.5 mm) in women.  Findings consistent with Non ST elevation myocardial infarction or unstable angina: New horizontal or down-sloping ST depression ≥0.05 mV (0.5 mm) in two anatomically contiguous leads and/or T inversion ≥0.1 mV (1 mm) in two anatomically contiguous leads with prominent R wave or R/S ratio >1. |
| **Chest X-Ray (CXR)** | A CXR should be performed in all patients unless a probable underlying cause that does not require imaging has already been identified in a stable patient.  A nondiagnostic CXR is typical in patients with **ACS and stable angina.or pulmonary embolus (PE)**  Approximately 90 percent of patients with **acute aortic dissection** will have some CXR abnormality  Pneumonia and pneumothorax are often diagnosed by CXR.  Acute heart failure is suggested by pulmonary vascular congestion and cardiomegaly. In patients with severe vomiting or recent instrumentation of the oesophagus, mediastinal emphysema and pleural effusion suggest o**esophageal rupture**. |

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| **Blood Tests** | |
| **CK-MB** | Creatine kinase MB (CK-MB) isoform levels rise to twice normal at six hours and peak within approximately 24 hours post myocardial injury. Note should be made of the time pain started and when the test was taken. An initial test should be performed on admission for anyone who may have ACS and a repeat after 6-9 hours to assess for ongoing changes,  Although it is no longer recommended for most patients being evaluated for ACS due to the higher accuracy of troponin assays, it is the available option here at present. |
| **Full Blood Count** | The white blood cell count may be elevated in any of the inflammatory or infectious etiologies of chest pain. Anaemia in a patient with exertional chest pain is suggestive of myocardial ischemia, but also consistent with aortic rupture. |
| **Urea and Electrolytes** | Raised urea and other markers indicating dehydration and acute kidney injury may accompany many forms of chest pain. Abnormalities in these tests often indicates a poorer prognosis. |
| **Further Possible Investigations** | |
| **Transthoracic Echocardiogram** | May show regional wall abnormalities in patients with acute coronary syndromes or prior myocardial injury.  May show signs of right heart strain and features of raised pulmonary pressure in pulmonary embolism.  Will demonstrate significant valvular disease and left ventricular dysfunction if present. |
| **USS Doppler (lower legs)** | Can be used as a surrogate investigation if there are concerns over a (non-massive) pulmonary embolism. |

## Management

Non-cardiac causes of chest pain such as pneumonia and musculoskeletal pain should be managed as described in their separate guidance. The following will focus on the management of stable angina and ACS.

### Management of stable angina

*Anti-anginal therapy*:

1. **Beta-blockers**

First line therapy to reduce anginal episodes and improve exercise tolerance. The only antianginal drugs proven to prevent reinfarction and to improve survival in patients who have sustained a myocardial infarction

Bisoprolol: Start at 1.25 mg OD immediately and uptitrate to 10 mg daily, as tolerated and according to symptomatic response (can be given in divided doses).

Dose can be increased in 1.25-2.5 mg steps, aiming to double every 2 weeks until target dose is reached.

1. **Calcium** **Channel Blockers**

Used in combination with beta blockers when initial treatment with beta blockers is not successful or as a substitute for a beta blocker when beta blockers are contraindicated or cause side effects.

Amlodipine: Start at 5 mg OD and uptitrate to 10 mg OD as tolerated and according to symptomatic response.

This should be initiated following maximal uptitration of a beta-blocker.

1. **Nitrates**

First-line therapy for the treatment of acute anginal symptoms. Usually in the form of a sublingual preparation> Patients should be instructed to use them at the onset of angina. They may also be recommended for the prophylaxis of anginal episodes.

GTN sublingual tablets: 300-500 micrograms as required. Up to three times with 5 minute intervals for relief of acute anginal pain.

*Anti-platelet therapy:*

1. **Aspirin**

In the absence of a contraindication, all patients with definite angina should be treated with aspirin. Patients who have a gastrointestinal bleed on low-dose aspirin should, after the episode is controlled, be treated with aspirin plus a proton pump inhibitor.

Aspirin: 75 mg OD.

1. **Clopidogrel**

Can be given as an **alternative** to aspirin in stable angina if the patient has aspirin intolerance. Available from CSD pharmacy only for staff.

Clopidogrel: 75 mg OD.

*Preventative therapy:*

1. **Statins**

Reduce cholesterol and prevent future cardiovascular disease.

Atorvastatin: 40-80 mg Nocte.

1. **Risk factor reduction**

A central component of the management of patients with stable angina. This includes treatment of hypertension, cessation of smoking, weight reduction, glycaemic control in diabetics and participation in regular physical activity.

### Management of NSTEMI / unstable angina:

*Anti-thrombotic therapy:*

1. **Anti-platelet agents**

Aspirin 300 mg AND clopidogrel 300 mg given as a stat dose as soon as possible.

Ideally aspirin 75 mg and clopidogrel 75 mg OD to continue for 1 year following this.

Aspirin can be used as a monotherapy in the absence of clopidogrel, though with lower efficacy.

1. **Anticoagulation**

Enoxaparin should be started immediately and given for 5 days post ACS (1 mg/kg 12 hourly): Available for members of staff only – patients should be encouraged to buy this if they can.

Unfractionated heparin can be used as an alternative to enoxaparin, but has worse clinical outcomes. We cannot measure APTT out-of-hours, so are not able to use it iv. If there is no alternative then sc heparin can be given at a dose of 15,000 units BD, as this is likely to be better than nothing.

*Anti-ischaemic and analgesic therapy:*

1. **Oxygen**

Administer supplemental oxygen to ACS patients with an arterial saturation less than 90 percent.

1. **GTN**

Sublingual GTN (300-500microgams): Consider intravenous GTN in patients with persistent pain after three sublingual GTN tablets, hypertension, or heart failure.

1. **Morphine**

IV morphine should be avoided if possible and reserved for patients with an unacceptable level of pain. If needed give intravenous morphine 2 to 4 mg, with increments of 2 to 8 mg repeated at 5- to 15-minute intervals.

*Further inpatient management:*

Initiation of anti-anginal medications as per stable angina.

A beta-blocker should be started in the first 24 hours if able.

When stable an ACE inhibitor may be started if there is evidence of heart failure, hypertension or chronic kidney disease.

### Management of STEMI

1. **Fibrinolytic therapy**

Streptokinase 1500000 units (15 million units) to be initiated as soon as possible and within 12 hours of symptom onset as an infusion over 60 minutes. Outside of 12 hours, there is limited evidence for benefit.

Assess for bleeding risk prior to make a risk-benefit decision. Streptokinase is antigenic, and can cause immunologic sensitization and allergic reactions, particularly with repeat administration

Consultant input is advised.

1. **Anti-thrombotic therapy**

Aspirin 300 mg and clopidogrel 300 mg should be given as a stat dose as soon as possible and continued as 75 mg OD following this for 1 year. Aspirin monotherapy can be continued after this.

1. **Anticoagulant Therapy**

As per NSTEMI protocol. Anticoagulate with enoxaparin or unfractionated heparin for 5 days.

1. **Further inpatient management**

As per NSTEMI protocol. Early initiation of a beta-blocker (1st 24 hours) is recommended.

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