Ischaemic heart disease: ST elevation myocardial infarction

Clive Lawson

Abstract

Acute ST elevation myocardial infarction (STEMI) is most commonly caused by thrombotic occlusion of a major epicardial coronary artery, and initial management is geared towards achieving rapid and effective reperfusion. Most patients present with persistent typical cardiac pain and have either ST elevation or (new) left bundle branch block on their ECG. Where STEMI is diagnosed or suspected, patients should be transferred to the nearest heart attack centre. Primary percutaneous coronary intervention is the acute treatment of choice but thrombolysis should be considered if this cannot be delivered within 120 minutes. Complications of STEMI include a wide range of arrhythmias and heart failure, resulting from either pump failure or major mechanical complications that can require urgent surgical intervention. Post-infarction echocardiography is vital in diagnosing mechanical complications, assessing left ventricular (LV) function and detecting LV thrombus. Longer term outcomes are improved by aggressive secondary prevention with a combination of medication and lifestyle interventions supported by a structured cardiac rehabilitation programme. The appropriate choice and duration of antithrombotic medication requires an appraisal of thrombotic and bleeding risk.

Keywords Cardiac rehabilitation; myocardial infarction; percutaneous coronary intervention; secondary prevention; ST elevation; thrombolysis

Key points

- Most patients with acute ST elevation myocardial infarction (STEMI) present with persistent typical cardiac chest pain; however, atypical presentations occur and are more common in elderly, female or diabetic individuals
- The diagnosis is confirmed by the presence of ST segment elevation or new left bundle branch block on a 12-lead electrocardiogram
- When STEMI is confirmed, patients should be loaded with oral dual antiplatelet therapy (DAPT) and rapidly transferred to the nearest heart attack centre for primary percutaneous coronary intervention; if this cannot be delivered within 120 minutes, thrombolysis should be considered
- Adverse events after STEMI include shock, heart failure, arrhythmias and mechanical complications
- Prognosis after STEMI is improved by secondary preventive interventions including DAPT, β-blockers, angiotensin-converting enzyme inhibitors and statins, as well as lifestyle changes and cardiac rehabilitation

Introduction

Ludwig Hektoen identified coronary artery thrombosis as the cause of myocardial infarction (MI) in 1879 but it was not until the 1980s, over a century later, that reperfusion therapy became part of routine clinical practice. The syndrome of ST elevation MI (STEMI) is a high-risk presentation because of the volume of myocardium at risk of permanent damage and a propensity for major acute complications in the absence of timely intervention.

Since the 1980s mortality from STEMI has fallen progressively and sequentially with the introductions of thrombolysis, balloon angioplasty and coronary stenting; however, it remains substantial, with up to 8% of patients dying within 30 days. Clinical services are now orientated towards rapid recognition of STEMI and achieving effective reperfusion as soon as possible, managing complications that arise and reducing long-term morbidity and mortality.

Clive Lawson *MD FRCP is a Consultant Cardiologist at Tunbridge Wells Hospital and Academic Lead for Year 5 at Kent and Medway Medical School, Canterbury, UK. Competing interests: none declared.*

Diagnosis

Clinical presentation

Most individuals with acute MI present with persistent typical squeezing/tight retrosternal cardiac pain, often radiating to the arms or throat or jaw, and usually associated with systemic symptoms (e.g. nausea/vomiting, sweating, weakness, lightheadedness, anxiety, a sense of doom). Others, typically elderly, diabetic or female patients, present with atypical symptoms (e.g. epigastric or back pain, breathlessness, transient loss of consciousness).

Individuals with STEMI usually look unwell with a grey pallor and/or sweating. They are often relatively bradycardic for their clinical presentation, having a 'normal' heart rate despite being in severe pain. Many present with STEMI-related complications including arrhythmias, out-of-hospital cardiac arrest and acute heart failure.

The electrocardiogram (ECG)

If patients present with symptoms consistent with MI, a 12-lead ECG should be performed as soon as possible. Diagnostic criteria for STEMI are given in Table 1. Changes should be present in at least two contiguous ECG leads.

The criteria for leads V1–V2 allow differentiation from the 'high take-off' pattern, which is a benign normal variant, where some ST elevation is present in these leads on the resting ECG. In the earliest stages of STEMI hyperacute changes may be seen, with slurring of the ST segment into the T wave (Figure 1a); this can be difficult to distinguish from the 'high take-off' pattern

ECG criteria for STEMI

ECG pattern	Criterion
J point ST segment elevation:	
• V1–V2 (men <40 years)	>2.5 mm
• V1–V2 (men >40 years)	>2.0 mm
• V1–V2 (women)	>1.5 mm
• All other leads	>1.0 mm
Posterior MI:	
• ST depression in V1–V3	>1.0 mm
LBBB/ventricular pacing:	
• Concordant ST elevation	>1.0 mm (5 points)
• Concordant ST depression (in V1–3)	>1.0 mm (3 points)
• Excessive discordant ST depression	>5.0 mm (2 points)
	Positive if ≥3 points

Table 1

unless there are reciprocal changes, typically ST depression in remote leads. Diagnostic ST elevation and reciprocal changes (Figure 1b) can take time to develop and repeat ECGs may be needed if there is doubt.

If infarction predominantly involves the posterior wall of the left ventricle the major ECG change is ST *depression* in the anterior chest leads (Figure 1c).

Left bundle branch block (LBBB) is problematic as this is common in people with chronic cardiac conditions and some ST elevation is usually present in the anterior leads; it is also impossible to know if LBBB is 'new' unless a previous ECG is available.

The modified Sgarbossa criteria can be helpful if there is LBBB (or ventricular pacing). These have a high specificity but only a moderate sensitivity for acute coronary artery occlusion (Figure 1d), so if the clinical picture is in keeping with a STEMI presentation, the patient should be referred to the regional heart attack centre.

An important ECG pattern where individuals present with suspected MI is ST elevation in lead aVR; this is a marker of extensive ischaemia and most commonly seen with associated widespread ST segment depression in patients with threatened left main-stem occlusion. This is a very high-risk presentation and requires urgent intervention. A variant of this is the de Winter ECG (Figure 2), which is associated with proximal occlusion of the left anterior descending coronary artery.

Differentiating acute pericarditis/myocarditis from STEMI on the ECG can also be difficult; however, the ST elevation in the former typically is widespread, concave upward and not restricted to a single coronary territory. The ECG in pericarditis can show PR segment depression and does not show reciprocal ST depression. Other causes of ST elevation include Brugada syndrome, Takotsubo cardiomyopathy, hyperkalaemia and hypercalcaemia.

STEMI versus occlusive myocardial infarction

Recent analyses have suggested that conventional ST elevation criteria can have a sensitivity of <50% for identifying acute coronary artery occlusion. This has led to an evolving paradigm shift in clinical care with broader criteria for emergency angiography based on the recognition of possible 'occlusive MI'.[1] These include persistent cardiac-type chest pain, haemodynamic or electrical instability, broader ECG criteria and bedside echocardiography (looking for left ventricular (LV) regional wall motion abnormalities).

Initial care

Once the diagnosis has been made the patient should be loaded with dual antiplatelet therapy (DAPT), usually aspirin 300 mg and a potent $P2Y_{12}$ inhibitor, either prasugrel 60 mg or ticagrelor 180 mg. If the person is taking an oral anticoagulant, prasugrel

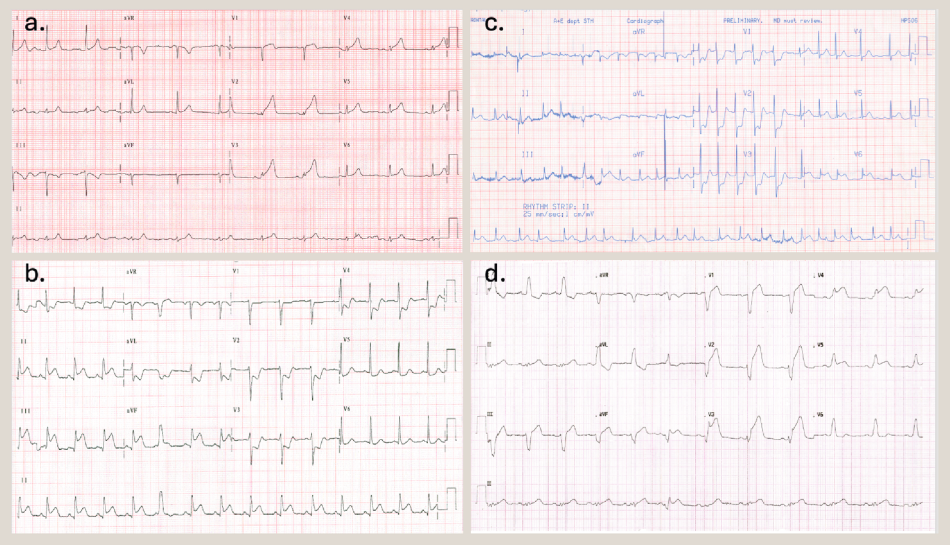

Figure 1 STEMI ECGs. (**a**) Hyperacute anterior STEMI. Note the slurring of the ST segment into the T wave in leads V1–V5. (**b**) Inferior STEMI. Note the clear-cut ST elevation in leads II, III and aVF with reciprocal ST depression in V2–V5, I and aVL. (**c**) Posterolateral STEMI. Note the marked ST depression in V1–V3 with subtle ST elevation in V6. (**d**) Anterior STEMI with LBBB. Note the excessive (>5 mm) discordant ST elevation in V2–V3 and concordant ST elevation in V4–V5.

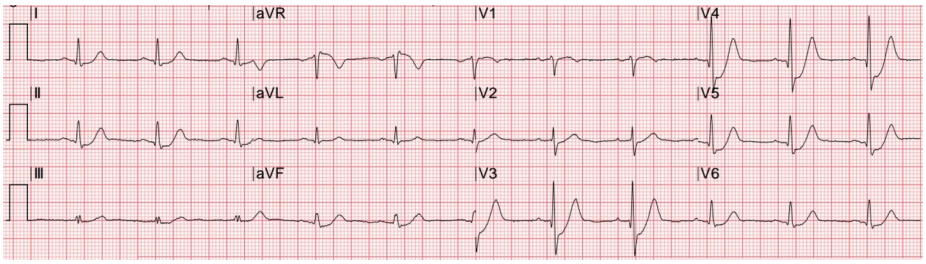

Figure 2 The de Winter ECG. Note the subtle ST elevation in aVR with widespread ST depression and peaked T waves in V3–V6.

and ticagrelor should be avoided, and clopidogrel 600 mg given instead.

Most patients are in considerable distress; if so, they should be given opiates (morphine or diamorphine) and an anti-emetic. Oxygen is only required if the patient is hypoxic (transcutaneous oxygen saturation (SaO_2) <90%).[2] Atropine should be given if the patient is hypotensive and bradycardic. In addition, intravenous fluid should be given if they are hypotensive and have no signs of pulmonary oedema.

Reperfusion strategy

Primary percutaneous coronary intervention (PPCI) is the reperfusion strategy of choice,[2] and most healthcare systems have been designed to ensure this is available 24/7. Most patients are taken directly to the regional heart attack centre by the paramedic team; others require emergency inter-hospital transfer.

In the catheter laboratory, radial artery access for PPCI is preferred over femoral access as the risk of haemorrhagic complications is lower. Initial antithrombotic therapy in the form of unfractionated heparin is usually given once arterial access has been achieved; bivalirudin can be used if heparin is contraindicated (e.g. heparin-induced thrombocytopenia).

The likely culprit vessel can usually be inferred from the ECG (Table 2) but must be confirmed at angiography before proceeding to PPCI. Unless the patient is haemodynamically unstable, baseline angiography of both the left and right coronary arteries is performed; priority is then given to identifying the culprit lesion and crossing it with a guidewire. Achieving 'first medical contact to wire crossing' within 90 minutes is a key performance indicator for PPCI services.[2]

Angiography also allows the identification of non-atherosclerotic pathologies that can present as STEMI. These include spontaneous coronary artery dissection (e.g. pregnancy), thromboembolism (e.g. atrial fibrillation (AF)), spasm (e.g. cocaine use) and Takotsubo cardiomyopathy.

Flow is usually restored after initial balloon dilation(s), after which one or more stents can be deployed: drug-eluting stents are preferred over bare metal stents. Glycoprotein IIb/IIIa inhibitors (e.g. abciximab) and thrombus aspiration can help when thrombus burden is high but are not routinely used. The aim of treatment is to return the flow in the infarct-related vessel to normal (i.e. thrombolysis in myocardial infarction (TIMI) grade III flow, the same rate as in a normal coronary artery).

Where PPCI cannot be delivered in a timely manner, defined as within 120 minutes of diagnosis (e.g. in remote communities), thrombolysis should be considered. The efficacy of thrombolysis is not guaranteed and patients treated in this manner should be transferred to the heart attack centre in case rescue percutaneous coronary intervention (PCI) is required. Reperfusion may be recognized clinically by resolution of the chest pain and >50% resolution of ST elevation compared with the presenting ECG; this is usually assessed 60–90 minutes after treatment.

Likely culprit artery in STEMI based on presenting ECG pattern

ECG pattern	Culprit vessel
ST elevation:	
• V1–V4 (anterior)	LAD or diagonal branch
• V1–V6, I and aVL (anterolateral)	Proximal LAD
• II, III and aVF (inferior)	RCA or distal circumflex
• V5–V6, I and aVL (lateral)	Circumflex, distal LAD or diagonal branch
• aVR (global)	Left mainstem or proximal LAD
ST depression:	
• V1–V3 (posterior)	Circumflex or distal RCA
LBBB	LAD

LAD, left anterior descending artery; RCA, right circumflex artery.

Table 2

Post-reperfusion care

After PPCI patients should be nursed in a high-dependency environment according to their clinical need, usually meaning a dedicated coronary care unit.

High-dose statin therapy (typically atorvastatin 80 mg once a day) is started immediately and DAPT continued. Prasugel is the preferred second agent for STEMI patients treated by PPCI unless the bleeding risk is high or the person has a history of stroke. DAPT is covered with a proton pump inhibitor agent (e.g. lansoprazole) to reduce the risk of gastrointestinal bleeding. Where blood glucose concentrations are high a variable rate intravenous insulin infusion should be given to keep concentrations <11.0 mmol/litre while avoiding hypoglycaemia.

Once the patient is haemodynamically stable, and usually within 24 hours, β-adrenoceptor blockers (e.g. bisoprolol) and angiotensin-converting enzyme (ACE) inhibitors (e.g. ramipril, perindopril) are started and rapidly uptitrated to the maximum tolerated dose.

Cardiac biomarkers, typically troponin T or I, should be measured on admission and again after 18–24 hours when concentrations typically peak; values are useful to allow an estimate of infarct size and provide an indicator of prognosis. Troponin concentrations can remain high for several days after STEMI; if reinfarction is suspected, creatine kinase MB concentrations can be helpful in confirming the diagnosis, as these usually return to normal much more quickly.

An echocardiogram should be performed before hospital discharge, ideally during the first 24 hours of admission, to assess LV function and to look for LV thrombus. In the early stages after STEMI the myocardium can be 'stunned' and LV dysfunction can partially recover over up to 6 weeks.

For individuals who develop heart failure with an LV ejection fraction (LVEF) <40% a mineralocorticoid antagonist is also recommended; eplerenone is preferred in this setting. All patients who develop heart failure irrespective of LVEF should be discharged taking an sodium–glucose co-transporter 2 antagonist (e.g. dapagliflozin).

Angiographically severe non-culprit stenoses identified in the major coronary arteries on initial angiography should be treated to achieve complete revascularization, ideally before discharge or, if not, within 6 weeks.[2] This is usually performed by PCI but if disease is very extensive coronary artery bypass grafting may be required.

Conservative management

Advanced age per se is not a contraindication to PPCI; however, frail elderly patients and those with severe co-morbidities may not be best served by aggressive interventional treatment. These individuals should be discussed with the heart attack centre and, where appropriate, managed conservatively, generally following the medical aspects of a non-STEMI protocol.

The UK National Institute for Health and Care Excellence (NICE) recommends ticagrelor as the second antiplatelet agent in this setting.[3] Longer term management should be tailored to individual circumstances.

Post-infarction complications

The incidence of major complications after STEMI has been much reduced since the introduction of reperfusion therapy. However, they still occur and tend to do so relatively early, usually in the first 24–48 hours.

Arrhythmias

Ventricular tachyarrhythmias often occur before admission, often provoking out-of-hospital cardiac arrest, but are less common after successful PPCI. An accelerated idioventricular rhythm (broad complexes, normal rate, no P waves) is a marker of successful reperfusion and resolves without needing intervention.

AF is common and is managed conservatively with an initial strategy of rate control and anticoagulation. AF that occurs during the first 24–48 hours usually resolves spontaneously; if not, a decision about elective cardioversion can be taken at a later stage.

Sinus bradycardia or arrest is common during the acute presentation because of vagal activation and usually responds to atropine. Atrioventricular nodal block of varying degrees is also common (typically with inferior infarcts) and usually resolves. Infra-nodal block (typically with anterior infarcts) usually indicates extensive infarction and carries a worse prognosis.

Temporary pacing may be required; however, it is best avoided unless the patient is haemodynamically compromised as there is an increased risk of bleeding complications, including ventricular perforation, in STEMI patients. In most cases high-degree heart block resolves with conservative treatment but permanent pacing should be considered if resolution does not occur within 5 days.

Cardiogenic shock and mechanical complications

Cardiogenic shock caused by pump failure after PPCI carries a high mortality and requires careful supportive management, usually in an intensive therapy unit.

Right ventricular infarction, usually presenting as hypotension after inferior STEMI, can respond to intravenous fluids alone. Otherwise, care involves careful fluid management, maintenance of sinus rhythm and the use of inotropic (e.g. dobutamine, adrenaline (epinephrine)) and pressor (e.g. noradrenaline (norepinephrine)) agents with invasive haemodynamic monitoring.

In this setting PCI of non-infarct related vessels and intra-aortic balloon pumping are associated with increased overall mortality and are not routinely used. Mechanical support devices such as Impella (Ambiomed, Danvers, Massachusetts, USA) can be considered in select cases.

Post-infarct ventricular septal defect, severe mitral regurgitation caused by papillary muscle rupture and ventricular free wall rupture carry a high mortality and require urgent liaison with the cardiothoracic surgical team.

Such patients usually show abrupt clinical deterioration, often with recurrent chest pain, tachycardia, hypotension, pulmonary oedema, elevated jugular venous pressure and/or the development of a new systolic murmur. These can usually be diagnosed clinically and confirmed by a bedside echocardiogram. In this setting an intra-aortic balloon pump can provide important haemodynamic support to facilitate transfer for surgical intervention.

If structural complications are identified at the point of admission, revascularization should not be undertaken.

Heart failure

Pulmonary oedema should be managed with intravenous furosemide, intravenous nitrates and/or non-invasive ventilation. After stabilization patients should be started on standard heart failure medications and followed up by the community heart failure team for education and post-discharge medical optimization.

LV thrombus

After STEMI the combination of regional LV hypokinesia, particularly if extensive and involving the LV apex, and endocardial injury can predispose to the development of LV thrombus. This should be suspected in patients presenting with remote vascular events but is usually detected on routine echocardiography. If LV thrombus is suspected, a contrast echocardiogram is the diagnostic investigation of choice.

Currently, only vitamin K antagonists (e.g. warfarin) are licensed for the treatment of LV thrombus but direct oral anticoagulants (e.g. apixaban, edoxaban) appear to be equally

effective. Anticoagulation should be continued for a minimum of 6 months and can be continued in the longer term based on echocardiographic resolution and the balance of haemorrhagic and thrombotic risk.

STEMI presenting as prolonged out-of-hospital cardiac arrest

It is not possible to determine the long-term outcome from a patient's initial neurological condition after successful resuscitation for out-of-hospital cardiac arrest.

After PPCI the patient is ventilated electively without active cooling but avoiding fever. If there has been a very long downtime, STEMI may be managed conservatively without PPCI as the bleeding risk is substantially increased. This is from a combination of factors including physical trauma from cardiopulmonary resuscitation, post-resuscitation coagulopathy and organ impairment, and the use of potent anti-thrombotic medication.

Neurological prognostication is complex and should be deferred until at least 72 hours to avoid premature withdrawal of life-sustaining treatment.

Discharge and post-discharge care

The development of PPCI services has greatly shortened inpatient stays after STEMI presentations. Low-risk patients, identified by having full revascularization, an uncomplicated inpatient course, age <70 years and LVEF >45%, may be discharged home 2–3 days after admission.

Cardiac rehabilitation

The benefits of attending cardiac rehabilitation after STEMI include improved well-being and exercise tolerance, better adherence to lifestyle changes (e.g. smoking cessation), reduced readmission and lower mortality compared with non-attenders.[4] Patients should be identified during their hospital stay and offered an individualized programme including structured exercise and education.

Driving

Driving regulations vary across the world. In the UK, Driver and Vehicle Licensing Agency (DVLA) guidance currently recommends that STEMI patients who have been fully revascularized and have an LVEF >40% can resume car driving after 7 days; otherwise driving should not restart for 4 weeks. Bus, coach and lorry drivers (DVLA Group 2 licensing) must have an LVEF >40% and be able to complete a normal exercise test to stage 3 of the Bruce protocol.

Outpatient review

An initial clinic review is usually undertaken 6–8 weeks after admission with a repeat echocardiogram if required. This allows a review of the person's condition and recovery, as well as providing an opportunity to make a more definitive plan about long-term medication.

In general, further management is as follow. DAPT should continue without interruption for 12 months after STEMI. Longer durations can be recommended where thrombotic risk is high, and shorter durations if the risk of bleeding is high. With modern drug-eluting stents DAPT can be interrupted after as few as 3 months if required (e.g. there is a need for urgent elective surgery). Long-term aspirin monotherapy is usually recommended, although increasing evidence suggests clopidogrel monotherapy may be superior.[5]

β-adrenoceptor blockers can be stopped after 12 months unless there is an indication to continue (e.g. heart failure, rate control in AF). ACE inhibitors or angiotensin receptor 2 blockers are usually continued long term, as is high-dose statin therapy.

NICE-recommended lipid targets include low-density lipoprotein cholesterol (LDL-C) <2.0 mmol/litre or non-high-density lipoprotein cholesterol <2.6 mmol/litre;[3] however, specialist guidelines are more aggressive and recommend LDL-C <1.4 mmol/litre.[2] If target concentrations cannot be achieved by statin therapy alone, options include the addition of ezetimibe, bempedoic acid, inclisiran or a PCSK9 inhibitor (e.g. evolocumab).

If appropriate, the review appointment also allows a check on smoking cessation, blood pressure management and control of diabetes. Patients should be considered for prophylactic implantable cardioverter defibrillator implantation if the LVEF is <35% at least 6 weeks after infarction despite full revascularization and optimal medical treatment. ◆

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TEST YOURSELF

To test your knowledge based on the article you have just read, please complete the questions below. The answers can be found at the end of the issue or online here.

Question 1

A 28-year-old woman presented with acute chest pain. She was a non-smoker and had no history of hypertension, diabetes or hyperlipidaemia. She was 32 weeks pregnant. Her grandfather had died of a myocardial infarct aged 78 years.

Investigation

- ECG showed sinus rhythm and 5 mm ST segment elevation in the anterolateral leads

What is the most likely coronary arterial pathology underlying this presentation?

A. Atherosclerotic plaque rupture
B. Spontaneous dissection
C. Thromboembolism
D. Vasculitis
E. Vasospasm

Question 2

A 78-year-old man presented with severe agitation. He had motor neurone disease and dementia and was bed-bound. His ECG showed 3 mm ST segment elevation in the inferior leads. Conservative management was recommended.

What is the most appropriate approach to antiplatelet therapy?

A. Long-term aspirin monotherapy
B. Long-term aspirin and 12 months of clopidogrel
C. Long-term aspirin and 12 months of prasugrel
D. Long-term aspirin and 12 months of ticagrelor
E. No antiplatelet therapy

Question 3

A 55-year-old woman was reviewed on the coronary care unit immediately after a primary percutaneous coronary intervention (PPCI) procedure for an ST-elevation myocardial infarction as the nursing staff were concerned about a change in her ECG. She had mild chest pain.

On clinical examination, she was well perfused, blood pressure was 115/75 mmHg, heart sounds were normal and the chest was clear. Her ECG showed a regular broad complex rhythm at 90 beats/minute with no visible P waves.

What is the most appropriate intervention?

A. DC cardioversion
B. Intravenous amiodarone
C. Oral bisoprolol
D. Reassurance only
E. Return to the catheter laboratory for angiography with or without rescue percutaneous coronary intervention