BMJ Best Practice

ST-elevation myocardial infarction

Straight to the point of care

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Summary

ST-elevation myocardial infarction (STEMI) presents with central chest pain that is classically heavy in nature, like a sensation of pressure or squeezing. Examination is variable, and findings range from normal to a critically unwell patient in cardiogenic shock.

Make a clinical working diagnosis of STEMI and start immediate treatment when a patient presents with symptoms suggestive of myocardial ischaemia and has persistent ST-segment elevation in at least 2 anatomically contiguous ECG leads.

Give a loading dose of aspirin as soon as possible to any patient with suspected acute coronary syndrome.

A rise in cardiac-specific troponins confirms the diagnosis but do not wait for laboratory results before starting treatment.

Immediate and prompt reperfusion can prevent or minimise myocardial damage and improve the chances of survival and recovery. Primary percutaneous coronary intervention (PCI) is the best management option for most patients, with fibrinolysis reserved for those without access to timely primary PCI.

Survivors of acute MI should receive cardiac rehabilitation and be closely followed up to ensure adequate modification of risk factors and optimisation of (and adherence to) pharmacotherapy for secondary prevention, and to monitor for the development of post MI complications and/or residual angina symptoms.

Definition

Acute myocardial infarction is myocardial cell death that occurs because of a prolonged mismatch between perfusion and demand. In the case of ST-elevation myocardial infarction (STEMI) this is caused predominantly by complete atherothrombotic occlusion of a coronary artery.

In the appropriate clinical context, a STEMI is diagnosed clinically when there is **new (or increased)** and **persistent** ST-segment elevation in **at least two contiguous leads of ≥1 mm in all leads other than leads V2-V3 where the following cut-off points apply**:[1]

- ≥2.5 mm in men <40 years old
- ≥2 mm in men >40 years old
- ≥1.5 mm in women regardless of age

    - 1 mm = 1 small square (at a standard ECG calibration of 10 mm/mV).

Contiguous ECG leads lie next to each other anatomically and indicate a specific myocardial territory.

Epidemiology

Cardiovascular disease (CVD) is the number one cause of death worldwide, accounting for 17.9 million deaths per year.[5] Ischaemic heart disease (IHD) is the most common cause of cardiovascular death; data from the European Society of Cardiology in 2019 showed that IHD accounted for 38% of CVD deaths in females and 44% in males.[2] [6]

The incidence and mortality of ischaemic heart disease has fallen over the last 30 years in Europe and is also decreasing in many developed countries, which may be due to better control of risk factors such as hypertension, diabetes, high cholesterol, and smoking.[7] However, mortality is increasing in developing and transitional countries, with more than 75% of CVD deaths occurring in developing countries.[5] These trends reflect changes in population longevity, urbanisation, and lifestyle changes.[5] Despite the overall reduction in incidence and mortality of CVD, in the UK the prevalence remains at about 3%, with CVD accounting for about 1.2 million hospitalisations per year.[8]

The incidence of STEMI has been steadily declining over the past 20 years. In England, Wales, and Northern Ireland, there were around 86,000 cases of MI between April 2021 and March 2022. There was a 16% increase in total MI cases compared with 2020-21 when admissions were substantially affected by COVID-19.[9]

MI affects both men and women, but tends to occur at a younger age in men.[10] The average age of a person having a first MI is 65.6 years for men and 72 years for women.[10] The incidence in women increases after the menopause. Women aged under 60 years with STEMI have higher 30-day mortality rates from STEMI than men under 60 years, even after adjusting for medications, primary percutaneous coronary intervention, and other co-existing comorbidities.[10][11]

About 90% of patients with coronary heart disease report at least one major risk factor for coronary artery disease, including cigarette smoking, dyslipidaemia, hypertension, diabetes, and abdominal obesity.[12]

Risk factors

Strong

smoking

Smoking causes 1 in 4 deaths from cardiovascular disease in the US and is the single most important modifiable risk factor for cardiovascular disease.[14] Cigarette smokers are substantially more likely than non-smokers to develop coronary artery disease (CAD), to have a stroke, and to develop peripheral vascular disease, and are at increased risk of fatal and non-fatal recurrences of these diseases.[7] [14] [15] Smoking increases risk for CAD by direct promotion of atherosclerosis, reduced oxygen delivery in the blood, increased thrombogenesis, and direct coronary artery spasm.[16] Even mild and passive smoking, and exposure to environmental tobacco, is associated with increased risk; risk increases further as the number of cigarettes smoked per day increases.[7] [15] [17] [18] [19] Current use of smokeless tobacco also increases the risk of CAD compared with people who have never used.[15] [18] [19]

Patients who stop smoking reduce their risk of recurrent cardiovascular disease by about one third compared with patients who do not stop smoking.[20] Surprisingly, current smoking is associated with

a lower risk of acute death in the setting of acute coronary syndrome.[21] This is referred to as the 'smoker's paradox' and reflects the tendency for smokers to develop thrombi on less severe plaques and at an earlier age than non-smokers.

hypertension

A major risk factor for acute coronary syndrome (ACS), and for poor outcomes in patients with ACS. About 69% of people who have a first myocardial infarction have BP >140/90 mmHg.[2] Hypertension is one of the most prevalent risk factors for coronary artery disease in the US; approximately 30% of Americans have BP >140/90 mmHg, placing them at greater risk of myocardial infarction, and of poor outcomes in the event of ACS.[2] [21] [22] [23] Even pre-hypertension (untreated systolic BP 120-139 mmHg and untreated diastolic BP 80-89 mmHg, or both) increases risk twofold compared with normal levels.[15] High blood pressure induces ventricular hypertrophy and endothelial dysfunction/damage, and promotes atherosclerosis, all of which predispose patients to cardiac events. By increasing cardiac after-load and myocardial oxygen consumption, uncontrolled hypertension can contribute to and worsen anginal symptoms.

Effective treatment of hypertension dramatically reduces the risk of cerebrovascular events, heart failure, and future myocardial infarction.[2]

diabetes

Patients with diabetes mellitus are at increased risk of coronary artery disease (CAD).[21] They have a two- to fourfold increased risk of cardiovascular disease compared with people who do not have diabetes.[24] The mechanisms are not fully known but they may reflect vascular abnormalities of inflammation, endothelial and smooth muscle function, obesity, hypertension, dyslipidaemia, and hypercoagulability.

CAD accounts for 75% of all deaths in the diabetic population.[21] Diabetes is associated with more extensive CAD, unstable lesions, and less favourable long-term outcomes (death, myocardial infarction, acute coronary syndrome re-admission), with approximately double the risk of long-term mortality from CAD than that of people without diabetes following myocardial infarction.[21] [22]

An HbA1c of <53 mmol/mol (<7%) is the goal of treatment for patients with diabetes.[15] [25] However, for patients with coronary heart disease, this goal may be less stringent (i.e., <64 mmol/mol [<8%]).[25]

obesity and metabolic syndrome

Estimates suggest that more than half of adults in Western society are overweight or obese.[15] [26] [27] Adipokines and other hormones secreted by adipose tissue are highly linked to inflammation and atherosclerosis.[28] Obesity is associated with diastolic dysfunction and is a strong stimulus for left ventricular hypertrophy.[29] [30] Obesity and the metabolic phenotype (abdominal obesity with known history of hyperlipidaemia, hypertension, and insulin resistance) predispose to coronary artery disease, and increase cardiovascular and all-cause mortality.[15] [21] [27] [29] [31] Bariatric surgery for weight loss reduces risk of major cardiovascular events (fatal acute coronary syndrome and stroke), incident heart failure, and cardiovascular mortality.[32]

sedentary behaviour and physical inactivity

Sedentary behaviour is associated with an increased risk of cardiovascular disease.[15] Epidemiological studies suggest a cause-and-effect relationship between physical activity and

cardiorespiratory fitness and reduced cardiovascular mortality.[33] The relative risk of coronary artery disease (CAD) associated with physical inactivity ranges from 1.5 to 2.4, an increase comparable to that for high cholesterol, high blood pressure, and cigarette smoking.[34]

Physical activity has anti-atherosclerotic, psychological, antithrombotic, anti-ischaemic, and anti-arrhythmic effects that are important in primary and secondary prevention of CAD.[33] Regular exercise increases cardiorespiratory fitness and lowers myocardial oxygen demand.[35] Sustained, regular physical activity lowers blood pressure, reduces lipid levels, reduces adiposity, increases insulin sensitivity, and decreases inflammation, stress, and adrenergic activity.[36] In patients with CAD, there is a direct correlation between the volume of moderate to vigorous physical activity and reduction in cardiovascular risk and mortality.[37] [38]

dyslipidaemia

Elevated low-density lipoprotein (LDL)-cholesterol, elevated triglycerides, decreased high-density lipoprotein (HDL), and elevated ratio of LDL to HDL are all independently associated with increased risk of atherosclerosis.[39]

There is a linear relationship between reduction in LDL-cholesterol and risk of myocardial infarction or other major vascular events; absolute risk reduction of major vascular events depends on the baseline risk of cardiovascular events and degree of LDL-cholesterol lowering.[40] In postmenopausal women, dysfunctional HDL may mean that high HDL levels (usually considered protective) are also associated with an increased risk of atherosclerosis.[41]

Lipid-lowering therapy reduces future ischaemic events and limits disease progression.[2] [21] [42] [43] Current guidelines recommend high-dose statin therapy in patients with known coronary artery disease (CAD) or CAD equivalent, irrespective of LDL levels.[2] [44] [45] Other lipid-lowering treatments can be considered in patients who are contraindicated or intolerant of statins.

chronic kidney disease

Approximately 30% to 40% of patients with acute coronary syndrome have chronic kidney disease (CKD).[2] [46] Excess cardiovascular disease in patients with CKD is caused, at least in part, by higher prevalence of traditional risk factors in this group; there is a very high prevalence of comorbid cardiovascular diseases in patients with CKD, ranging from ischaemic heart disease to arrhythmias and venous thromboembolism.[15] Decreasing glomerular filtration rate is associated with increasing risk of cardiovascular events, including death.[21] [47]

atherosclerosis (history of angina, myocardial infarction, stroke, transient ischaemic attack, peripheral vascular disease)

Atherosclerotic heart disease is the underlying mechanism in coronary artery disease (CAD). It evolves over decades and can begin in childhood. One study found intimal lesions in the aorta in all those aged 15-19 years, and in the right coronary artery in more than half of those of this age.[48] Atherosclerosis is typically silent until an acute event occurs (e.g., acute coronary syndrome [ACS]). A sedentary lifestyle, excess caloric intake, and cigarette smoking are strongly associated with atherosclerosis.

In an acute setting, the presence or absence of the traditional risk factors for CAD are not specific or sensitive for diagnosing ACS. However, they do appear to be important in determining prognosis in ACS and targeting secondary prevention strategies.[21]

Long-standing angina pectoris is a risk factor for coronary events.[49] Presence of peripheral arterial disease increases the likelihood of associated coronary atherosclerosis.[21]

family history of premature coronary artery disease

Defined as premature coronary artery disease in family members (men aged <50 years; women aged <55 years).[50] Family history includes a first-degree relative with a history of myocardial infarction, sudden cardiac death, aortic dissection, percutaneous coronary intervention, or coronary artery bypass graft. Inherited (primary) disorders of lipoprotein metabolism are an important cause, though other genetic variants may also play a role.[51] On physical examination, patients may have eruptive xanthomas, lipaemia retinalis (lipid accumulation within retinal vessels), or tendinous xanthomas. In the acute setting of acute coronary syndrome (ACS), presence or absence of family history does not help in treatment, but presence of family history increases the probability of ACS, and is associated with an increased risk of 30-day cardiac events in patients with ACS.[21]

age >60 years

Acute coronary syndrome (ACS) is more common in older patients; the majority of patients presenting with ACS are age >65 years (median age 68 years).[21] Patients with non-STEMI (NSTEMI) are often older than patients with STEMI; half of patients with NSTEMI are aged 70 years or older, whereas half of those with STEMI are aged 64 years or younger.[9] The mean age of patients presenting with STEMI is 60 years for men, and 71 years for women.[52]

cocaine use

Cocaine accounts for up to 25% of acute myocardial infarction in people aged 18-45 years.[53] In the hour after cocaine is used, the risk of myocardial infarction is 24 times the baseline risk.[54] This is probably due to cocaine-induced coronary vasospasm and thrombosis, in addition to a direct effect on heart rate and arterial pressure. Cocaine also has direct myocardial toxic properties.[21]

depression

An independent predictor of future myocardial infarction in otherwise healthy people.[55] [56]

stent thrombosis or restenosis

Stent thrombosis or in-stent restenosis may cause STEMI, non-STEMI (NSTEMI), or unstable angina. Both stent thrombosis and restenosis have complex causes, triggers, pathophysiology, and risk factors. Of importance, premature cessation of antiplatelet agents in patients with stents (drug-eluting and bare-metal) may trigger an acute coronary syndrome.[2] [21]

sleep apnoea

Untreated moderate to severe obstructive sleep apnoea (OSA) has been associated with a 17% increase in relative risk of cardiovascular events compared with risk in patients without OSA.[57] Patients with pre-existing OSA are at increased risk of further cardiac events following acute coronary syndrome compared with patients who do not have OSA.[58] [59]

Weak

migraine

People with migraine are more likely to have acute coronary syndrome and have higher rates of cardiovascular mortality.[60] It is unclear whether this is an independent risk factor for cardiovascular disease, or due to higher prevalence of cardiovascular risk factors in patients with migraine.[60]

adverse pregnancy outcomes

Women who have had adverse outcomes of pregnancy (hypertensive disorders of pregnancy, gestational diabetes, preterm delivery, and intrauterine growth restriction) are at increased risk of future cardiovascular disease.[61] [62]

Aetiology

MI is usually a consequence of coronary artery disease. Atherosclerosis with plaque fissuring or rupture and thrombus formation is the underlying aetiology for STEMI in most patients. A small proportion of patients present with STEMI caused by coronary spasm reducing myocardial perfusion, coronary embolism, or following chest trauma or spontaneous coronary or aortic dissection.

Pathophysiology

Atherosclerotic plaques form gradually over years.[13] They begin with the accumulation of low-density lipoprotein cholesterol and saturated fat in the intima (the inner layer) of blood vessels. This is followed by the adhesion of macrophages to endothelium, then diapedesis and entry into the intima, where they accumulate lipids and become foam cells. Foam cells are a rich source of proinflammatory mediators. The lesion up to this point is referred to as a fatty streak, and may be reversible to a certain extent.

Subsequent evolution involves migration of smooth muscle cells from the media, and their proliferation and deposition of extracellular matrix, including proteoglycans, interstitial collagen, and elastin fibres.[13] Some of the smooth muscle cells in advanced plaques exhibit apoptosis. Plaques often develop areas of calcification as they evolve. The plaque initially evolves with the artery remodelling outwards, followed by encroachment on the arterial lumen. Eventually the stenosis can limit flow under conditions of increased demand, causing angina.

STEMI typically occurs after abrupt and catastrophic disruption of a cholesterol-laden plaque. This results in exposure of substances that promote platelet activation and aggregation, thrombin generation, and thrombus formation, causing interruption of blood flow. If the occlusion is severe and persistent, myocardial cell necrosis follows.

On interruption of blood flow in the coronary artery, the zone of myocardium supplied by that vessel immediately loses its ability to shorten and perform contractile work. Early hyperkinesis of the non-infarcted zones occurs, probably as a result of acute compensatory mechanisms including increased sympathetic activity and Frank-Starling mechanism. As necrotic myocytes slip past each other, the infarction zone thins and elongates, especially in anterior infarction, leading to infarction expansion.

If a sufficient quantity of myocardium undergoes ischaemic injury, left ventricular (LV) systolic function becomes depressed; cardiac output, stroke volume, blood pressure, and compliance are reduced; and end systolic volume increases. Clinical heart failure occurs if 25% of myocardium has abnormal contraction, and cardiogenic shock occurs on loss of >40% of LV myocardium. Decreased compliance and increased LV end diastolic pressure give rise to diastolic dysfunction.

Classification

Acute coronary syndrome

Acute coronary syndrome (ACS) encompasses a spectrum of conditions, including patients presenting with recent changes in clinical symptoms or signs, with or without changes on 12-lead electrocardiogram (ECG) and with or without acute elevations in cardiac troponin (cTn) concentrations.[2] Historically ACS has been divided into three clinical categories according to the presence or absence of ST-segment elevation on a presenting ECG and on elevations of cardiac troponin T or I.[3] [4]

1. ST-elevation myocardial infarction (STEMI): ECG shows persistent ST-segment elevation in at least two anatomically contiguous leads.

2. Non-STEMI (NSTEMI): ECG does not show ST-segment elevation, but cardiac biomarkers are elevated. The ECG may show ischaemic changes such as ST-segment depression, T-wave inversion, or biphasic T waves.

3. Unstable angina pectoris: non-specific ischaemic ECG changes, but cardiac biomarkers are within the normal range.

Fourth Universal Definition of Myocardial Infarction[1]

In the contemporary setting MI can also be classified according to variations in pathological, clinical, and prognostic factors, alongside the different management strategies recommended for each type.[1]

- Acute myocardial infarction (types 1, 2, and 3 MI)
    - Defined as acute myocardial injury with clinical evidence of acute myocardial ischaemia and with detection of a rise and/or fall of cardiac troponin values with at least one value >99th percentile of the upper reference limit (URL)
    - A type 1 MI is characterised by identification of a coronary thrombus by angiography (or autopsy)
        - Includes STEMI and NSTEMI
    - In type 2 MI there is evidence of an imbalance between myocardial oxygen supply and demand that is not associated with an acute atherothrombotic event
        - Includes vasospasm, coronary microvascular dysfunction, and non-atherosclerotic coronary dissection
    - A type 3 MI is cardiac death in a patient with symptoms of myocardial ischaemia and new ischaemic ECG changes prior to a cardiac troponin level becoming abnormal or available
- Coronary procedure-related MI (types 4 and 5 MI)
    - Type 4a MI – related to percutaneous coronary intervention
    - Type 4b MI – related to stent thrombosis
    - Type 4c MI – related to stent restenosis

- Type 5 MI – related to coronary artery bypass graft surgery

Case history

Case history #1

A 54-year-old man with a medical history of hypertension, diabetes, dyslipidaemia, smoking, and family history of premature coronary artery disease presents with retrosternal crushing chest pain (10/10 in intensity), radiating down the left arm and left side of the neck. He feels nauseated and light-headed and is short of breath. Examination reveals hypotension, diaphoresis, and considerable discomfort with diffuse bilateral crackles on chest auscultation. ECG reveals convex ST-segment elevation in leads V1 to V6.

Case history #2

A 70-year-old woman is 2 days post-operative for knee replacement surgery. Her past medical history includes type 2 diabetes and a 40 pack-year history of smoking. She reports feeling suddenly unwell with dizziness, nausea, and vomiting. She denies any chest pain. On examination she is hypotensive and diaphoretic. ECG shows convex ST-segment elevation in leads II, III, and aVF with reciprocal ST segment depression and T-wave inversion in leads I and aVL.

Other presentations

Patients with STEMI may also be asymptomatic or present with atypical chest pain or epigastric pain.

Recommendations

Urgent

Assessment and diagnosis of STEMI is a **time-critical process**. The shortest possible delay from symptom onset to coronary reperfusion maximises the patient's chances of survival and recovery.[1] [2] [70][71] [72] [73] [74] [75]

Obtain an **ECG immediately** and certainly **no more than 10 minutes** from the point of first medical contact.[2] [76] [77]

Establish the patient's **haemodynamic status** and specifically look for any signs of **cardiogenic shock**.

- Cardiogenic shock complicates 5% to 10% of STEMI admissions. Look for persistent hypotension (**systolic blood pressure <90 mmHg**) and/or any signs of **end-organ hypoperfusion** or fulminant heart failure.[78] [79] [80]

Make a **clinical working diagnosis of STEMI** based on a combination of **acute chest pain** (or equivalent symptoms suggestive of myocardial ischaemia) together with **persistent ST-segment elevation** in at least two contiguous ECG leads.[1] [2]

As soon as a clinical diagnosis of STEMI is made, **make an immediate assessment of eligibility for coronary reperfusion therapy** (irrespective of age, ethnicity, sex, or level of consciousness) and seek input from the **interventional cardiology team**. Primary percutaneous coronary intervention is the preferred reperfusion strategy for any eligible patient who presents within 12 hours of symptom onset provided it can be delivered within 120 minutes of the time when fibrinolysis could have been given.[2] [75] [81]

Give all patients with suspected acute coronary syndrome **a single loading dose of aspirin as soon as possible**, unless they have aspirin hypersensitivity.[75]

- Check your local protocol or discuss the patient with a senior colleague if they have hypersensitivity to aspirin.

Key Recommendations

Presentation

Patients typically present with **central chest pain** that is **heavy** in nature, like a sensation of pressure or squeezing. It may **radiate** to the left arm, neck, or jaw and can be associated with **nausea, vomiting, dyspnoea, lightheadedness, palpitations, or syncope (chest pain-equivalent symptoms)**.[1] [2] [82]

- Beware of presentations where chest pain is not the predominant feature **(chest-pain equivalent symptoms)**, particularly in older patients, women, and patients with diabetes.[2] [82]

Focus your history on:[2] [71] [74]

- Characteristics of symptoms of myocardial ischaemia (including **time since symptom onset**, which will inform the most appropriate reperfusion strategy)
- Previous cardiac history

- Evidence of cardiac risk factors.

Examination is variable, and findings **range from normal to a critically unwell** patient in cardiogenic shock. Your priorities from examination and history-taking are to:[2] [71] [75] [82]

- Confirm a STEMI diagnosis
- Rule out alternative diagnoses/causes of ST-segment elevation
- Establish the patient's haemodynamic status
- Look for complications of acute MI.

Always consider **right ventricular involvement** when there is a **triad of hypotension**, **elevated jugular venous pressure**, and **clear lung fields**.[83]

Clinical diagnosis

In a patient who has chest pain or other ischaemic symptoms, make a clinical diagnosis of STEMI when there is **new (or increased)** and **persistent ST-segment elevation** in at least two contiguous leads of ≥1 mm in all leads other than leads V2-V3 where the following cut-off points apply:[1]

- ≥2.5 mm in men <40 years old
- ≥2 mm in men >40 years old
- ≥1.5 mm in women regardless of age

    - 1 mm = 1 small square (at a standard ECG calibration of 10 mm/mV)
    - Contiguous ECG leads lie next to each other anatomically and indicate a specific myocardial territory.

Think **posterior STEMI** when there is deep ST-segment depression in leads V1-V3.[1] [2]

Consider **complete left main coronary artery obstruction** if the following are both present, especially if the patient has haemodynamic compromise:[2] [84]

- ST depression ≥1 mm in ≥6 surface leads (i.e., inferolateral ST depression)
- ST elevation in aVR or lead V1.

ECG diagnosis of STEMI is trickier in the presence of **left bundle branch block** (LBBB).

- One of the best indicators is **concordant ST-segment elevation** (i.e., in leads with positive QRS deflections).[85] The **Sgarbossa criteria** can also be helpful.[1] [2] [86]
- **New LBBB** does not on its own indicate a STEMI.[87] [88]
- Manage any patient with bundle branch block and clinical suspicion of ongoing myocardial ischaemia as per the standard STEMI protocol. This applies to both left and right bundle branch block, whether new or previously known.[2]

Cardiac biomarkers

Although STEMI can usually be diagnosed by ECG alone, a **rise in cardiac-specific troponins** definitively confirms the diagnosis.[1][2] [82] [89] [90] [91] [92]

- However, **do not delay coronary reperfusion** to wait for cardiac biomarker laboratory results (or any other blood results).

- Start treatment and assess eligibility for coronary reperfusion immediately a clinical diagnosis has been made based on ECG changes in a patient with symptoms/signs of myocardial ischaemia.

Full Recommendations

Diagnostic criteria

Practical diagnosis of STEMI

Assessment, diagnosis, and management of acute STEMI is a **time-critical** process.[1] [2][71] [74] [75]

- Always remember the guiding principle that 'time is muscle' – the **shortest possible delay from symptom onset to coronary reperfusion is vital** to protect the myocardium from ischaemic damage and maximise the patient's chances of survival.[72] [73]
- For a patient diagnosed with STEMI, nearly half of potentially salvageable myocardium is lost within 1 hour of the coronary artery occlusion and two-thirds is lost within 3 hours.[93]

Make a working clinical diagnosis of STEMI and start immediate treatment if the patient meets **BOTH** of the following criteria:[2]

1. **New ST-segment elevation** at the J-point in **at least two contiguous leads** on a 12-lead ECG **of ≥1 mm in all leads** (in the absence of left ventricular hypertrophy or left bundle branch block) **other than leads V2-V3** where the following cut-off points apply:[1] [2]
    - ≥2.5 mm in men <40 years old
    - ≥2 mm in men >40 years old.
2. **Persistent typical central chest pain** or other symptoms consistent with myocardial ischaemia (**chest pain-equivalent symptoms**) within the last **12 hours.**

As soon as a clinical diagnosis is made, **immediately assess eligibility for coronary reperfusion therapy.** [2] [75]

- **Do not wait for** a definitive diagnosis from cardiac **troponin** levels as coronary reperfusion is a time-critical intervention.[1] [2]
- **Primary percutaneous coronary intervention** (PCI) is the preferred reperfusion strategy for any eligible patient who presents **within 12 hours of symptom onset** provided it **can be delivered within 120 minutes of the time when fibrinolysis could have been given**.[75] [81] Start medical treatment and refer immediately to the interventional cardiology team.[2] [72] [73] [75]
- **Give fibrinolysis** (unless contraindicated) if primary PCI **cannot** be delivered **within 120 minutes** of the time when fibrinolysis could be started.[75] [81]
    - Fibrinolysis can be administered as part of the **pre-hospital** management of STEMI.[2] [72]

Practical tip

The European Society of Cardiology recommends to 'Think A.C.S.' at initial assessment of chest pain:[2]
- **A**bnormalities or evidence of ischaemia on ECG assessment
- **C**linical context: take a targeted clinical history to assess the clinical context of the presentation
- **S**tability: targeted clinical examination to assess for clinical and haemodynamic stability.

The admitting team can decide whether immediate invasive management is required based on this initial assessment.

Formal diagnostic criteria for STEMI

A definitive diagnosis of STEMI requires evidence of a **rise in cardiac troponin levels – but do not wait** for this to be confirmed before starting treatment.[1]

- Evidence of **myocardial injury** (via acutely elevated cardiac troponin levels) is required for a **definitive confirmation** of the diagnosis of STEMI.[1] [2] [82]
- However, **coronary reperfusion** is a **time-critical intervention** that must be started as soon as a clinical diagnosis is made.[2] [75]

STEMI is classified as a type 1 MI under the Fourth Universal Definition of Myocardial Infarction.[1]

- Type 1 MIs are caused by ischaemia due to rupture or erosion of an atherosclerotic plaque leading to partial, or in the case of STEMI total, intraluminal occlusion of the coronary artery.[1]

Under the Fourth Universal Definition, an acute type 1 MI is definitively diagnosed based on a rise and/or fall of cardiac troponin levels, with at least one value above the 99th percentile of the upper reference limit in a patient who also has at least one of the following:[1] [82]

- Symptoms of acute myocardial ischaemia
- ECG changes indicative of new ischaemia
- Development of pathological Q waves in the ECG
- A new regional left ventricular wall motion abnormality consistent with a coronary artery territory (e.g., on transthoracic echocardiography)
- New loss of viable myocardium (e.g., on cardiac magnetic resonance imaging)
- Presence of intracoronary thrombus found on coronary angiography.

The presenting ECG is central to determining whether a type 1 MI is a STEMI or a non-ST-elevation MI (NSTEMI).[1]

Clinical presentation

Patients typically present with **central chest pain:** [1] [2] [82]

- Classically **retrosternal, crushing, heavy, severe** and **diffuse** in nature
- Might be described by the patient as **'pressing or squeezing'**
- May occur **at rest or on activity**
- May be constant or intermittent, or wax and wane in intensity
- Sometimes **radiating** to the left arm, neck, or jaw.

The chest pain may be **associated with** nausea, vomiting, dyspnoea, diaphoresis, lightheadedness, palpitations, or syncope.[2] [82]

- **Dyspnoea** is a common feature secondary to **pulmonary congestion** from left ventricular systolic dysfunction. It can also occur due to other **mechanical and electrical complications**

of acute MI, which occur less commonly in the context of contemporary rapid revascularisation, for example:

- Left ventricular aneurysm
- Ventricular septal rupture
- Left ventricular free wall rupture
- Acute mitral regurgitation – papillary muscle rupture/functional (ischaemic) mitral regurgitation
- Pericardial effusion
- Cardiac tamponade
- Supraventricular tachyarrhythmias
- Ventricular tachyarrhythmias
- Bradycardia and atrioventricular block.

- **Nausea and vomiting** are common features.
  - These are non-specific symptoms but are commonly associated with inferior-wall STEMI due to increased vagal tone.
  - May be the only indicator of inferior-wall STEMI.

- Patients sometimes report anxiety and/or an impending **sense of doom**.
- Some patients present with **palpitations.** [2] [94]
  - Tachycardia
    - Supraventricular tachyarrhythmias such as atrial fibrillation
    - Ventricular tachyarrhythmias such as ventricular tachycardia
  - Bradycardia
    - Sinus bradycardia
    - Atrioventricular block secondary to inferior STEMI
    - Atrioventricular block secondary to anterior STEMI
  - Irregular heart beat
    - Supraventricular tachyarrhythmias such as atrial fibrillation
    - Ventricular extrasystoles

Be aware of patients who present **without** chest pain as the predominant feature (i.e., with **chest pain-equivalent symptoms**).[2] [82]

- Women, older patients, and patients with diabetes are more likely to present with such features.[2] [95]
- Patients might describe their chest symptoms as **burning, throbbing, tight** or a feeling like **trapped wind**.
  - The patient may describe **indigestion** rather than chest pain.

- In the absence of chest pain, there may be **epigastric pain, back (interscapular) pain, neck or jaw pain, or arm pain** (typically left-sided).
- Patients may present with **breathlessness, sweating, palpitations, dizziness, nausea, or vomiting but no chest pain**.
- Clinical suspicion is key to making the diagnosis. It is, therefore, vital to make a **full assessment** based on the history, examination, and serial ECGs.[1][92]

Practical tip

Do not rely on a positive patient response to glyceryl trinitrate as a reliable diagnostic indicator of ischaemic chest pain.[2] [82] [96]

- Response to nitrates can be misleading. Patients who get symptom relief still need confirmatory ECG testing to inform the diagnosis.
- Complete normalisation of ST-segment elevation along with resolution of chest pain after buccal or sublingual nitrates suggests coronary vasospasm (with or without associated MI).[2]

Patients with STEMI **may be asymptomatic** – this is known as a **silent STEMI.** [1]

- STEMI is rarely truly asymptomatic but some patients have only **mild, non-specific symptoms** that can lead to a delay in presenting or to the STEMI going undiagnosed.
- In practice, a patient who has a 'silent' STEMI may present to their primary care doctor a few days after the episode of non-specific symptoms, at which point evidence of ST-segment elevation might still be present on the ECG. Seek advice from the cardiology team for such patients.

History

Your history should cover the following.[82][97]

- **Characteristics of symptoms**; in particular, chest pain:[82]
  - "Have you ever had this type of pain before?"
    - Nature, severity, and duration of pain
    - Radiation
    - Associated symptoms
  - "Do you normally have chest pain when you exert yourself? If so, is it similar in quality to the chest pain experienced when you don't?"
- **Time since symptom onset** – this is crucial to inform the appropriate reperfusion strategy.[2] [74] [75]
  - If symptoms are intermittent, it is important to ask when the last episode of pain occurred.[82]
- Any **history of cardiovascular disease**; in particular, **ischaemic heart disease**.[82]
  - Also check for any previous episodes of investigation or treatment for chest pain.
- **Cardiovascular risk factor profile:** [82]
  - Smoking status
  - Hypertension

- Diabetes mellitus
- Hypercholesterolaemia
- Family history of premature coronary artery disease (<60 years)
- Established coronary artery disease
- Advanced age
- Obesity
- Metabolic syndrome
- Physical inactivity
- Chronic kidney disease
- Cocaine use
- Existing peripheral vascular disease or cerebrovascular disease.

- **Medication history:**
  - Will help to consolidate the risk factor profile assessment, especially if the history given by the patient is limited or vague
  - Record any use of chronic **oral anticoagulation** – this will influence what type of arterial access can be used if the patient proceeds to coronary angiography or primary percutaneous coronary intervention (PCI).

Practical tip

The choice of coronary reperfusion strategy depends on **time since symptom onset** – but obtaining an exact time for this can be difficult.

- Patients can often give only an approximate idea of when their symptoms began.
- Patients sometimes ignore chest pain (or associated symptoms) until they can no longer tolerate it.
- The reliability of the assessment of time since symptom onset is determined by a combination of the patient's ability to give an accurate history and the experience and skill of the clinician taking the history.
- If you question the patient carefully, they may describe warning signs, or less severe or less long-lasting symptom episodes preceding the more severe episode that has prompted them to seek medical help.

The **total ischaemic time** encompasses time from symptom onset until coronary reperfusion with either primary PCI or fibrinolysis.

- If the patient contacts emergency medical services in the community, then the total ischaemic time = Patient Delay + Emergency Medical Services Delay + System Delay.
- If the patient presents directly to a hospital (PCI-capable or non-PCI-capable), then the total ischaemic time = Patient Delay + System Delay.

Physical examination

The clinical picture of acute MI varies from asymptomatic through to fulminant acute heart failure and cardiogenic shock.

- Your priorities are to **establish the following** as quickly as possible:[82]

- Confirm the STEMI **diagnosis**
- **Rule out** alternative diagnoses/causes of ST-segment elevation (see *Important differentials to consider* below)
- Establish the patient's **haemodynamic status** – seek immediate senior support and specialist input if there are any signs of **cardiogenic** shock (see *Cardiogenic shock* below)
- Look for **complications** of acute MI (see *Acute MI complications* sections below).

- Your **examination** should check:[82]
  - Blood pressure, heart rate, and heart rhythm
  - Consciousness level (e.g., Glasgow Coma Scale, AVPU [alert, verbal, pain, unresponsive] scale)
    - However, do not use the level of consciousness after cardiac arrest caused by suspected acute STEMI to determine whether the patient is eligible for coronary angiography ± primary percutaneous coronary intervention (PCI)[75]
  - Airway patency
  - Oxygen saturations
  - Pulse: radio-radial and radio-femoral delay
  - Jugular venous pressure (JVP) – a raised JVP could indicate:
    - Congestive cardiac failure
    - Right ventricular involvement after an inferior or extensive anterior STEMI
    - Underlying (chronic) lung disease
    - Large pericardial effusion or cardiac tamponade
    - Pulmonary embolism (which can also give rise to ST-segment elevation).

Look for pallor, cool/clammy to touch skin, and any signs of **peripheral shutdown.**

- These are common presenting features due to high sympathetic output resulting in peripheral vasoconstriction.

Auscultate the heart and lungs.

- **Muffled heart sounds** could suggest a pericardial effusion or even cardiac tamponade.
- Is there a **third (S3) or fourth (S4) heart sound**?
  - These added heart sounds could suggest severe heart failure.
- A **murmur** might suggest:
  - Acute ventricular septal defect
  - Acute mitral regurgitation
  - Underlying chronic valvular heart disease.
- **Crackles/crepitations** or **cardiac wheeze** would suggest congestive cardiac failure ± pulmonary oedema.
- Is the patient coughing up **pink frothy sputum**?

- This would suggest congestive cardiac failure ± pulmonary oedema.

Check for peripheral oedema and hepatomegaly.

Document the **Killip class**. This classifies degree of heart failure after acute MI and predicts 30-day mortality:[97]

- Killip class I = no signs of heart failure/pulmonary congestion
- Killip class II = S3 and basal crackles/crepitations
- Killip class III = acute pulmonary oedema
- Killip class IV = cardiogenic shock.

Evidence: Killip class and prognosis

Risk of death is strongly correlated to Killip class.

- The original paper on the impact of Killip class on prognosis **dates from 1967**, when it was reported that the associated **in-hospital mortality rates** were 6% for class I, 17% for class II, 38% for class III, and 81% for class IV.[98]
- With advances in treatment, particularly the introduction of coronary reperfusion therapy, **mortality rates have fallen by 30% to 50% in each Killip class**. In the GUSTO international trial of 41,021 patients, after adjustment for all other factors, the OR associated with Killip class III versus I for an average-age patient was 4.37 (95% CI, 3.34 to 5.71), whereas the OR for Killip class IV versus I was 7.86 (95% CI, 5.88 to 10.49).[97]

Cardiogenic shock

Cardiogenic shock complicates 5% to 10% of STEMI admissions.[79] [80][99][100]

- In-hospital mortality remains high (≥50%).[100]
- There is a bimodal presentation: the majority occur within 24 hours; the remainder occur within the first week.[100] [101]

Seek immediate senior support and specialist input if your clinical assessment suggests **cardiogenic shock.** [79]

See Shock .

Patients present with signs of hypoperfusion and/or fulminant heart failure, such as:

- Altered mental status/reduced consciousness
- Tachypnoea
- Severe dyspnoea
- Tachycardia
- Orthopnoea
- Cool peripheries
- Grey, ashen, pale appearance.

Cardiogenic shock is defined as **persistent hypotension** (systolic blood pressure [SBP] <90 mmHg) together with **signs of end-organ hypoperfusion.** [78] [79] [80]

- Clinical criteria:[78] [80]

- SBP <90 mmHg despite adequate volume replacement, or if inotropes and/or mechanical circulatory support are needed to maintain SBP ≥90 mmHg
- Urine output <30 mL/hour
- Cool extremities.

- Haemodynamic criteria:[78] [80]
  - Cardiac index ≤2.2 L/minute/m^2
  - Wedge pressure ≥15 mmHg.

- Cardiogenic shock results from extensive left ventricular infarction and/or mechanical complications such as:[80]
  - Papillary muscle rupture
  - Ventricular septal rupture
  - Left ventricular free wall rupture leading to pericardial tamponade
  - Right ventricular infarction.

Important differentials to consider

Always consider **alternative diagnoses** that might explain the presenting symptoms and/or ST elevation on ECG, including:[82]

- Aortic dissection (ST elevation can be present on the ECG)
- Pulmonary embolism (ST elevation or ST depression can be present on the ECG)
- Pericarditis (ST elevation can be present on the ECG)
- Myocarditis (ST elevation can be present on the ECG)
- Pneumothorax
- Pneumonia
- Intracranial pathology (e.g., subarachnoid haemorrhage)
- Gastro-oesophageal reflux disease
- Oesophageal spasm
- Costochondritis
- Anxiety or panic.

Be aware of **spontaneous coronary artery dissection (SCAD)**, especially in younger women presenting with ST-segment elevation and chest pain.

- SCAD is defined as an **epicardial coronary artery dissection** that is **not associated with atherosclerosis or trauma** and is not iatrogenic.[102] [103]
  - The left anterior descending artery is the most commonly affected artery.[102] [104] [105] [106] [107] [108] [109]
  - It can lead to coronary artery obstruction secondary to intramural haematoma or intimal disruption (in contrast to the atherosclerotic plaque rupture or intraluminal thrombus seen with STEMI).

- Patients **almost always** present with an acute coronary syndrome causing **chest pain and elevation in cardiac enzymes**.[102]

- A minority can present in **cardiogenic shock** (2% to 5%).[104] [105]

- Consider the possibility of SCAD in **any young patient (especially female)** who presents with an **acute MI** and who has **no history of or risk factors** for cardiovascular disease.[102]
  - SCAD occurs **predominantly in women** (average age 45-53 years) and may account for up to 35% of MI presentations seen in women ≤50 years old and up to 43% of pregnancy-associated MIs.[102] [103] [105] [109] [110][111]
  - Patients tend to have **few or no** conventional cardiac risk factors.[102]

- SCAD is an **important cause** of ST-segment deviation on the ECG, with different patient series reporting that:[102] [104] [105] [106] [107] [108]
  - 26% to 87% of patients with SCAD present with STEMI
  - 13% to 69% present with non-STEMI (NSTEMI).

- If SCAD is suspected, **coronary angiography** should be performed as soon as possible to confirm the diagnosis.[102] [103]
- There is a paucity of evidence available to guide management decisions once SCAD is confirmed on angiography. The most appropriate approach depends on individual patient characteristics.
  - **Conservative management** with **close ongoing monitoring** is preferred for most patients who are **clinically stable**, as observational data suggest the SCAD lesion will usually heal.[102] [103]
  - Urgent intervention with **PCI or coronary artery bypass grafting** should be considered for **high-risk patients** (e.g., ongoing ischaemia, haemodynamic instability, left main artery dissection).[112]

Practical tip

Takotsubo cardiomyopathy syndrome can **mimic MI** and has **similar mortality** to STEMI/NSTEMI.[1]

- It is triggered by a wide spectrum of **physical and emotional triggers** and is also referred to in the literature as broken heart syndrome, apical ballooning cardiomyopathy, or stress cardiomyopathy.[113] [114] [115]
    - 'Takotsubo' comes from the Japanese word for octopus trap.
- Takotsubo cardiomyopathy is characterised by a **temporary left ventricular wall motion abnormality** associated with **signs and symptoms of acute coronary syndrome.** [114] [115] For example:
    - Chest pain
    - ST-segment deviation on ECG
    - Raised cardiac biomarkers such as troponin (although the peak value is often modest).[1]
- In-hospital mortality is similar to STEMI and NSTEMI.[1]
- It is estimated to represent 1% to 3% of all patients and 5% to 6% of female patients who present with suspected STEMI.[114] Over 90% of affected patients are post-menopausal women.[1]

Patients **may** present with **ST-segment elevation** on their ECG.[114] [115]

- ST-segment elevation is present in over 40% of patients but the extent of the elevation is usually **widespread across the lateral and precordial leads**, beyond that of a single coronary artery distribution.[1]

Suspect takotsubo cardiomyopathy if the clinical manifestations and ECG abnormalities are **out of proportion** to the degree of elevation of cardiac biomarkers;[1] echocardiography findings may include hyperdynamic basal segments with apical ballooning.

- Manage the patient as for STEMI in the first instance if there are signs and symptoms consistent with myocardial ischaemia.
- Refer for **urgent coronary angiography** and **left ventriculography** to confirm or exclude takotsubo cardiomyopathy.
    - If there are coronary culprit lesions on angiography that correspond to the regional wall motion abnormalities, the patient is treated the same as for an acute coronary syndrome.
    - If there are no coronary culprit lesions that correspond to the regional wall motion abnormalities and acute infectious myocarditis can be ruled out, the patient is treated as a takotsubo cardiomyopathy.

Co-existing coronary artery disease **does not exclude** the diagnosis.

- It is present in 10% to 29% of patients with takotsubo cardiomyopathy.[114] [115] [116] [117] [118]

The distinction between MI and takotsubo cardiomyopathy syndrome can be made by the presence of **QTc prolongation >500 ms** during the acute phase and the **recovery of left ventricular function** over 2-4 weeks.[1]

Investigations

ECG interpretation for STEMI

Perform a 12-lead ECG **within 10 minutes** of first medical contact in any patient who presents with chest pain and/or other signs of possible STEMI.[1] [2]

- If the patient presents in the community, obtain a **pre-hospital ECG** and send it digitally to the receiving hospital as quickly as possible.[2] [82] [119]
- If the **ECG is equivocal** despite a high clinical suspicion of acute MI, perform **serial ECGs** in the appropriate hospital setting and compare these with historical ECGs, if available.[1] [2] [82]

In the appropriate clinical context (chest pain or other symptoms of ischaemia), make a **clinical working diagnosis of STEMI** if there is **new (or increased) and persistent ST-segment elevation** in two or more contiguous leads of ≥1 mm in all leads other than leads V2-V3 where the following cut points apply:[1]

- ≥2.5 mm in men <40 years old
- ≥2 mm in men >40 years old
- ≥1.5 mm in women regardless of age.
    - 1 mm = 1 small square (at a standard ECG calibration of 10 mm/mV).

Make the diagnosis when these criteria are met in the **absence of** left ventricular hypertrophy, left bundle branch block (LBBB), or a paced rhythm on the ECG.

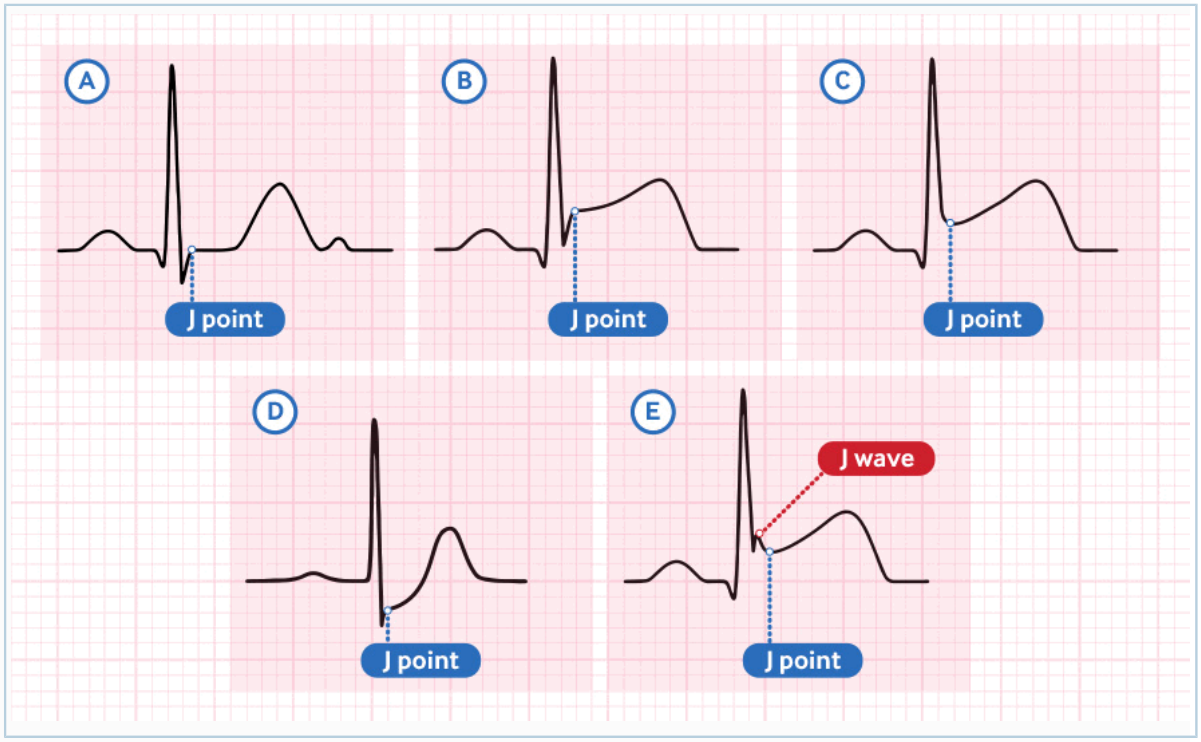
- Severe cases of left ventricular hypertrophy can appear identical to LBBB.
- A paced rhythm can appear identical to LBBB.
- Note that the **presence of left ventricular hypertrophy, LBBB, or a paced rhythm does not preclude a diagnosis of STEMI** if the patient presents with typical symptoms of myocardial ischaemia.
- See *Less common ECG presentations* below for more on diagnosis of STEMI in the presence of LBBB.

Consult a **cardiology specialist immediately** if the ECG changes are **equivocal.**

- An urgent transthoracic echocardiogram to look for regional wall motion abnormalities is indicated.[120]

Practical tip

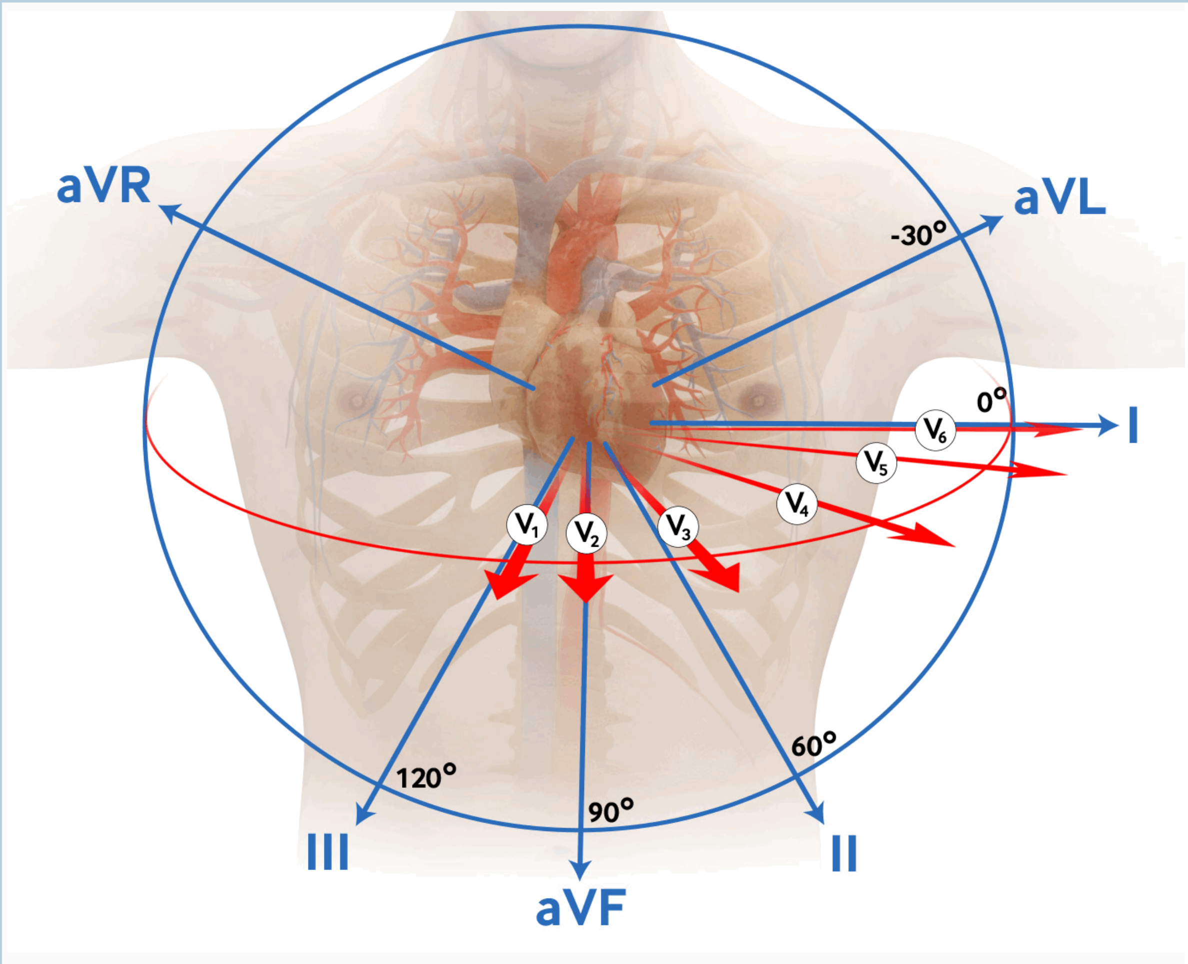
Measure ST-segment elevation from the J point.[1] [2]



Identifying the J point on the ECG
Created by the BMJ Knowledge Centre

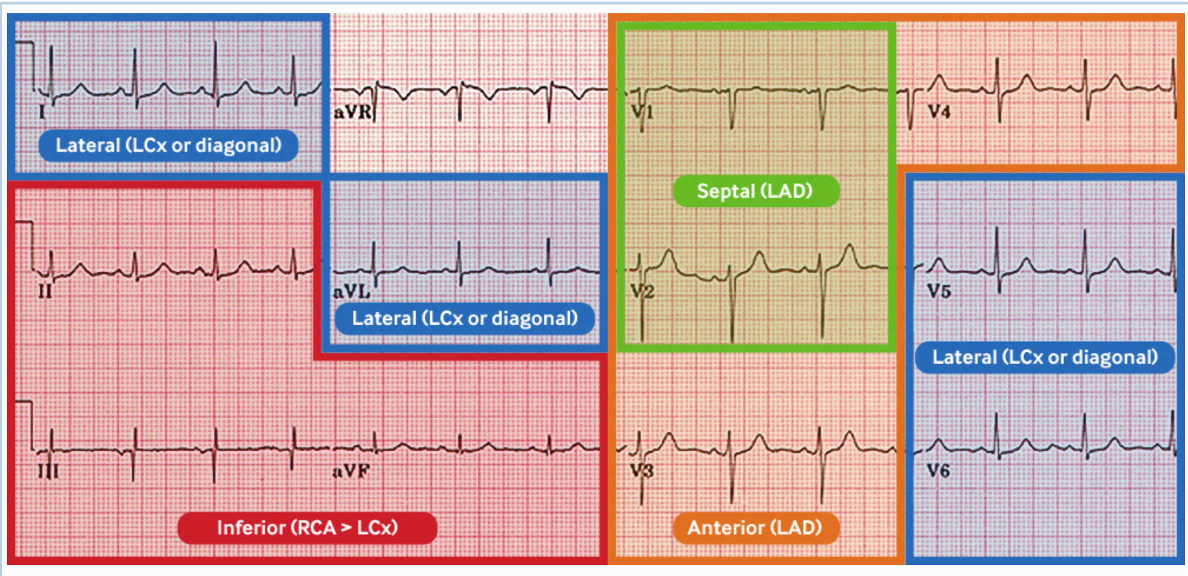
Practical tip

Contiguous leads lie next to each other anatomically and indicate a specific myocardial territory.



12-lead ECG placement

Created by Npatchett (own work) [CC BY-SA 4.0], via Wikimedia Commons



Coronary anatomy and ECG leads

Created by the BMJ Knowledge Centre

Leads	Anatomic location	Coronary artery
II, III, aVF	Inferior	RCA >> LCx
V2 - V4	Anterior	LAD

Less common ECG presentations

Posterior STEMI [1] [2]

- Consider this when there is **ST-segment depression in leads V1-V3** along with characteristic signs and symptoms of myocardial ischaemia.
- **Confirm with posterior lead ECG**: ST-segment elevation ≥0.5 mm in V7-V9.

Right ventricular infarction [1] [2] [121]

- Can complicate an inferior STEMI.
- Check right precordial leads (V3R and V4R) for ST-segment elevation.
- Look for **ST elevation ≥1 mm in aVR and V1**.
- Confirmation will have an impact on choice of therapeutic intervention.

Left main coronary obstruction [2] [84]

- Consider **complete left main coronary artery obstruction** if the following are both present, especially if the patient has haemodynamic compromise:
  - ST depression ≥1 mm in ≥6 surface leads (i.e., inferolateral ST depression)
  - ST elevation in aVR or lead V1.

STEMI in the presence of LBBB

ECG diagnosis of STEMI is trickier in the presence of LBBB.[2] [86] [122]

- Bundle branch block (BBB) precludes accurate assessment, but it may be possible to make the diagnosis if **marked ST-segment abnormalities** are present.[2]
  - The presence of **concordant ST-segment elevation (i.e., in leads with positive QRS deflections)** is one of the best indicators of total coronary occlusion and ongoing MI in the context of a patient with concomitant LBBB.[85]
- Manage any patient with BBB and clinical suspicion of ongoing myocardial ischaemia as per **the standard STEMI protocol**. This applies to both left and right bundle branch block, whether new or previously known.[2]
- Presumed new LBBB alone does not indicate the presence of a STEMI.[87] [88]
- If in doubt, **seek immediate input from the cardiology team**.

More info: Sgarbossa criteria for diagnosis of MI in the presence of LBBB

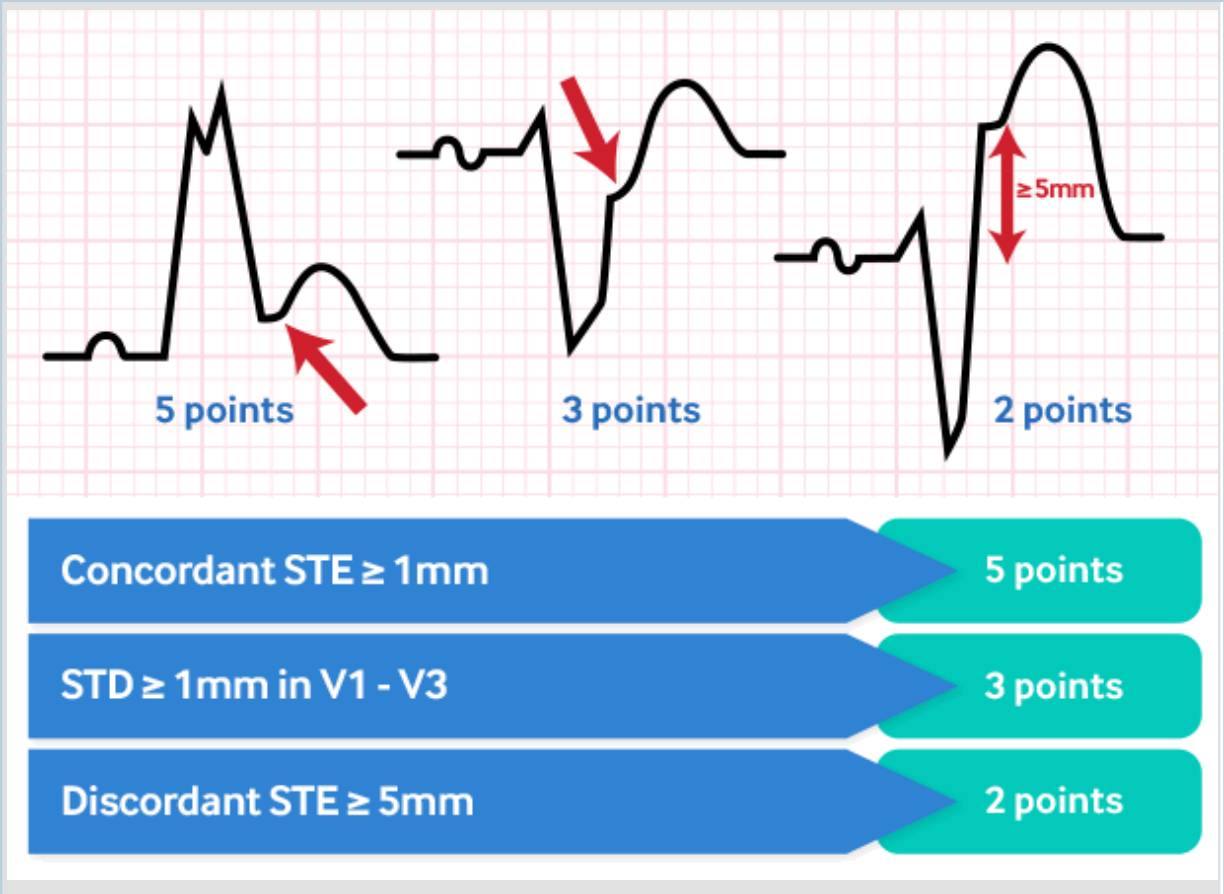
Consider using the Sgarbossa criteria to improve the diagnostic accuracy for STEMI in patients who have LBBB at presentation.

Original criteria [1] [86]

- ST elevation of ≥1 mm, concordant with the QRS complex: **5 points**.
- ST depression ≥1 mm in leads V1, V2, or V3: **3 points**.
    - This has a sensitivity of 19% and a specificity of 81% to diagnose acute MI.[123]
- ST elevation ≥5 mm, discordant with the QRS complex: **2 points**.
    - This has a sensitivity of 10% and a specificity of 99% to diagnose acute MI.[123]
- An aggregated **score of 3+ is 90% specific for MI but only 36% sensitive.**

Components of the Sgarbossa criteria have high specificity but low sensitivity so are useful to confirm acute MI but less useful to rule it out. [124]

- The low sensitivity means you must **maintain a high index of suspicion if the presentation is consistent with MI regardless of the criteria score**.
- 'Weighted' Sgarbossa criteria rely on the points system; however, only two of the criteria carry a score ≥3 to make the diagnosis of acute MI.[125]
- 'Unweighted' Sgarbossa criteria are applied without the points system – this is more sensitive but less specific.[125]
- The criteria were originally based on the outcome of acute MI as measured by creatine kinase-MB rather than angiographic evidence of acute coronary occlusion – further reducing sensitivity because the criteria encompass both STEMI and NSTEMI.[125]

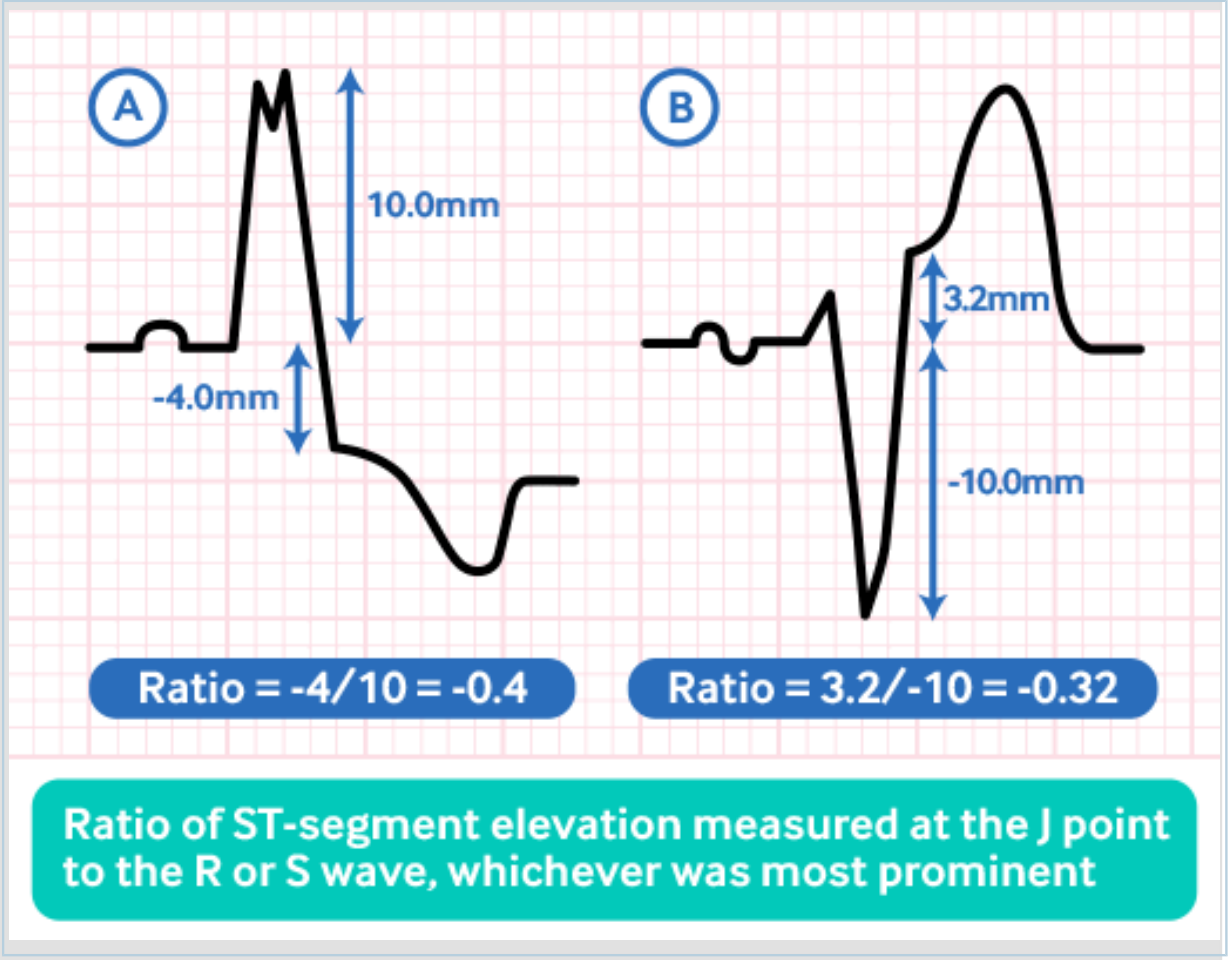


Sgarbossa criteria for MI in the presence of LBBB
Created by the BMJ Knowledge#Centre

The modified Sgarbossa criteria have better sensitivity but worse specificity for STEMI. [125] [126]

- The original rule for >5 mm discordance is replaced with a proportionately excessive discordance: ST-elevation/S-wave amplitude ≤-0.25.
- The modified criteria were found to be more sensitive versus the 'weighted' (80% vs. 49%; P <0.001) and 'unweighted' (80% vs. 56%; P <0.001) Sgarbossa criteria.[125]
    - Modified criteria specificity is not significantly different from the 'weighted' criteria.[125]
    - Modified criteria specificity is significantly greater than the 'unweighted' original criteria.[125]

DIAGNOSIS



ST/S ratio under the modified Sgarbossa criteria
Created by the BMJ Knowledge Centre

Previous silent/unrecognised STEMI

Be aware of the possibility that abnormal ECG features might be due to a **previous silent/unrecognised STEMI.** [1]

- Residual ST elevation on the ECG from an old STEMI may be detected either incidentally in an asymptomatic patient who is having an ECG for another reason, or occasionally in a patient with symptoms of ischaemia who is experiencing a non-ST-elevation MI (NSTEMI) against the background of a previous history of STEMI.
- In such cases, there may be ST-segment elevation on the current ECG that was **already present on old ECGs** (e.g., in the case of left ventricular aneurysm formation). It is **important to distinguish this from new ST elevation** as management will differ.
    - The historical STEMI may have been 'silent' and gone unrecognised at the time. The resulting old **ECG features will usually be fixed**.
- It is helpful to take a thorough history, together with diligent ECG interpretation and comparison with old ECGs (plus medical records) if available.
- Seek specialist input from the cardiology team.

The following criteria for previous (or silent/unrecognised) MI can be helpful. There may be:[1]

- **Pathological Q waves** with or without symptoms, in the absence of non-ischaemic causes[1]

- **Loss of viable myocardium in a pattern consistent with a coronary artery territory** (regional wall abnormality) seen **on cardiac imaging**:
  - Echocardiography
  - Myocardial perfusion scintigraphy (MPS)
  - Positron emission tomography (PET)
  - Cardiac magnetic resonance imaging
- **Pathological findings of a healed or healing MI** on cardiac imaging.

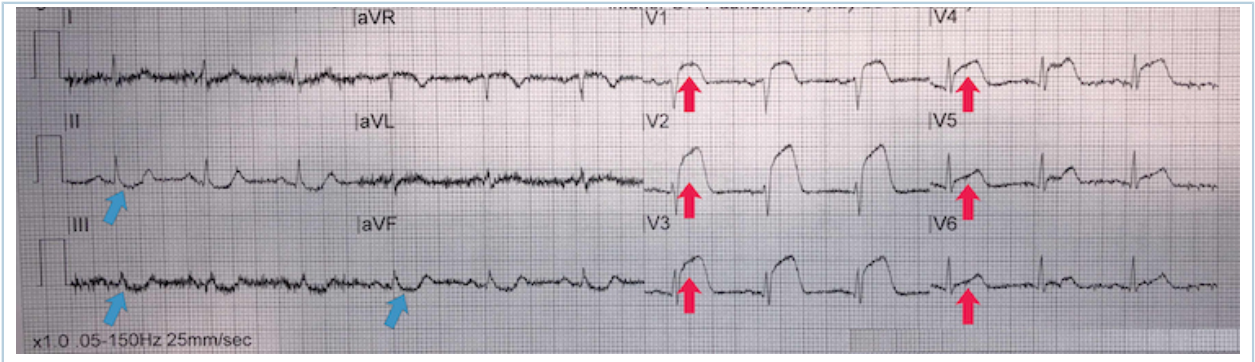
Practical tip

A Q wave is defined as any negative deflection preceding an R wave in the QRS complex.

- A Q wave represents the normal left-to-right depolarisation across the interventricular septum.
- Small Q waves tend to be normal in most leads (e.g., left-sided leads such as I, aVL, V5, and V6).
- More pronounced Q waves (>2 mm deep) may be seen in lead III or aVR as a normal variant.
- Q waves are not usually seen in the right-sided leads (V1-V3).
- Pathological Q waves can be a sign of previous MI. The precise definition of pathological Q waves has been debated. The 2018 Fourth Universal Definition of MI defines them as follows:[1]
  - Any Q wave in leads V2-V3 >20 milliseconds (0.02 seconds) or any QS complex in leads V2 and V3
  - Q wave ≥30 milliseconds (0.03 seconds) and ≥1 mm deep or QS complex in leads I, II, aVL, aVF, or V4-V6 in any two leads of a contiguous lead grouping (I, aVL; V1-V6; II, III, aVF)
  - R wave >40 milliseconds (0.04 seconds) in V1-V2 and R/S >1 with a concordant positive T wave in the absence of a conduction defect.

ECG examples

Anterior STEMI



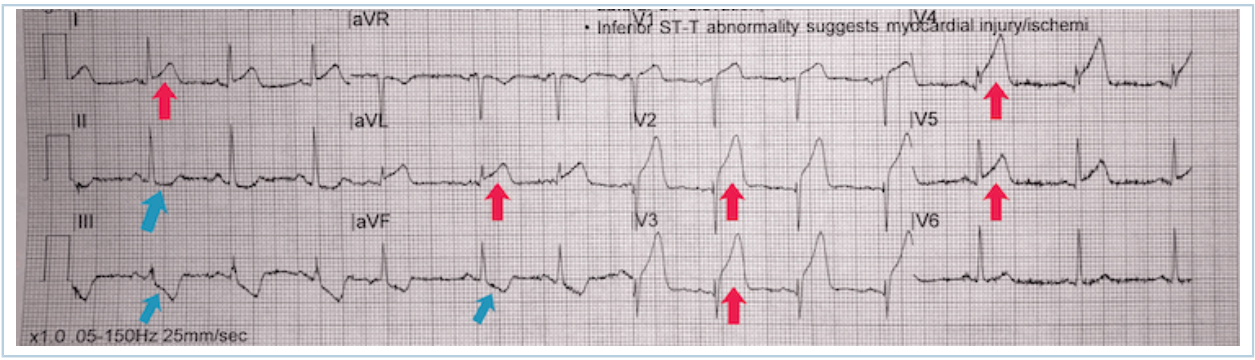
Anterior STEMI
From the personal collection of Dr Aung Myat (used with permission)

Key learning points

- Tombstone' ST elevation in anterior chest leads V1-V6 = anterior STEMI (red arrows)
- Reciprocal inferior ST-segment depression in II, III, and aVF (blue arrows)

- This is a high-risk ECG

Anterolateral STEMI example I

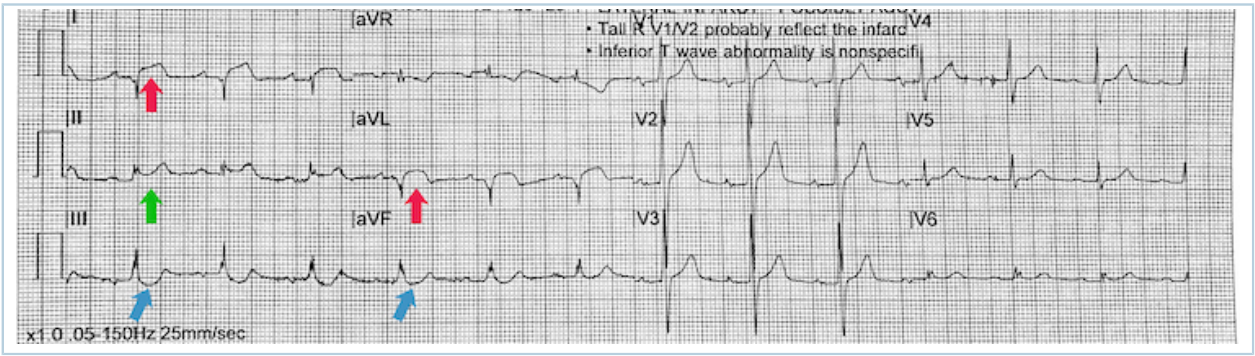


Anterolateral STEMI example I
From the personal collection of Dr Aung Myat (used with permission)

Key learning points

- 'Tombstone' ST elevation in leads V2-V6, I, and aVL = anterolateral STEMI (red arrows)
- Reciprocal inferior ST-segment depression in II, III, and aVF (blue arrows)

High lateral STEMI

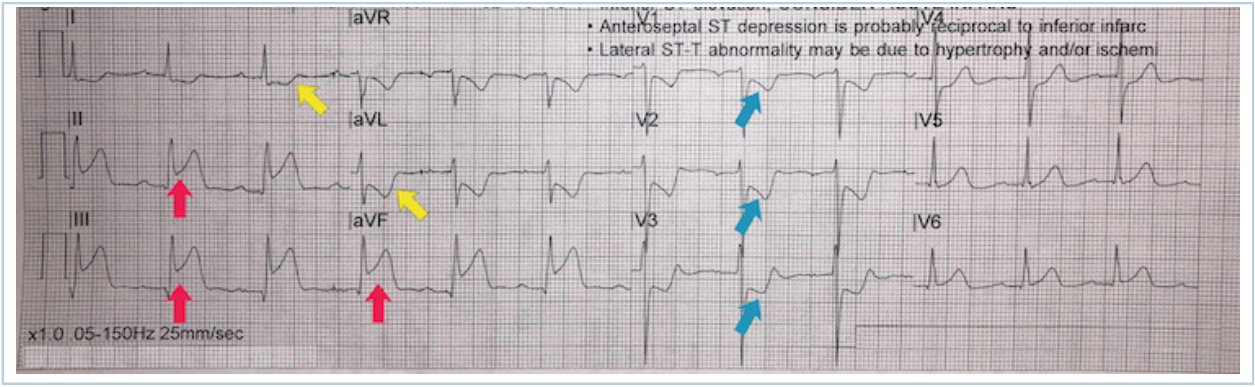


High lateral STEMI
From the personal collection of Dr Aung Myat (used with permission)

Key learning points

- ST-segment elevation in high lateral chest leads I and aVL = high lateral STEMI (red arrows)
- Reciprocal inferior ST-segment depression in leads III and aVF (blue arrows)
- There is also saddle-shaped ST-segment elevation in lead II (green arrow) – difficult to state the significance of this

Inferoposterior STEMI example II

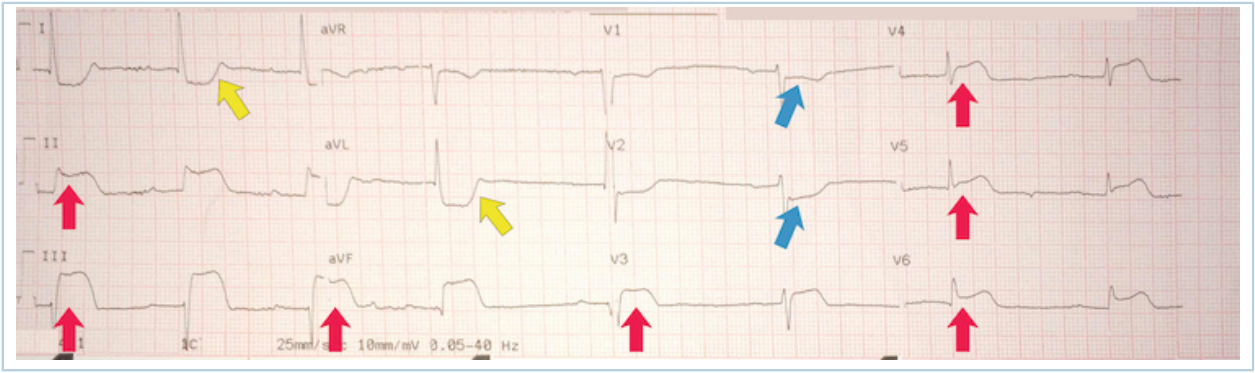


Inferoposterior STEMI#example II
From the personal collection of Dr Aung Myat (used with permission)

Key learning points

- Note the deep ST-segment depression and R:S wave ratio of >1 in V1-V3 = posterior STEMI (blue arrows)
- ST-segment elevation in leads II, III, and aVF = inferior STEMI (red arrows)
- Reciprocal ST-segment depression in the lateral leads I and aVL (yellow arrows)
- Consider performing a posterior lead ECG (leads V7-V9) for further confirmation of a posterior STEMI

Inferoposterolateral STEMI example III



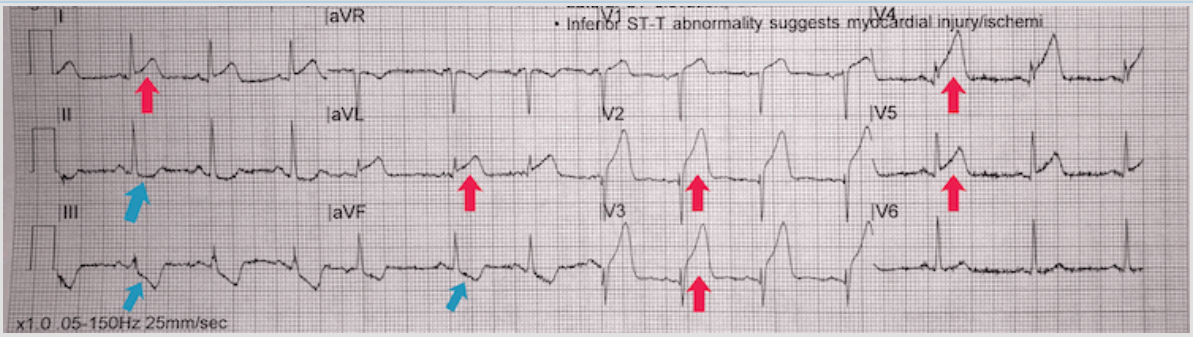
Inferoposterolateral STEMI example III
From the personal collection of Dr Aung Myat (used with permission)

Key learning points

- Note the deep ST-segment depression and R:S wave ratio of >1 in V1-V2 = posterior STEMI (blue arrows)
- ST-segment elevation in leads II, III, and aVF = inferior STEMI (red arrows)
- ST-segment elevation in leads V3-V6 = lateral STEMI (green arrows)
- Reciprocal deep ST-segment depression in leads I and aVL (yellow arrows)
- The patient has marked sinus bradycardia – there can be several reasons for this but with this degree of ischaemia on the ECG it suggests current or impending haemodynamic instability

More info: Library of ECGs with learning points

Anterolateral STEMI example II

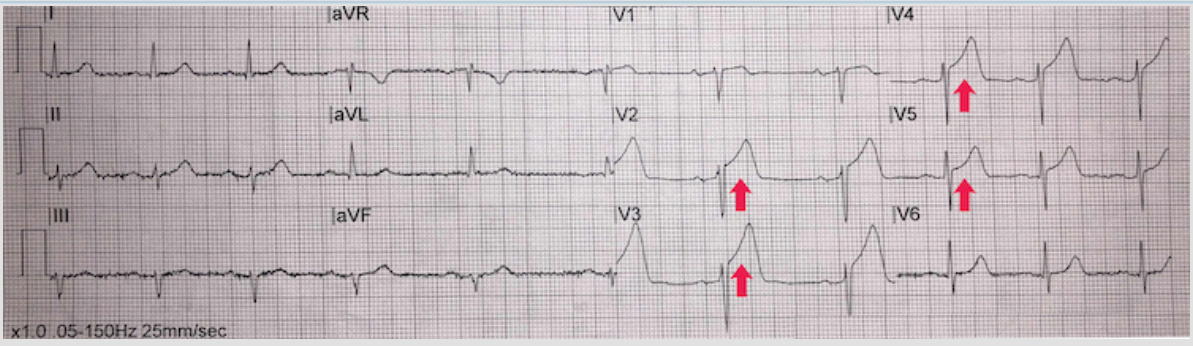


Anterolateral STEMI example II
From the personal collection of Dr Aung Myat (used with permission)

Key learning points

- ST elevation in leads V2-V5, I, and aVL = anterolateral STEMI (red arrows)
- Reciprocal inferior ST-segment depression in II, III, and aVF (blue arrows)
- Note there are no pathological Q waves in the anterior chest leads (V2-V6) – contrast with Anterolateral STEMI example I)

Anteroseptal STEMI example I

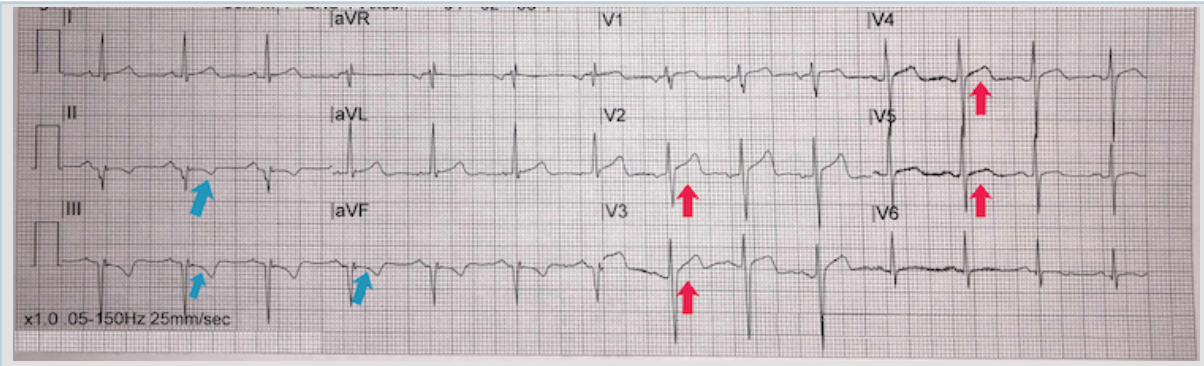


Anteroseptal STEMI example I
From the personal collection of Dr Aung Myat (used with permission)

Key learning points

- ST-segment elevation in leads V2-V5 = anteroseptal STEMI (red arrows)
- There are, however, no reciprocal ischaemic changes on the ECG
- Reciprocal ST-segment depression represents either true distant ischaemia as a by-product of a collateral circulation or an electrical phenomenon arising from a mirror reflection of ST-segment elevation elsewhere
- Take a careful history and examination with this ECG – the anteroseptal ST-segment elevation may represent 'high take-off'(or early benign repolarisation) rather than true myocardial ischaemia
- If there are signs and symptoms of ongoing myocardial ischaemia then treat as a STEMI

Anteroseptal STEMI example II

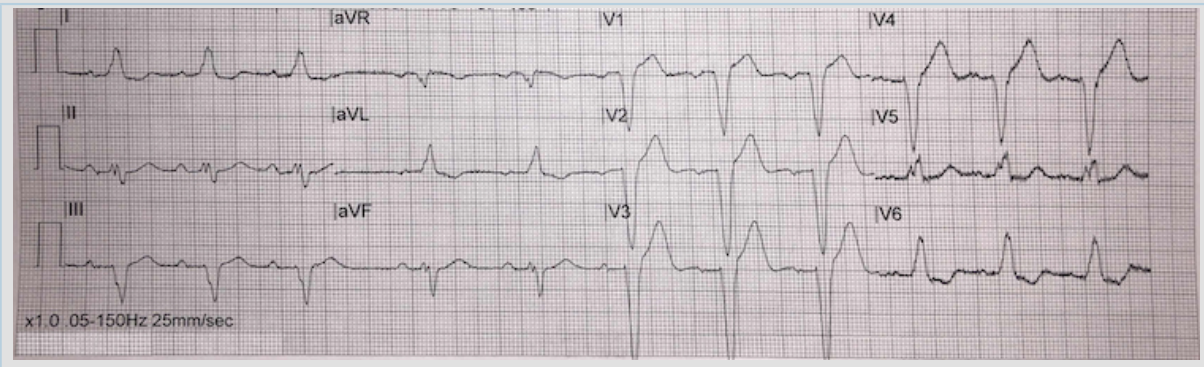


Anteroseptal STEMI example II
From the personal collection of Dr Aung Myat (used with permission)

Key learning points

- ST-segment elevation in leads V2-V5 = anteroseptal STEMI (red arrows)
- Reciprocal ST-segment depression in the inferior leads II, III, and aVF
- Compared with Anteroseptal STEMI example I: here the ST-segment elevation is less pronounced but the reciprocal ST-segment depression inferiorly make this ECG more compelling for an acute MI
- Again, take a careful history and examination looking for signs and symptoms of ongoing myocardial ischaemia

Left bundle branch block example I

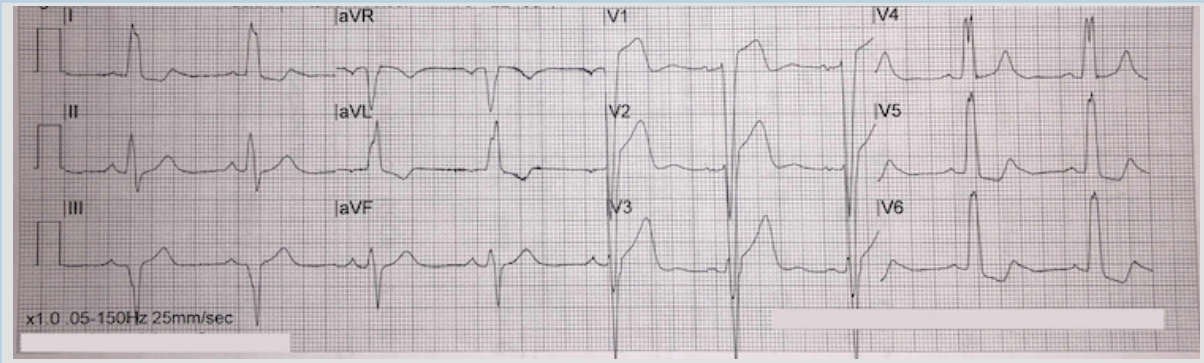


Left bundle branch block example I
From the personal collection of Dr Aung Myat (used with permission)

Key learning points

- **Left bundle branch block (LBBB) ECG criteria**:
  - QRS duration >120 milliseconds (i.e., greater than 3 small squares on the ECG)
  - Monomorphic R wave in leads I, V5, and V6 – look for the characteristic 'M-pattern' shape (notched R wave) to the QRS complex in leads V5 and V6
  - Deep and broad S wave in leads V1-V2
  - ST-segment depression and T-wave inversion in left-sided leads (V5, V6, I, and aVL)
  - ST-segment elevation and positive T waves in V1-V3 (ST-segment elevation rarely exceeds >5 mm – refer to Sgarbossa versus modified Sgarbossa criteria)

Left bundle branch block example II

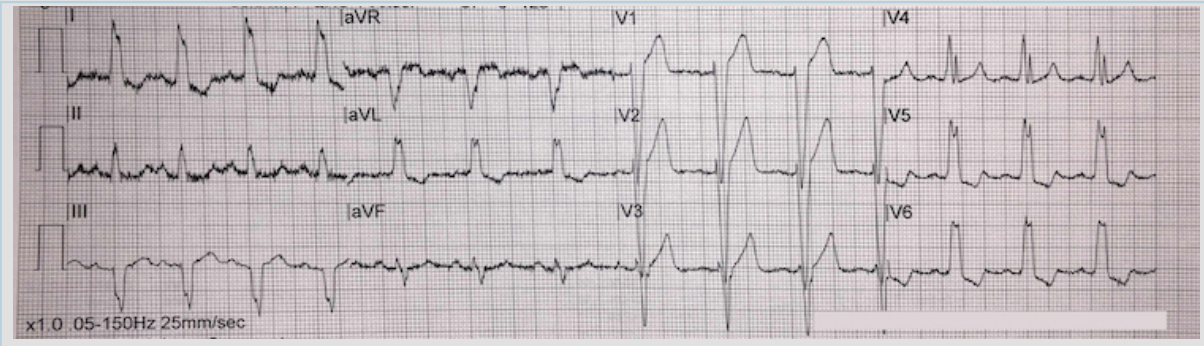


Left bundle branch block example II
From the personal collection of Dr Aung Myat (used with permission)

Key learning points

- **LBBB ECG criteria:**
  - QRS duration >120 milliseconds (i.e., greater than 3 small squares on the ECG)
  - Monomorphic R wave in leads I, V5, and V6 – look for the characteristic 'M-pattern' shape (notched R wave) to the QRS complex in leads V5 and V6
  - Deep and broad S wave in leads V1-V2 – more pronounced when compared with example I
  - ST-segment depression and T-wave inversion in left-sided leads (V5, V6, I, and aVL)
  - ST-segment elevation and positive T waves in V1-V3 (ST-segment elevation rarely exceeds >5 mm – refer to Sgarbossa versus modified Sgarbossa criteria)

Left bundle branch block example III



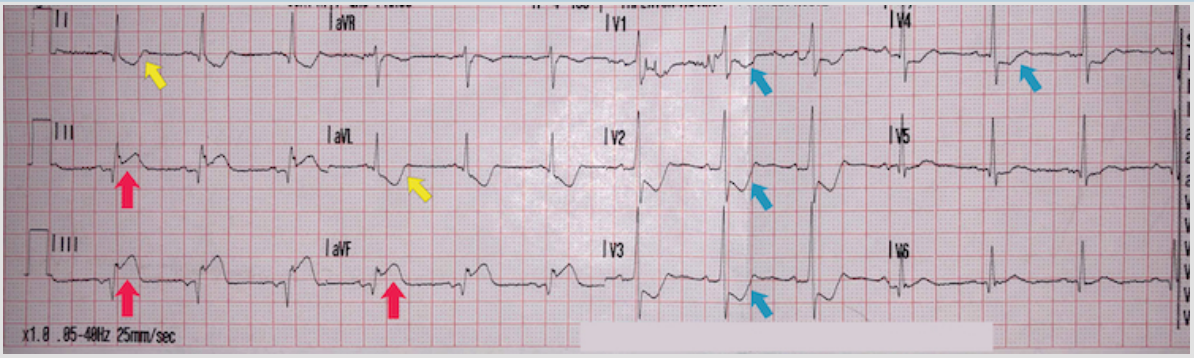
Left bundle branch block example III
From the personal collection of Dr Aung Myat (used with permission)

Key learning points

- **LBBB ECG criteria:**
  - QRS duration >120 milliseconds (i.e., greater than 3 small squares on the ECG)
  - Monomorphic R wave in leads I, V5, and V6 – look for the characteristic 'M-pattern' shape (notched R wave) to the QRS complex in leads V5 and V6, which are very pronounced here

- Deep and broad S wave in leads V1-V2 – just as pronounced when compared with example II
- ST-segment depression and T-wave inversion in left-sided leads (V5, V6, I, and aVL)
- ST-segment elevation and positive T waves in V1-V3 (ST-segment elevation rarely exceeds >5 mm – refer to Sgarbossa versus modified Sgarbossa criteria)

Inferoposterior STEMI example I

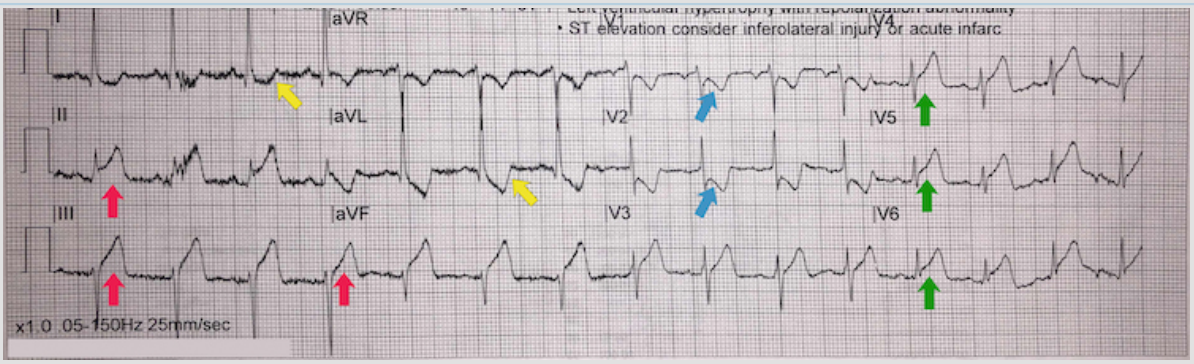


Inferoposterior STEMI example I
From the personal collection of Dr Aung Myat (used with permission)

Key learning points

- Note the deep ST-segment depression and R:S wave ratio of >1 in V1-V4 = posterior STEMI (blue arrows)
- ST-segment elevation in leads II, III, and aVF = inferior STEMI (red arrows)
- Reciprocal ST-segment depression in the lateral leads I and aVL (yellow arrows)
- Consider performing a posterior lead ECG (leads V7-V9) for further confirmation of a posterior STEMI

Inferoposterolateral STEMI example I



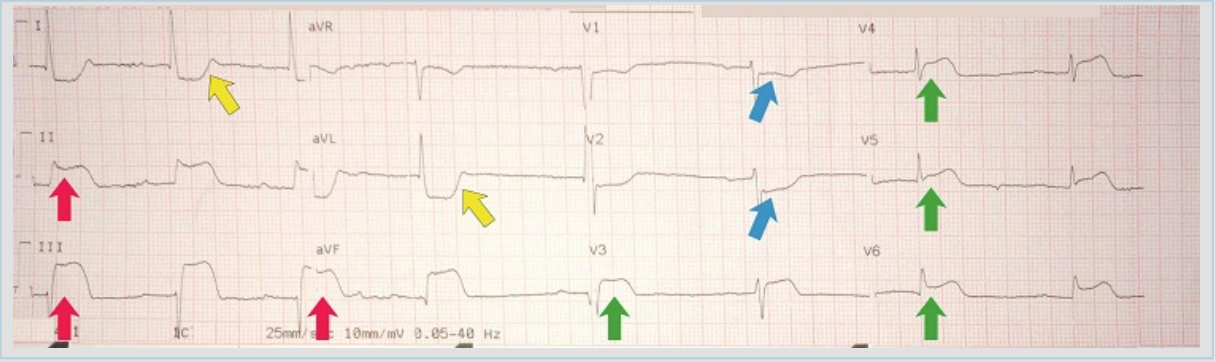
Inferoposterolateral STEMI example I
From the personal collection of Dr Aung Myat (used with permission)

Key learning points

- Note the deep ST-segment depression and R:S wave ratio of >1 in V1-V2 = posterior STEMI (blue arrows)
- ST-segment elevation in leads II, III, and aVF = inferior STEMI (red arrows)

- Reciprocal ST-segment depression in the lateral leads I and aVL (yellow arrows)
- Also note the ST-segment elevation in leads V4-V6 (green arrows) – this could suggest lateral involvement of the STEMI and/or represent occlusion of a very large dominant right coronary artery with a posterior descending branch artery wrapping around the left ventricular apex
- Consider performing a posterior lead ECG (leads V7-V9) for further confirmation of a posterior STEMI

Inferoposterolateral STEMI example II

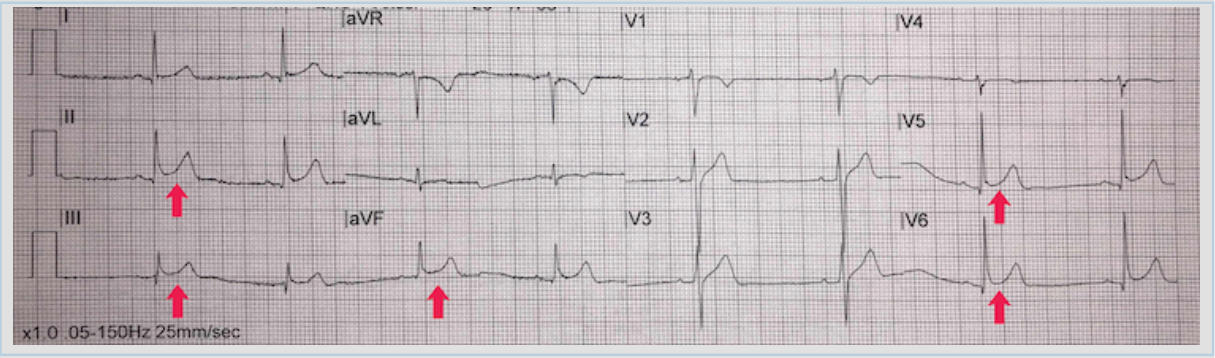


Inferoposterolateral STEMI example II
From the personal collection of Dr Aung Myat (used with permission)

Key learning points

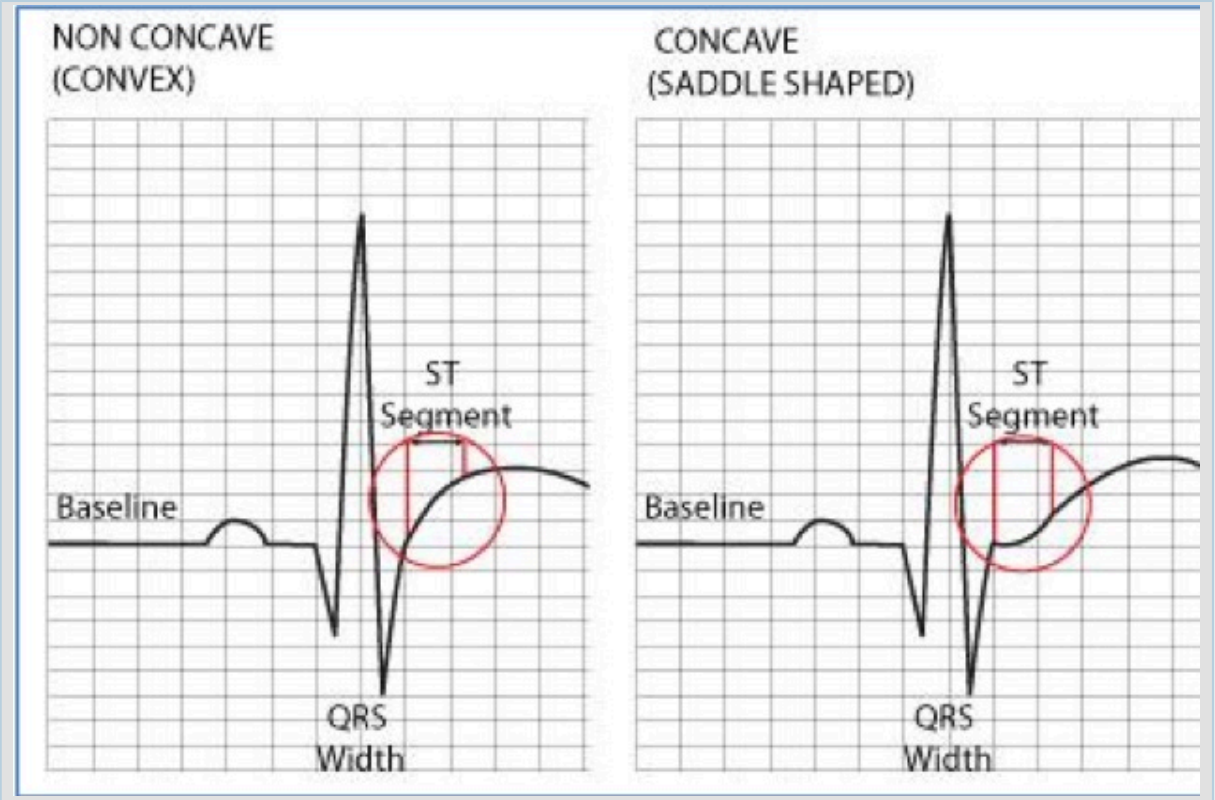
- Note the deep ST-segment depression and R:S wave ratio of >1 in V1-V2 = posterior STEMI (blue arrows)
- ST-segment elevation in leads II, III, and aVF = inferior STEMI (red arrows)
- ST-segment elevation in leads V3-V6 = lateral STEMI (green arrows)
- Reciprocal deep ST-segment depression in leads I and aVL (yellow arrows)
- The patient has marked sinus bradycardia – there can be several reasons for this but with this degree of ischaemia on the ECG it suggests current or impending haemodynamic instability

Possible' inferolateral STEMI



Possible' inferolateral STEMI
From the personal collection of Dr Aung Myat (used with permission)

DIAGNOSIS

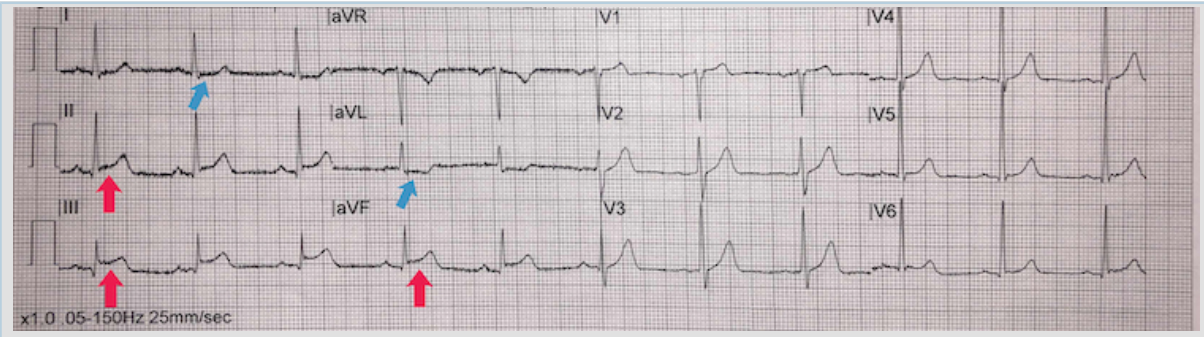


Possible' inferolateral STEMI: ST-segment shift
Created by the BMJ Knowledge Centre

Key learning points

- ST-segment elevation in the inferior leads II, III, and aVF and the lateral leads V5-V6 (red arrows)
- The ST-segment shift, however, is more saddle-shaped (concave) rather than convex – see second figure above
- A convex ST-segment elevation morphology is more likely to be associated with an acute myocardial infarction
- There are no reciprocal ischaemic changes elsewhere on the ECG either
- Take a thorough history and examination looking for the signs of myocardial ischaemia and also seek a specialist cardiology consult when managing a patient who presents with this ECG – **it can still be a STEMI**
- Also consider acute pericarditis as a differential

Inferior STEMI example I

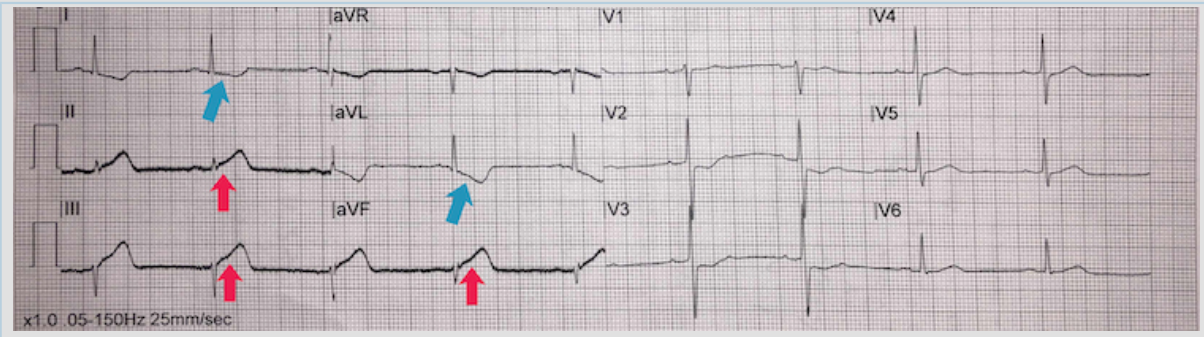


Inferior STEMI example I
From the personal collection of Dr Aung Myat (used with permission)

Key learning points

- ST-segment elevation in leads II, III, and aVF = inferior STEMI (red arrows)
- Subtle reciprocal ST-segment depression in the lateral leads I and aVL (blue arrows)

Inferior STEMI example II

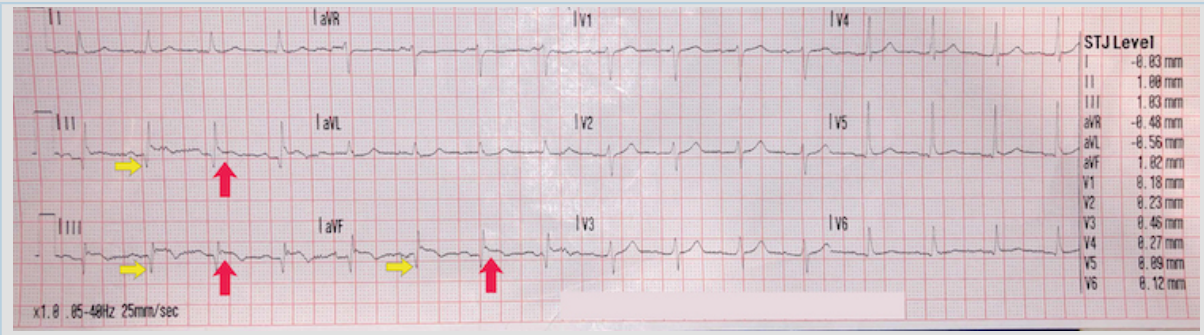


Inferior STEMI example II
From the personal collection of Dr Aung Myat (used with permission)

Key learning points

- Subtle ST-segment elevation in leads II, III, and aVF = inferior STEMI (red arrows)
- Deep reciprocal ST-segment depression in the lateral leads I and aVL (blue arrows)
- Take a thorough history and examination for signs and symptoms of myocardial ischaemia

Inferior STEMI example III



Inferior STEMI example III
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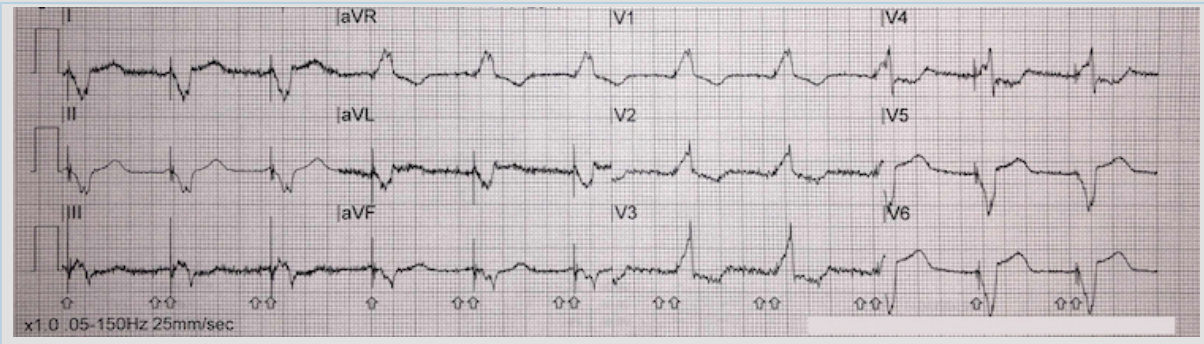
Key learning points

- ST-segment elevation in leads II, III, and aVF = inferior STEMI (red arrows)
- Pathological Q waves in leads II, III, and aVF (yellow arrows)
- Note the lack of reciprocal ST-segment changes in other leads
- These changes suggest a late-presentation inferior STEMI
- Consult the cardiology team to discuss management options once you have performed a thorough history and examination

Paced rhythm example I

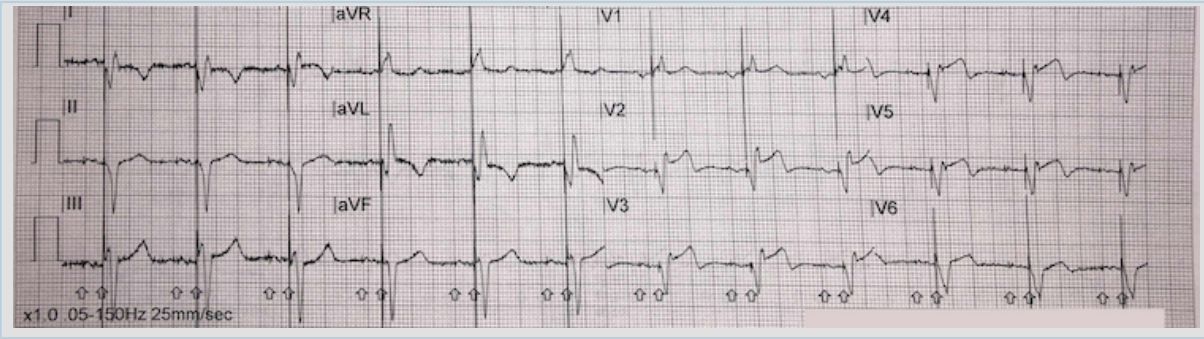


Paced rhythm example I
From the personal collection of Dr Aung Myat (used with permission)

Key learning points

- There is a right bundle branch pattern here due to cardiac pacing
    - Note the QRS is negative in lead I and positive in V1
- There is atrial pacing here with the pacing spike preceding the P wave
- There is ventricular pacing here also with the pacing spike preceding the QRS complex
- Right ventricular pacing = QRS morphology similar to left bundle branch block
- Left epicardial pacing = QRS morphology similar to right bundle branch block pattern
- We cannot diagnose a STEMI from a paced rhythm ECG but this does not mean an acute MI can be excluded if a patient presents with the signs and symptoms of ongoing myocardial ischaemia

Paced rhythm example II

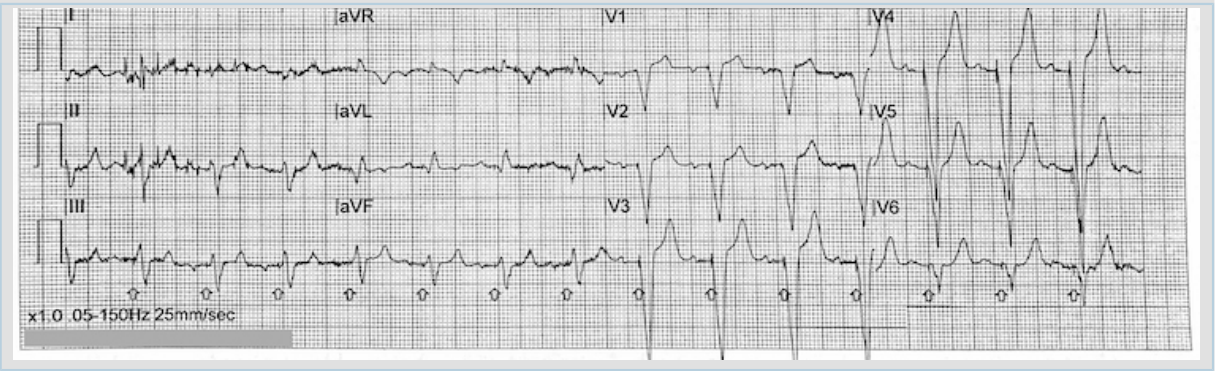


Paced rhythm example II
From the personal collection of Dr Aung Myat (used with permission)

Key learning points

- There is atrial pacing here with the pacing spike preceding the P wave
- There is ventricular pacing here with the pacing spike preceding the QRS complex
- We cannot diagnose a STEMI from a paced rhythm ECG but this does not mean an acute MI can be excluded if a patient presents with the signs and symptoms of ongoing myocardial ischaemia

Paced rhythm example III

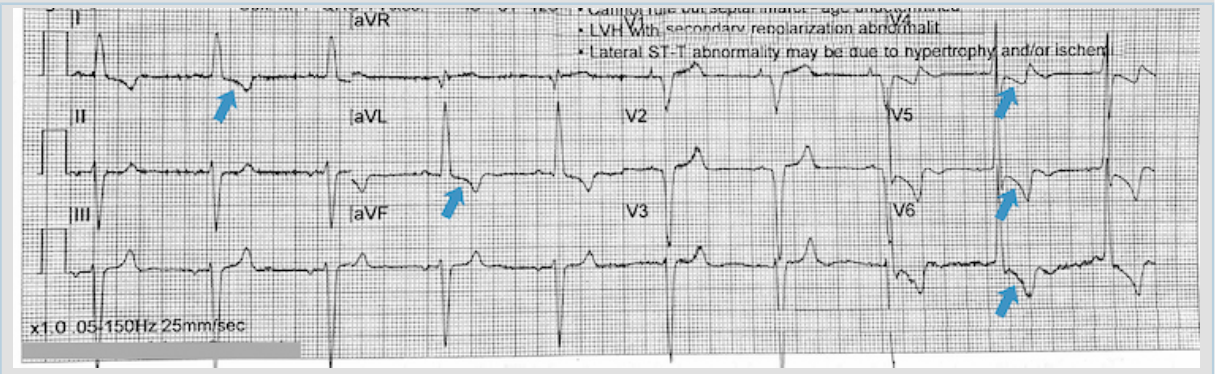


Paced rhythm example III
From the personal collection of Dr Aung Myat (used with permission)

Key learning points

- There is a bundle branch pattern here due to cardiac pacing
- There is only ventricular pacing here with the pacing spike preceding the QRS complex
- Right ventricular pacing = QRS morphology similar to left bundle branch block – this is the case on this ECG
- We cannot diagnose a STEMI from a paced rhythm ECG but this does not mean an acute MI can be excluded if a patient presents with the signs and symptoms of ongoing myocardial ischaemia

Left ventricular hypertrophy example I

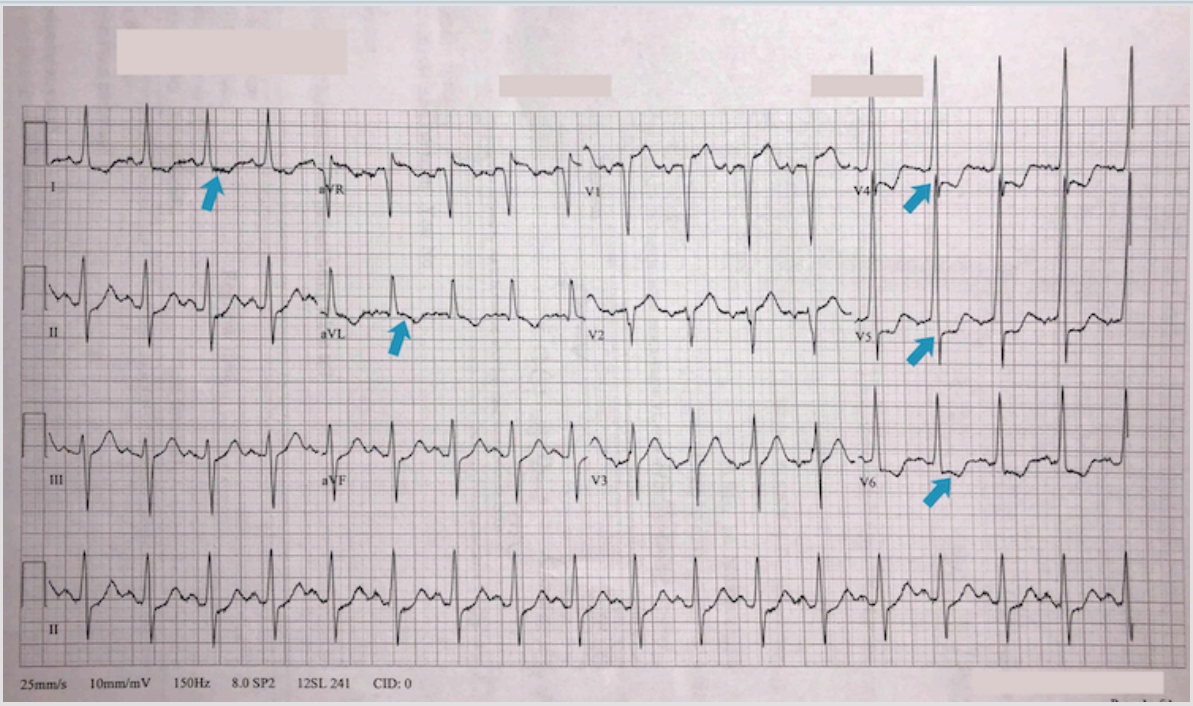


Left ventricular hypertrophy example I
From the personal collection of Dr Aung Myat (used with permission)

Key learning points

- A STEMI cannot be diagnosed from the ECG on a background of left ventricular hypertrophy (LVH)
- Numerous criteria for diagnosing LVH
- Sokolov-Lyon voltage criteria: S wave depth in V1 + tallest R wave height in V5 or V6 >35 mm
- Non-voltage criteria: ST-segment depression and T-wave inversion in left-sided leads I, aVL, V4-V6 (blue arrows) – this is often referred to as a **left heart strain pattern**
- Voltage and non-voltage criteria must be present to confirm an ECG diagnosis of LVH

Left ventricular hypertrophy example II

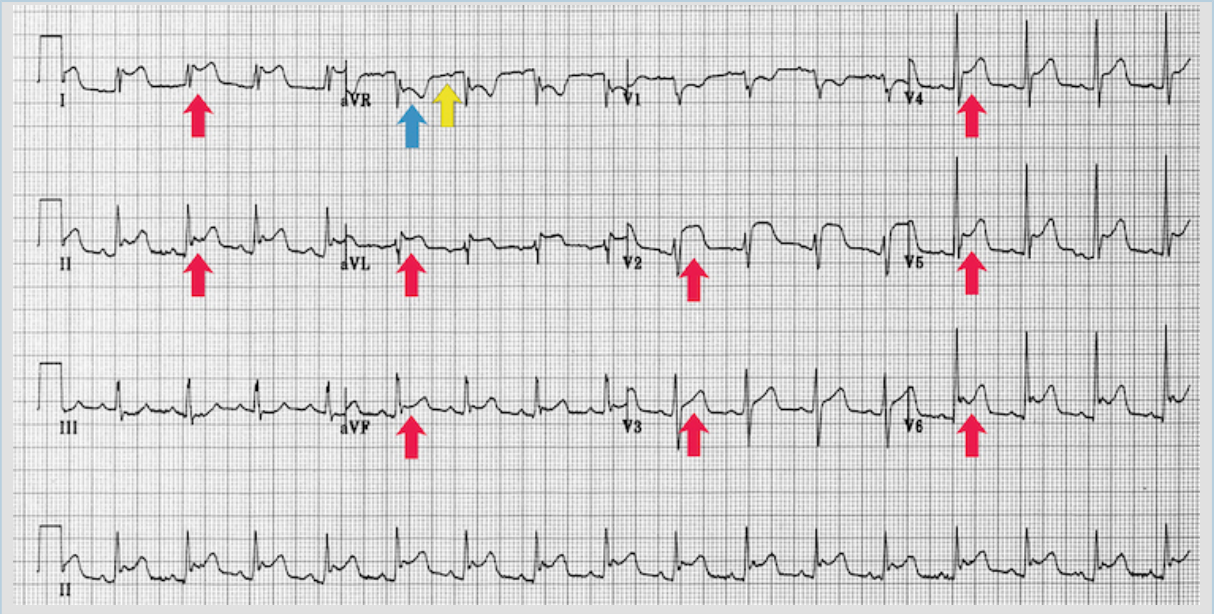


Left ventricular hypertrophy example II
From the personal collection of Dr Aung Myat (used with permission)

Key learning points

- A STEMI cannot be diagnosed from the ECG on a background of left ventricular hypertrophy (LVH)
- Sokolov-Lyon voltage criteria: S wave depth in V1 + tallest R wave height in V5 or V6 >35 mm
- Non-voltage criteria: ST-segment depression and T-wave inversion in left-sided leads I, aVL, V4-V6 (blue arrows) – this is often referred to as a **left heart strain pattern**
- Voltage and non-voltage criteria must be present to confirm an ECG diagnosis of LVH
- Also note this type of ECG may be a normal variant in athletes and people of African-Caribbean heritage

Acute pericarditis



Acute pericarditis
From the personal collection of Dr Aung Myat (used with permission)

Key learning points

- Global saddle-shaped (concave) ST-segment elevation (red arrows)
- Reciprocal ST depression (blue arrow) and PR depression (yellow arrow) in leads II and aVF
- Sinus tachycardia can also feature due to the increased sympathetic drive from pain and/or to maintain cardiac output if there is a large pericardial effusion associated with the inflamed pericardium
- This ECG appearance does not exclude STEMI – ensure a thorough history and examination and if any doubt seek specialist help

Coronary angiography

After making a clinical diagnosis of STEMI, offer coronary angiography, with follow-on primary percutaneous coronary intervention (PCI) if indicated, as the preferred coronary reperfusion strategy, if:[75]

- The patient presents **with persistent typical central chest pain or** other symptoms consistent with myocardial ischaemia (**chest pain-equivalent symptoms**) within the last 12 hours
- Primary PCI can be delivered within **120 minutes** of the ECG-based diagnosis.

When primary PCI is **not an immediate option** and cannot be provided within 120 minutes of ECG diagnosis, **fibrinolysis should be initiated**expeditiously as part of a pharmaco-invasive strategy, provided the patient has presented **within 12 hours of symptom onset**.[75]

- **Seek immediate specialist advice** from cardiology to discuss management options for any STEMI patient **who presents >12 hours after symptom onset**. Coronary angiography ± primary PCI should be considered if there is evidence of continuing myocardial ischaemia, haemodynamic instability or cardiogenic shock, or life-threatening arrhythmias.[2]

If a patient with acute STEMI has had fibrinolysis, offer **angiography**:[75]

- Immediately if an ECG 60-90 minutes after fibrinolysis shows residual ST-segment elevation (rescue PCI)
- After seeking specialist cardiology advice if the patient has recurrent myocardial ischaemia after fibrinolysis
- During the same hospital admission if the patient is stable after successful fibrinolysis (routine PCI strategy).

Radial arterial access is preferred to femoral access in all patients undergoing coronary angiography.[75]

Laboratory work-up

Do not delay coronary reperfusion treatment to wait for blood results.[2]

- Start management as soon as a STEMI has been **clinically diagnosed** based on ECG findings together with signs and symptoms consistent with ongoing myocardial ischaemia.[82]
    - **Immediately assess** the patient's **eligibility for coronary reperfusion therapy** (irrespective of age, ethnicity, sex, or level of consciousness).[75]
    - **Alert the interventional cardiology team** using the agreed local protocol.

Cardiac biomarkers

Request a **baseline high-sensitivity cardiac troponin (cTn) along** with your set of routine blood work whenever a patient presents with a possible acute MI – but **do not delay coronary reperfusion** if the patient has a clinical diagnosis of STEMI.[1] [82]

- **Troponin I and T** are the **preferred biomarkers** for definitive confirmation of an MI, with high-sensitivity assays preferred to standard ones.[1] [82]
- Cardiac troponins are biological **markers of cardiac muscle death** (cardiomyocyte necrosis) that are released into the circulation when damage to cardiac muscle has occurred.[1] [89]
- Creatine kinase-MB fraction is less sensitive and less specific and is now rarely used or measured.[1]

A pathological rise in troponin level followed by a later fall provides **definitive confirmation of an acute MI** in a patient who has clinical/ECG evidence of ongoing myocardial ischaemia.[1] However, STEMI can usually be diagnosed by ECG alone.[89]

- Acute MI is definitively confirmed by a rise and/or fall in cardiac troponin (with **at least one value >99th percentile of the upper reference limit**) in a patient who has symptoms or signs of ischaemia.[1] [82]

Troponin level deviations and normal cut-offs are assay-specific so **check local protocols.**

- Note there is **significant variability in**:[1]
    - The **time to peak value** – levels usually begin to rise around **2-3 hours after onset** of myocardial ischaemia but this varies according to the underlying mechanism
    - The time when a normal value may become greater than the 99th percentile of the upper range limit (URL)[127]
        - The 99th percentile threshold is designated as the decision level for the presence of myocardial injury

- It **varies between men and women** and is established separately for each specific assay[1] [82]

- The time window to observe a fluctuating pattern of values.

- Because assays are not standardised, **values from one assay cannot be compared with those from a different assay**.[1]

Always interpret troponin values in the **context of**:[82]

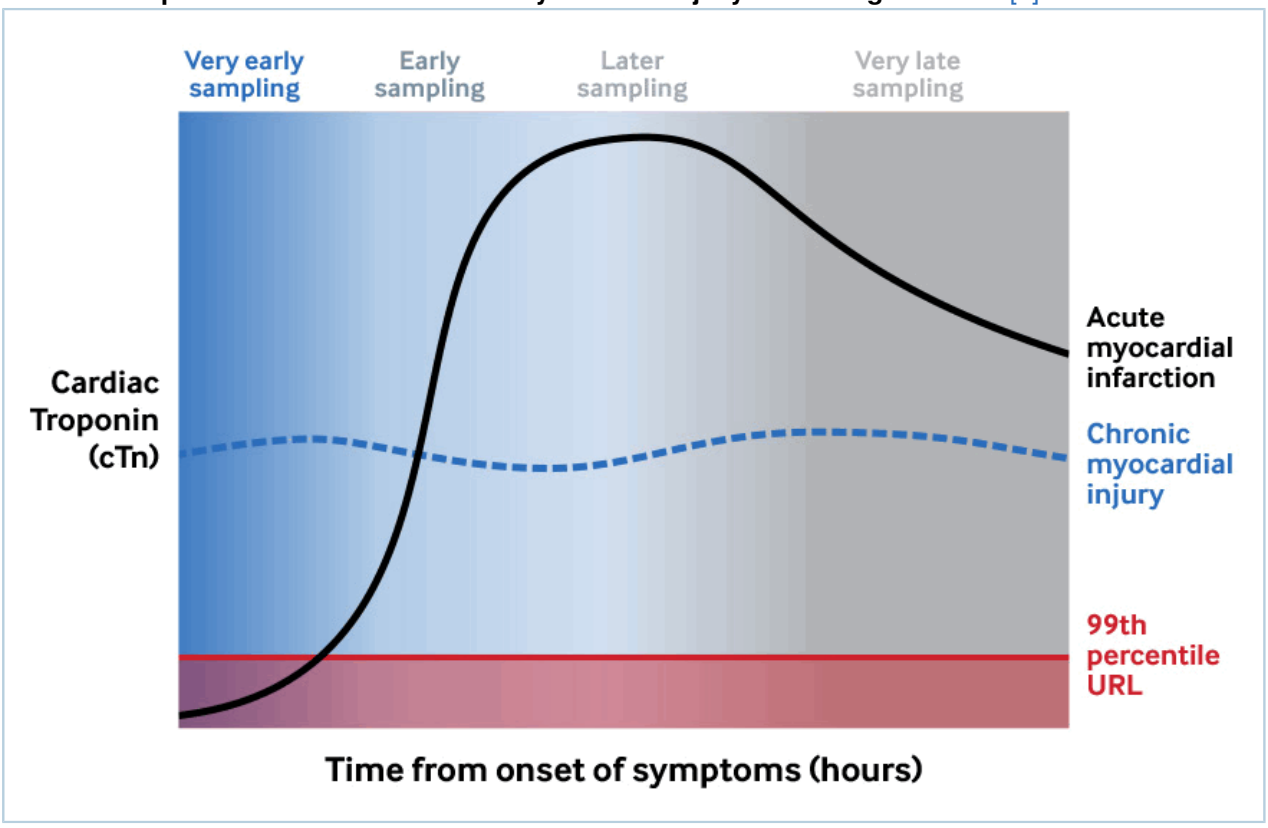
- Current and previous **ECGs**
- Signs and symptoms reported by the patient (in particular, the **time since symptom onset**)
- The possibility of an **alternative cause for an elevated troponin**. This may be cardiac (e.g., myocarditis, aortic dissection, severe heart failure) or non-cardiac (e.g., pulmonary embolism, impaired renal function, underlying sepsis)[82]

    - The demonstration of a **rising and/or falling pattern** is important to distinguish acute myocardial injury from a chronically elevated cTn

- Historical troponin levels recorded for the individual patient (e.g., measured during previous admissions)

    - Some patients may have a **higher baseline**compared with the general population.[82]

Cardiac troponin kinetics after acute myocardial injury including acute MI [1]



Cardiac troponin kinetics after acute myocardial injury including acute MI
Created by the BMJ Knowledge Centre

Other blood tests at presentation

Your routine blood panel should also include the following.

- Full blood count
  - Look for **anaemia**, which may influence the duration of dual antiplatelet therapy prescribed.
  - **Raised inflammatory markers** may be a direct result of the acute-phase response to acute MI or may point to a concomitant infection.
- Urea and electrolytes and estimated glomerular filtration rate (eGFR)
  - **Potassium, calcium, and magnesium** homeostasis is crucially important to prevent both bradyarrhythmias and tachyarrhythmias during the peri-infarct interval.
  - **Baseline renal function** at the time of hospital admission will provide a benchmark to allow for subsequent up-titration of medications and allow for the identification of contrast-induced nephropathy after primary angioplasty (if the patient is eligible for coronary reperfusion therapy).
    - Note patients on potentially **nephrotoxic drugs** on admission, especially if they are eligible for primary angioplasty.
    - Reduce the risk of **contrast-induced nephropathy** if an invasive strategy is planned in a patient with chronic kidney disease.[2]
- Plasma glucose[2]
  - Look for **uncontrolled hyperglycaemia** in all patients (not just those with diabetes). Hyperglycaemia is common in the setting of acute MI, with or without a history of diabetes.
  - Also look for **hypoglycaemia** in critically ill patients.
- Serum lipids
  - Not useful in the acute period of STEMI management but will **inform assessment of the patient's risk factor profile** for recurrent cardiovascular events, and aid in setting targets for lipid-lowering therapy.
- C-reactive protein (CRP)
  - May be raised as a direct result of the acute-phase response to acute MI but may also point to a **concomitant infection**.
  - Not useful in the acute period of STEMI management but an elevated level may inform assessment of the patient's continued risk for recurrent cardiovascular events.[128]
  - In a secondary analysis of the VISTA-16 trial, elevated levels of high-sensitivity CRP during the index admission and the subsequent 16 weeks after an acute coronary syndrome were associated with a higher risk of the combined end point of cardiovascular death, myocardial infarction, non-fatal stroke, unstable angina, and all-cause death.[128]

Check **arterial blood gas only if there is** severe dyspnoea, hypoxia, and/or clinical evidence of pulmonary oedema or cardiogenic shock, and in survivors of cardiac arrest.

- Patients may require **airway stabilisation** and **aggressive oxygen supplementation** before proceeding to primary percutaneous coronary intervention.
  - This should not be performed routinely and **must not delay coronary reperfusion therapy** if there is no current or impending objective respiratory compromise.

- Taking an arterial blood gas may cause trauma to the radial artery, which is typically the access point for coronary angiography.

- Survivors of cardiac arrest may have:
  - Low PaO_2
  - High $PaCO_2$
    - $PaCO_2$ is an independent predictor of achieving sustained return of spontaneous circulation after cardiac arrest[129]
  - Raised lactate
  - Severe metabolic acidosis.

- Cardiogenic shock
  - Elevated serum lactate (>2 mmol/L [18.2 mg/dL]) is an indicator of shock.[79] [80]
  - Rising lactate levels during resuscitation indicate an extremely poor prognosis.[79]
  - Metabolic acidosis can be an indicator of shock.
  - Base deficit increase correlates with the occurrence and severity of shock.

- Signs of acute pulmonary oedema:
  - Respiratory acidosis
  - Respiratory alkalosis (in the early stages)
  - Metabolic acidosis
  - Reduced PaO_2
  - Reduced oxygen saturations
    - Acidosis is a significant predictor of mortality in acute heart failure patients.[130]

Imaging

Do not delay coronary reperfusion treatment to undertake imaging investigations.

- Start treatment and assess the patient's eligibility for coronary reperfusion therapy immediately after a clinical diagnosis of STEMI has been made.

Chest radiographs

Use a chest x-ray to **exclude alternative causes** and to aid **indirect assessment of cardiac function.** [82]

- Widened mediastinum may indicate acute aortic dissection.
- Pulmonary oedema suggests impaired left ventricular systolic function.

- A large globular cardiac contour or significantly increased cardiothoracic ratio may suggest pericardial effusion.

Echocardiogram

Use a point-of care transthoracic echocardiogram to:[2]

- Look for **regional wall motion abnormalities of the left ventricle** in patients with an atypical presentation or equivocal ECG[120] [131]
- Assess the patient's eligibility for coronary reperfusion therapy in the event of a **delayed presentation**
    - In practice, however, the patient's clinical status and the presence of pathological Q waves in the ECG are usually enough to assess their eligibility for coronary reperfusion therapy if presentation is delayed
- Look for **mechanical complications** of acute MI (see *Acute MI complications – mechanical* below):[131]
    - Left ventricular function
    - Right ventricular function
    - Ventricular septal rupture
    - Left ventricular free wall rupture
    - Acute mitral regurgitation
    - Pericardial effusion
    - Cardiac tamponade.

An echocardiogram can also be used to:

- Exclude STEMI in patients who present with **global saddle-shaped ST-segment elevation** (as seen with acute pericarditis)[131]
- Confirm the diagnosis of **takotsubo** cardiomyopathy (usually after normal coronary arteries are found on a STEMI angiogram)[1] [131]
- Suggest **alternative aetiologies** associated with chest pain (e.g., acute aortic disease, pulmonary embolism).[2] [131]

A pre-discharge echocardiogram is indicated for **all patients post-acute MI** to assess left ventricular function after coronary reperfusion therapy **and to guide prognostication**.[71] [75]

Emerging investigations

Cardiac myosin-binding protein C (cMyC)

CMyC **may perform better** than cardiac troponin T or I in patients who present early after symptom onset.[132]

- CMyC is a cardiac-restricted protein that is released more rapidly than cardiac troponin after acute MI.
- CMyC is also more abundant than cardiac troponins.
- It may become the gold standard test for the early diagnosis of acute MI, though is not currently used in clinical practice.

Early risk stratification

Risk stratification scores for STEMI are of **limited use in the emergency department setting.**

- This is largely because the emphasis should be on **rapid triage** based largely on ECG diagnosis, and assessment of eligibility for immediate coronary reperfusion therapy.
- Nonetheless, risk scores can:
    - Provide evidence-based prognostic information
    - Be used to support decisions on the optimum reperfusion strategy.

The European Society of Cardiology recommends the **GRACE risk score for acute coronary syndrome.** [2] [133] [134] Note that the GRACE score is of less value in STEMI than in non-STEMI, where it used for triage and to guide timing of invasive intervention.

- This calculates the overall risk of death while in hospital and from hospital discharge to 6 months.
- The Thrombolysis In Myocardial Infarction (TIMI) Risk Score for STEMI is an alternative.[135] [136]

Acute MI complications

Left ventricular dysfunction [2] [71]

- Severity depends on the duration of ischaemia, premorbid functional state, and presence of concomitant mechanical complications of acute MI.
- Can be transient (myocardial stunning) or persistent.
- May be clinically silent or lead to symptoms and signs of heart failure.

Left ventricular aneurysm [71]

- Affects <5% of those with large transmural MIs (especially anterior STEMI)
- May present with signs of heart failure, ventricular arrhythmias, or clinical sequelae of thromboembolism.
- ECG changes can support clinical suspicion.
- Will require imaging to confirm the diagnosis.

Left ventricular thrombus [2] [71]

- Relatively frequent complication of large anterior STEMI.[137]
- No specific clinical signs.
- May present with signs of heart failure or sequelae of systemic thromboembolism.
- (Contrast) echocardiography required to confirm diagnosis.

Right ventricular infarction [71]

- May present with the triad of hypotension, elevated jugular venous pressure (JVP), and clear lung fields.[83]
- Can complicate up to one third of inferior STEMIs.
- Check for ST elevation in aVR, V1, and right precordial leads.
- Confirm the diagnosis with echocardiography.

Ventricular septal rupture [2] [71]

- Presents with acute heart failure or cardiogenic shock.
- Look for a loud systolic murmur.
- Rupture after anterior STEMI = apical ventricular septal defect (VSD).
- Rupture after inferior STEMI = basal VSD (worse prognosis).
- Resulting left-to-right shunt may give rise to signs of acute right heart failure.
- The diagnosis is confirmed by echocardiography and Doppler (to quantify the degree of shunting).

Left ventricular free wall rupture [2] [71]

- Presents with sudden-onset chest pain and/or haemodynamic collapse.
- Rupture leads to haemopericardium and ultimately tamponade.
- Followed by electromechanical dissociation and typically death.
- Mortality rates range from 20% to 95%.[138]
- More common after first MI, anterior STEMI, lack of reperfusion, or late fibrinolysis, and in elderly patients and women.
- The diagnosis is confirmed by echocardiography.

Acute mitral regurgitation (MR) [2] [71]

- Secondary to:
    - Papillary muscle rupture or
    - Functional/ischaemic' MR
        - Post-infarction left ventricular remodelling characterised by: papillary muscle displacement, leaflet tethering, and annular dilatation
- Look for signs of severe dyspnoea, acute pulmonary oedema, and/or cardiogenic shock.
- Classical pansystolic murmur of mitral regurgitation may not always be audible due to the severity of the regurgitation.
- Inferior STEMI can cause rupture of the posteromedial papillary muscle.
- Anterolateral STEMI can cause rupture of the anterolateral papillary muscle.
- The diagnosis is confirmed by echocardiography.

Pericarditis [2] [71]

- Diagnostic criteria:[2]
    - Pleuritic chest pain (relieved by sitting or leaning forward)
    - Pericardial friction rub
    - ECG changes: global ST-segment elevation or PR interval depression
    - Pericardial effusion (new or worsening).
- **Early post-MI pericarditis**: usually transient.
- **Late post-MI pericarditis**: also known as **Dressler syndrome**; more common after late-presentation STEMI.
- Both are related to late or failed reperfusion and larger infarct size.

Pericardial effusion [2] [71]

- Secondary to pericarditis.
- Can be a complication of primary percutaneous coronary intervention.
- In the absence of inflammatory signs: rule out **subacute left ventricular free wall rupture**.
- Look for signs of cardiac tamponade:
    - **Beck's triad** = hypotension with narrow pulse pressure + raised JVP + muffled heart sounds
    - **Pulsus paradoxus** = exaggerated fall in systolic pressure >10 mmHg during inspiration
    - **Electrical alternans** (and tachycardia) on ECG
    - Pleuritic chest pain
    - Tachypnoea
    - Weakness, anxiety, restlessness.
- Use echocardiography first-line if there is a high clinical suspicion of pericardial effusion and/or tamponade.

Bradycardia and atrioventricular block [71]

- Sinus bradycardia is common post-STEMI and is usually self-limiting.
- Mobitz type I second-degree atrioventricular (AV) block is associated with inferior STEMI.
    - Rarely causes haemodynamic compromise.
- Mobitz type II second-degree AV block and complete heart block:
    - May require pacing.

Practical tip

AV block secondary to inferior STEMI quite often resolves spontaneously or after coronary reperfusion.

- AV block secondary to anterior STEMI is associated with a **high mortality rate** and indicates an extensive area of myocardial damage. It will need consideration of permanent pacemaker implantation.
- Left bundle branch block or hemiblock suggests an extensive anterior MI.

Supraventricular tachyarrhythmias [2] [71]

- Atrial fibrillation most common.
- Determine the patient's haemodynamic status and treat accordingly.

Ventricular tachyarrhythmias [2] [71]

- Secondary to ischaemia: unstable, polymorphic, relatively fast ventricular tachycardia degenerating into ventricular fibrillation.
- Secondary to reperfusion (within 48 hours): ventricular extrasystoles, non-sustained ventricular tachycardia.
    - Usually benign if self-limiting.

- **Be aware of incessant ventricular tachycardia/electrical storm despite appropriate revascularisation.**
- It is crucial to identify and treat sustained ventricular arrhythmias in the peri-infarction period to prevent:
    - Further reduction in cardiac output
    - Worsening myocardial ischaemia
    - Degeneration into ventricular fibrillation

History and exam

Key diagnostic factors

chest pain (common)

Patients typically present with **central chest pain**:[1] [2] [82]

- Classically **retrosternal, crushing, heavy, severe**, and **diffuse** in nature
- Might be described by the patient as '**pressing or squeezing**'
- May occur **at rest or on activity**
- May be constant or intermittent, or wax and wane in intensity
- **Sometimes radiating to the left arm, neck, or jaw**
- May be associated with **nausea, vomiting, dyspnoea, diaphoresis, lightheadedness, palpitations, or syncope**.[82]

Always ask about the **characteristics of the chest pain** as part of your history; in particular:[82]

- "Have you ever had this type of pain before?"
- Nature, severity, duration of pain
- Radiation
- Associated symptoms
- **Time since symptom onset** – this is crucial to inform the appropriate reperfusion strategy[2] [74] [75]
    - If symptoms are intermittent, it is important to ask when the last episode of pain occurred.[82]

Practical tip

The choice of coronary reperfusion strategy depends on **time since symptom onset**– but obtaining an exact time for this can be difficult.

- Patients can often give only an approximate idea of when their symptoms began.
- Patients sometimes ignore chest pain (or associated symptoms) until they can no longer tolerate it.
- The reliability of the assessment of time since symptom onset is determined by a combination of the patient's ability to give an accurate history and the experience and skill of the clinician taking the history.
- If you question the patient carefully, they may describe warning signs, or less severe or less long-lasting symptom episodes preceding the more severe episode that has prompted them to seek medical help.

Practical tip

Do not rely on a positive patient response to glyceryl trinitrate as a reliable diagnostic indicator of ischaemic chest pain.[2] [82] [96]

- Response to nitrates can be misleading. Patients who get symptom relief still need confirmatory ECG testing to inform the diagnosis.
- Complete normalisation of ST-segment elevation along with resolution of chest pain after buccal or sublingual nitrates suggests coronary vasospasm (with or without associated MI).[2]

dyspnoea (common)

Dyspnoea is a common feature **secondary to pulmonary congestion** from left ventricular systolic dysfunction.[2]

It can also occur due to other mechanical and electrical complications of acute MI, which occur less commonly in the context of contemporary rapid revascularisation, for example:

- Left ventricular aneurysm
- Ventricular septal rupture
- Left ventricular free wall rupture
- Acute mitral regurgitation
    - Papillary muscle rupture
    - Functional (ischaemic) mitral regurgitation
- Pericardial effusion
- Cardiac tamponade
- Supraventricular tachyarrhythmias
- Ventricular tachyarrhythmias
- Bradycardia and atrioventricular block.

pallor (common)

Pallor is a common feature due to high sympathetic output resulting in **peripheral vasoconstriction**.

diaphoresis (common)

Marked sweating is a common feature due to **high sympathetic output.** [2] [139]

cardiac risk factors (common)

Check for any history of cardiovascular disease: in particular, **ischaemic heart disease.** [82]

- Also check for any **previous episodes** of investigation or treatment for **chest pain**.
- A history of coronary artery disease should increase your index of suspicion.[2]

A cardiovascular risk factor profile is an important part of your history-taking. Check:[82]

- Smoking status
- Hypertension
- Diabetes mellitus
- Hypercholesterolaemia
- Family history of premature coronary artery disease (<60 years)
- Established coronary artery disease
- Advanced age
- Obesity
- Metabolic syndrome
- Physical inactivity
- Chronic kidney disease
- Cocaine use.

abnormal breath sounds (uncommon)

Auscultate the heart and lungs.

- Crackles/crepitations or cardiac wheeze would suggest congestive cardiac failure ± pulmonary oedema.

additional heart sounds (uncommon)

Auscultate the heart and lungs.

- **Muffled heart sounds** could suggest a pericardial effusion or even cardiac tamponade.
- Is there a **third (S3) or fourth (S4) heart sound**?
    - These added heart sounds could suggest severe heart failure.
- A **murmur** might suggest:
    - Acute ventricular septal defect
    - Acute mitral regurgitation

- Underlying chronic valvular heart disease.

cardiogenic shock (uncommon)

Cardiogenic shock complicates 5% to 10% of STEMI admissions.[80] [99][100]

- In-hospital mortality remains high (≥50%).[80] [100]
- There is a bimodal presentation: the majority occur within 24 hours; the remainder occur within the first week.[100] [101]

Seek immediate senior support and specialist input if your clinical assessment suggests **cardiogenic shock**.

See Shock .

Patients present with signs of hypoperfusion and/or fulminant heart failure, such as:[80]

- Altered mental status/reduced consciousness
- Tachypnoea
- Severe dyspnoea
- Tachycardia
- Orthopnoea
- Cool peripheries
- Grey, ashen, pale appearance.

Cardiogenic shock is primarily a **clinical diagnosis** supported by **haemodynamic measures**. It is defined as **persistent hypotension** (systolic blood pressure [SBP] <90 mmHg) together with signs of **end-organ hypoperfusion.** [78] [79] [80] [140]

- Clinical criteria:[78] [80]
  - SBP <90 mmHg despite adequate volume replacement, or if inotropes and/or mechanical circulatory support are needed to maintain SBP ≥90 mmHg
  - Urine output <30 mL/hour
  - Cool extremities
  - Elevated lactate.
- Haemodynamic criteria:[78] [80]
  - Cardiac index ≤2.2 L/minute/m^2
  - Wedge pressure ≥15 mmHg.
- Cardiogenic shock **results from extensive left ventricular infarction and/or mechanical complications** such as:
  - Papillary muscle rupture
  - Ventricular septal rupture
  - Left ventricular free wall rupture leading to pericardial tamponade

- Right ventricular infarction.

Other diagnostic factors

nausea and/or vomiting (common)

Nausea and vomiting are common features.[2] [82]

- These are non-specific symptoms but are commonly associated with inferior-wall STEMI due to increased vagal tone.
- May be the only indicator of inferior-wall STEMI.

dizziness or light-headedness (common)

Patients commonly report feeling lightheaded or weak/lethargic.[2]

- This is due to **cerebral hypoperfusion** as a result of hypotension and/or symptomatic bradycardia.

distress and anxiety (common)

The patient may report an **impending sense of doom or death.**

palpitations (common)

Some patients present with palpitations.[2] [94]

- Tachycardia
  - Supraventricular tachyarrhythmias such as atrial fibrillation
  - Ventricular tachyarrhythmias such as ventricular tachycardia
- Bradycardia
  - Sinus bradycardia
  - Atrioventricular block secondary to inferior STEMI
  - Atrioventricular block secondary to anterior STEMI
- Irregular heart beat
  - Supraventricular tachyarrhythmias such as atrial fibrillation
  - Ventricular extrasystoles

reduced consciousness (uncommon)

Changes in mental status/reduced consciousness are associated with **cardiogenic shock or bradycardia and hypotension.** [79] [80] [100]

hypotension (uncommon)

Hypotension may be present in:

- **Cardiogenic shock** – systolic blood pressure (SBP) <90 mmHg despite adequate volume replacement, or if inotropes and/or mechanical circulatory support are needed to maintain SBP ≥90 mmHg[78] [79] [80]
- **Inferior STEMI**
- **Right ventricular infarction**
    - Complicating inferior STEMI or an extensive anterior STEMI
    - Always think of the triad of hypotension, elevated jugular venous pressure, and clear lung fields[83]
- Cardiac **tamponade**
- Haemodynamically significant atrioventricular block, supraventricular tachyarrhythmias, or ventricular arrhythmias.[100]

non-chest pain presentation (chest pain-equivalent symptoms) (uncommon)

Be aware of patient groups who present **without chest pain as the predominant feature** (i.e., with **chest pain-equivalent symptoms**).[2] [82]

- **Women, older patients, and patients with diabetes** are more likely to present with atypical features.[2] [95]
- Patients might describe their chest symptoms as **burning, throbbing, tight**, or a feeling like **trapped wind**.
    - The patient may describe **indigestion** rather than chest pain.
- In the absence of chest pain, there may be **epigastric pain, back (interscapular) pain, neck or jaw pain, or arm pain** (typically left-sided).
- Clinical suspicion is key to making the diagnosis. It is, therefore, vital to make a full assessment based on the history, examination, and serial ECGs.[1] [92]

Investigations

1st test to order

Test	Result
ECG Perform a 12-lead ECG **within 10 minutes of first medical contact** in any patient who presents with chest pain and/or other signs of possible STEMI.[1] [2] • If the patient presents in the community, obtain a **pre-hospital ECG** and send it digitally to the receiving hospital as quickly as possible.[2] [82] • If the **ECG is equivocal** despite a high clinical suspicion of acute MI, perform **serial ECGs** in the appropriate hospital setting and compare these with historical ECGs, if available.[1] [2] [82] In the appropriate clinical context (chest pain or other symptoms of ischaemia), make a **clinical working diagnosis of STEMI** if there is **new (or increased) and persistent ST-segment elevation** in two or more contiguous leads.[2] [71] • Make the diagnosis when these criteria are met in the **absence of left ventricular hypertrophy, left bundle branch block (LBBB), or a paced rhythm on the ECG**. • Severe cases of left ventricular hypertrophy can appear identical to LBBB. • A paced rhythm can appear identical to LBBB. • Note that the **presence of left ventricular hypertrophy, LBBB, or a paced rhythm does not preclude a diagnosis of STEMI** if the patient presents with typical symptoms of myocardial ischaemia. • See *Less common ECG presentations* below for more on diagnosis of STEMI in the presence of LBBB. • Consult a **cardiology specialist immediately** if the ECG changes **are equivocal.** • An urgent transthoracic echocardiogram to look for regional wall motion abnormalities is indicated.[120] Measure ST-segment elevation from the J point.[1] [2] As soon as a clinical diagnosis of STEMI is made based on ECG changes together with symptoms or signs of ischaemia, start treatment and **make an immediate assessment of eligibility for coronary reperfusion therapy** (irrespective of age, ethnicity, sex, or level of consciousness).[2] [71] [75] • Alert the **interventional cardiology** team straight away.	a STEMI is diagnosed in the appropriate clinical context (a patient with chest pain or other symptoms consistent with myocardial ischaemia) when there is new (or increased) and persistent ST-segment elevation in at least two contiguous ECG leads of ≥1 mm in all leads other than leads V2-V3 where the following cut-off points apply:[1] • ≥2.5 mm in men <40 years old • ≥2 mm in men >40 years old • ≥1.5 mm in women regardless of age

Test	Result

- **Do not wait for cardiac biomarker results** or other laboratory or imaging investigations. Assessment and diagnosis of STEMI is a **time-critical process**.
    - The **shortest possible delay from symptom onset to coronary reperfusion** maximises the patient's chances of survival and recovery.[1] [2] [71] [72] [73] [74] [75]

For a library of ECGs with learning points, see Diagnosis recommendations .

Less common ECG presentations

Posterior STEMI [1] [2]#

- Consider this when there is **ST-segment depression in leads V1-V3** along with characteristic signs and symptoms of myocardial ischaemia.
- **Confirm with posterior lead ECG**: ST-segment elevation ≥0.5 mm in V7-V9.

Right ventricular infarction [1] [83]##

- Can complicate an inferior STEMI.
- Check right precordial leads (V3R and V4R).
- Look for **ST elevation ≥1 mm in aVR and V1**.
- Confirmation will have an impact on choice of therapeutic intervention.

Left main coronary obstruction [2] [84]#

- Consider **complete left main coronary artery obstruction** if the following are both present, especially if the patient has haemodynamic compromise:
    - ST depression ≥1 mm in ≥6 surface leads (i.e., inferolateral ST depression)
    - ST elevation in aVR or lead V1.

STEMI in the presence of LBBB [86] [122]#

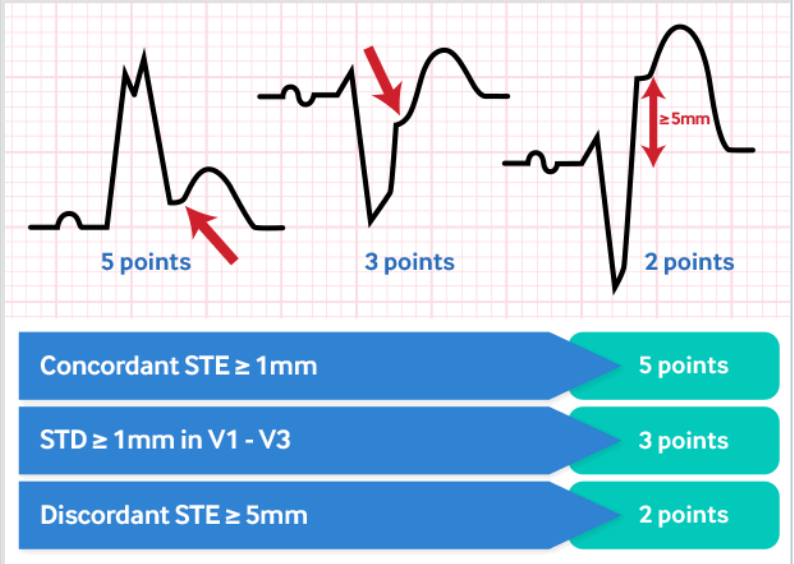
ECG diagnosis of STEMI is trickier in the presence of LBBB.[2]

- Bundle branch block (BBB) precludes accurate assessment, but it may be possible to make the diagnosis if **marked ST-segment abnormalities** are present.[2]

Test	Result
• The presence of **concordant ST-segment elevation (i.e., in leads with positive QRS deflections)** is one of the best indicators of total coronary occlusion and ongoing MI in the context of a patient with concomitant LBBB.[85] • Manage any patient with BBB and clinical suspicion of ongoing myocardial ischaemia **as per the standard STEMI protocol**. This applies to both left and right regardless of whether or not the BBB was previously known.[2] • Presumed new LBBB alone does not indicate the presence of a STEMI.[87] [88] • If in doubt, **seek immediate input from the cardiology team**. **More info: Sgarbossa criteria for diagnosis of MI in the presence of LBBB** ***Consider using the Sgarbossa criteria to improve the diagnostic accuracy for STEMI in patients who have LBBB at presentation.*** **Original criteria** [1] [86] • ST elevation ≥1 mm, concordant with the QRS complex: **5 points.** • ST depression ≥1 mm in leads V1, V2, or V3: **3 points**. • This has a sensitivity of 19% and a specificity of 81% to diagnose acute MI.[123] • ST elevation ≥5 mm, discordant with the QRS complex: **2 points**. • This has a sensitivity of 10% and a specificity of 99% to diagnose acute MI.[123] • An aggregated **score of 3+ is 90% specific for MI but only 36% sensitive**. **Components of the Sgarbossa criteria have high specificity but low sensitivity so are useful to confirm acute MI but less useful to rule it out.** [124] • The low sensitivity means you must **maintain a high index of suspicion if the presentation is consistent with MI regardless of the criteria score**. • 'Weighted' Sgarbossa criteria rely on the points system; however, only two of the criteria carry a score ≥3 to make the diagnosis of acute MI.[125] • 'Unweighted' Sgarbossa criteria are applied without the points system – this is more sensitive but less specific.[125] • The criteria were originally based on the outcome of acute MI as measured by creatine kinase-MB rather than angiographic evidence of acute coronary occlusion – further	

Test	Result

reducing sensitivity because the criteria encompass both STEMI and non-ST-elevation MI (NSTEMI).[125]

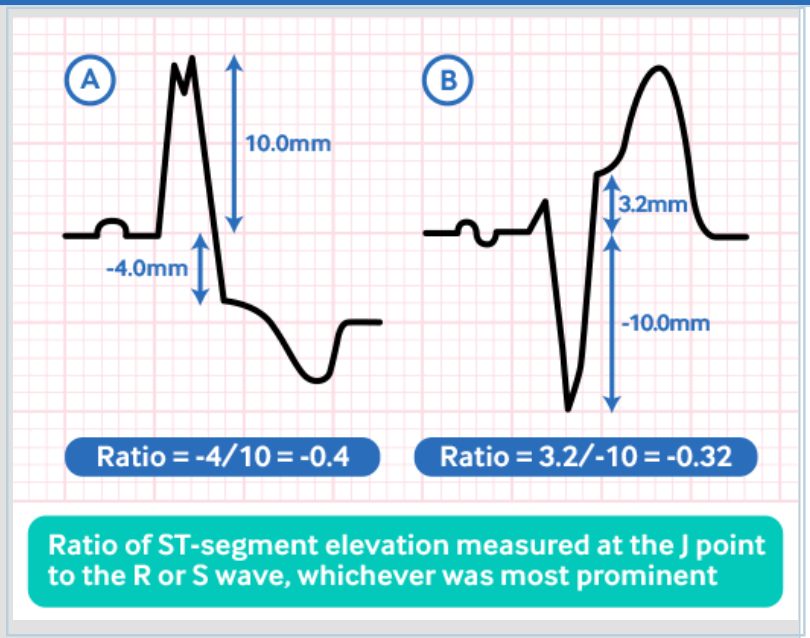


Sgarbossa criteria for MI in the presence of LBBB
Created by the BMJ Knowledge#Centre

The modified Sgarbossa criteria have better sensitivity but worse specificity for STEMI. [125] [126]

- The original rule for >5 mm discordance is replaced with a proportionately excessive discordance: ST-elevation/S-wave amplitude ≤-0.25.
- The modified criteria were found to be more sensitive versus the 'weighted' (80% vs. 49%; P <0.001) and 'unweighted' (80% vs. 56%; P <0.001) Sgarbossa criteria.[125]
    - Modified criteria specificity is not significantly different from the 'weighted' criteria.[125]
    - Modified criteria specificity is significantly greater than the 'unweighted' original criteria.[125]

Test	Result



ST/S ratio under the modified Sgarbossa criteria
Created by the BMJ Knowledge Centre

Previous silent/unrecognised STEMI

Be aware of the possibility that abnormal ECG features might be due to a **previous silent/unrecognised STEMI.** [1]

- Residual ST elevation on the ECG from an old STEMI may be detected either incidentally in an asymptomatic patient who is having an ECG for another reason, or occasionally in a patient with symptoms of ischaemia who is experiencing an NSTEMI against the background of a previous history of STEMI.
- In such cases, there may be ST-segment elevation on the current ECG that was **already present on old ECGs** (e.g., in the case of left ventricular aneurysm formation). It is **important to distinguish this from new ST elevation** as management will differ.
    - The historical STEMI may have been 'silent' and gone unrecognised at the time. The resulting old **ECG features will usually be fixed**.
- It is helpful to take a thorough history, together with diligent ECG interpretation and comparison with old ECGs (plus medical records) if available.
- Seek specialist input from the cardiology team.

The following criteria for previous (or silent/unrecognised) MI can be helpful. There may be:[1]

- **Pathological Q waves** with or without symptoms, in the absence of non-ischaemic causes[1]

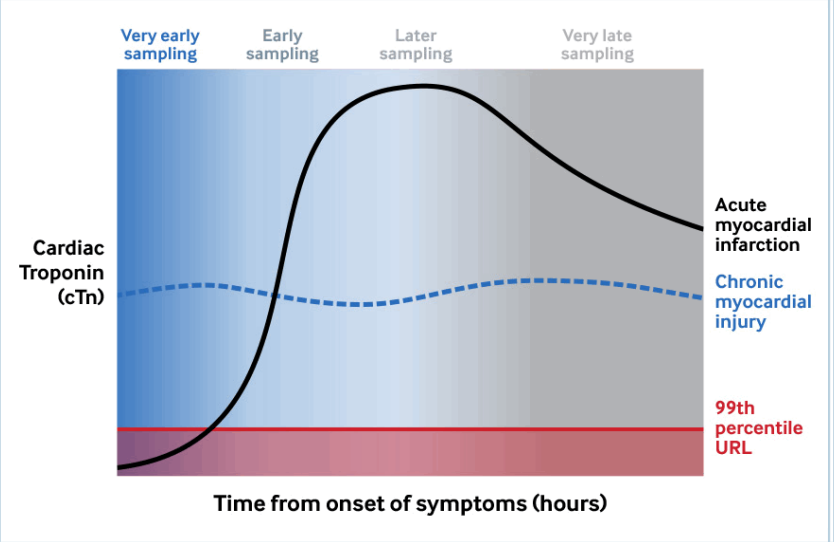
Test	Result

• **Loss of viable myocardium in a pattern consistent with a coronary artery territory** (regional wall abnormality) seen **on cardiac imaging**:
  • Echocardiography
  • Myocardial perfusion scintigraphy (MPS)
  • Positron emission tomography (PET)
  • Cardiac magnetic resonance imaging
• **Pathological findings of a healed or healing MI** on cardiac imaging.
Practical tip
A Q wave is defined as any negative deflection preceding an R wave in the QRS complex.
• A Q wave represents the normal left-to-right depolarisation across the interventricular septum.
• Small Q waves tend to be normal in most leads (e.g., left-sided leads such as I, aVL, V5, and V6).
• More pronounced Q waves (>2 mm deep) may be seen in lead III or aVR as a normal variant.
• Q waves are not usually seen in the right-sided leads (V1-V3).
• Pathological Q waves can be a sign of previous MI. The precise definition of pathological Q waves has been debated. The 2018 Fourth Universal Definition of MI defines them as follows:[1]
  • Any Q wave in leads V2-V3 >20 milliseconds (0.02 seconds) or any QS complex in leads V2 and V3
  • Q wave ≥30 milliseconds (0.03 seconds) and ≥1 mm deep or QS complex in leads I, II, aVL, aVF, or V4-V6 in any two leads of a contiguous lead grouping (I, aVL; V1-V6; II, III, aVF)
  • R wave >40 milliseconds (0.04 seconds) in V1-V2 and R/S >1 with a concordant positive T wave in the absence of a conduction defect.	

coronary angiography After making a clinical diagnosis of STEMI, offer coronary angiography, with follow-on primary percutaneous coronary intervention (PCI) if indicated, as the preferred coronary reperfusion strategy, if:[75] • The patient presents **within 12 hours of onset** of symptoms, **and** • Primary PCI can be delivered **within 120 minutes** of the ECG-based diagnosis. When primary PCI is **not an**	acute occlusion or critical stenosis

Test	Result
immediate option and cannot be provided within 120 minutes of ECG diagnosis, **fibrinolysis should be initiated** expeditiously as part of a pharmaco-invasive strategy, provided the patient has presented **within 12 hours of symptom onset**.[75] **Seek immediate specialist advice** from cardiology to discuss management options for any STEMI patient **who presents >12 hours after symptom onset**. Coronary angiography ± primary PCI should be considered if there is evidence of continuing myocardial ischaemia, haemodynamic instability or cardiogenic shock, or life-threatening arrhythmias.[2] If a patient with acute STEMI has had fibrinolysis, offer **angiography**:[75] • Immediately if an ECG 60-90 minutes after fibrinolysis shows residual ST-segment elevation (rescue PCI) • After seeking specialist cardiology advice if the patient has recurrent myocardial ischaemia after fibrinolysis • During the same hospital admission if the patient is stable after successful fibrinolysis (routine PCI strategy). Radial arterial access is preferred to femoral access in all patients undergoing coronary angiography.[75]	
cardiac troponin Request a **baseline high-sensitivity cardiac troponin (cTn)** along with your set of routine blood work whenever a patient presents with a possible acute MI. A pathological rise in troponin level followed by a later fall provides **definitive confirmation of an acute MI** in a patient who has clinical/ECG evidence of ongoing myocardial ischaemia (although STEMI can usually be diagnosed by ECG alone).[1] [89] • However, **do not delay coronary reperfusion to wait for troponin results**. • Start treatment and immediately assess eligibility for coronary reperfusion therapy as soon as a clinical diagnosis of STEMI is made based on ECG signs in a patient with chest pain or other symptoms consistent with myocardial ischaemia.[1] [82] **Troponin I and T** are the **preferred biomarkers** for definitive confirmation of an MI, with high-sensitivity assays preferred to standard ones.[1] [82] • Cardiac troponins are biological **markers of cardiac muscle death** (cardiomyocyte necrosis) that are released into the circulation when damage to cardiac muscle has occurred.[1] [89] • Creatine kinase-MB fraction is less sensitive and less specific and is now rarely used or measured.[1]	troponin elevated • acute MI is definitively confirmed by a rise and/or fall in cardiac troponin (with **at least one value >99th percentile of the upper reference limit**) in a patient who has symptoms or signs of ischaemia[1] [82]

Test	Result
Troponin level deviations and normal cut-offs are assay-specific so **check local protocols.** • Note there is **significant variability in**:[1] • The **time to peak value** – levels usually begin to rise around **2-3 hours after onset** of myocardial ischaemia but this varies according to the underlying mechanism • The time when a normal value may become greater than the 99th percentile of the upper range limit[127] • The 99th percentile threshold is designated as the decision level for the presence of myocardial injury • It **varies between men and women** and is established separately for each specific assay[1] [82] • The time window to observe a fluctuating pattern of values. • Because assays are not standardised, **values from one assay cannot be compared with those from a different assay**.[1] Always interpret troponin values in the **context of**:[1] [82] • Current and previous **ECGs** • Signs and symptoms reported by the patient (in particular, the **time since symptom onset**) • The possibility of an **alternative cause for an elevated troponin**. This may be cardiac (e.g., myocarditis, aortic dissection, severe heart failure) or non-cardiac (e.g., pulmonary embolism, impaired renal function, underlying sepsis)[82] • The demonstration of a **rising and/or falling pattern** is important to distinguish acute myocardial injury from a chronically elevated cTn[1] • Historical troponin levels recorded for the individual patient (e.g., measured during previous admissions) • Some patients may have **a higher baseline** compared with the general population.[82]	

Test	Result
 *Cardiac troponin kinetics after acute myocardial injury including acute MI* Created by the BMJ Knowledge Centre	
glucose Look for **uncontrolled hyperglycaemia** in all patients (not just those with diabetes).[2] • Hyperglycaemia is common in the setting of acute MI, with or without a history of diabetes. • Also check for **hypoglycaemia** in critically ill patients. **Do not delay coronary reperfusion treatment to wait for blood results.** [2]	normal or elevated plasma glucose
full blood count Look for **anaemia**, which may influence the duration of dual antiplatelet therapy prescribed. **Raised inflammatory** markers may be a direct result of the acute-phase response to acute MI or may point to a concomitant infection. **Do not delay coronary reperfusion treatment to wait for blood results.** [2]	normal range but can be elevated or reduced
electrolytes, urea, creatinine, and estimated glomerular filtration rate (eGFR) Potassium, calcium, and magnesium homeostasis is crucially important to prevent both bradyarrhythmias and tachyarrhythmias during the peri-infarct interval. **Baseline renal function** at the time of hospital admission will provide a benchmark to allow for subsequent up-titration of medications and allow for the identification of contrast-induced nephropathy after primary angioplasty (if the patient is eligible for coronary reperfusion therapy). • Note patients on potentially nephrotoxic drugs on admission, especially if they are eligible for primary angioplasty.	normal range but can be elevated or reduced

Test	Result
• Reduce the risk of **contrast-induced nephropathy** if an invasive strategy is planned in a patient with chronic kidney disease.[2] • Also note that chronic kidney disease is a cardiac risk factor.	
C-reactive protein (CRP) May be raised as a direct result of the acute-phase response to acute MI but may also point to a **concomitant infection.** • Not useful in the acute period of STEMI management but an elevated level may inform assessment of the patient's continued risk for recurrent cardiovascular events.[128] • In a secondary analysis of the VISTA-16 trial, elevated levels of high-sensitivity CRP during the index admission and the subsequent 16 weeks after an acute coronary syndrome were associated with a higher risk of the combined end point of cardiovascular death, myocardial infarction, non-fatal stroke, unstable angina, and all-cause death.[128]	normal range or elevated
serum lipids Not useful in the acute period of STEMI management but will **inform assessment of the patient's risk factor profile** for recurrent cardiovascular events, and aid in setting targets for lipid-lowering therapy. Cholesterol levels may be lowered by high catecholamine levels mediated by an acute MI in its early phases. • Serum lipids should, therefore, be repeated in 30-60 days. • Irrespective of serum lipid levels, high-intensity statins are mandated after STEMI.	normal range or elevated

Other tests to consider

Test	Result
arterial blood gas **Patients may be hypoxaemic and require supplemental oxygen.** • Check arterial blood gases **only when there is** severe dyspnoea, hypoxia, and/or clinical evidence of pulmonary oedema or cardiogenic shock, and in survivors of cardiac arrest. • Patients may require airway stabilisation and oxygen supplementation before proceeding to primary percutaneous coronary intervention. • This should not be performed routinely and must not delay coronary reperfusion therapy if there is no current or impending objective respiratory compromise. ◦ Taking an arterial blood gas may cause trauma to the radial artery, which is typically the access point for coronary angiography.	oxygen is indicated when:[2] • arterial oxygen saturation (SaO_2) <90% or • PaO_2 <7.9 kPa (<60 mmHg)
chest x-ray Use a chest x-ray to **exclude alternative causes** and to aid **indirect assessment of cardiac function.** [82] • Widened mediastinum may indicate acute aortic dissection. • Pulmonary oedema suggests impaired left ventricular systolic function. • A large globular cardiac contour or significantly increased cardiothoracic ratio may suggest pericardial effusion. **Imaging tests must not delay coronary reperfusion therapy.**	possible findings: • pulmonary oedema • widened mediastinum • cardiomegaly • permanent pacemaker • biventricular pacemaker • sternal wires • clear lung fields • normal cardiac contour
point-of-care transthoracic echocardiogram **Use a point-of-care transthoracic echocardiogram to**:[1] [2] • Look for **regional wall motion abnormalities of the left ventricle** in patients with an atypical presentation or equivocal ECG[120] [131] • Assess the patient's eligibility for coronary reperfusion therapy in the event of a **delayed presentation** ◦ In practice, however, the patient's clinical status and the presence of pathological Q waves in the ECG are usually enough to assess their eligibility for coronary reperfusion therapy if presentation is delayed	• left ventricular regional wall motion abnormalities • valvular defects • right ventricular function • pericardial effusion • left ventricular mural thrombus

Test	Result

• Look for **mechanical complications** of acute MI:[131]
  • Left ventricular function
  • Right ventricular function
  • Ventricular septal rupture
  • Left ventricular free wall rupture
  • Acute mitral regurgitation
  • Pericardial effusion
  • Cardiac tamponade.
 	
An echocardiogram can also be used to:
 	
• Exclude STEMI in patients who present with **global saddle-shaped ST-segment elevation** (as seen with acute pericarditis)[131]
• Confirm the diagnosis of **takotsubo** cardiomyopathy (usually after normal coronary arteries are found on a STEMI angiogram)[1] [131]
• Suggest **alternative aetiologies** associated with chest pain (e.g., acute aortic disease, pulmonary embolism).[2] [131]
 	
Imaging tests must not delay coronary reperfusion therapy.
 	
A pre-discharge echocardiogram is indicated for all patients post-acute MI to assess left ventricular function after coronary reperfusion therapy.[71] [75]	

Emerging tests

Test	Result
cardiac myosin-binding protein C (cMyC) CMyC **may perform better** than cardiac troponin T or I in patients who present early after symptom onset.[132] • CMyC is a cardiac-restricted protein that is released more rapidly than cardiac troponin after acute MI. • CMyC is also more abundant than cardiac troponins. • It may become the gold standard test for the early diagnosis of acute MI, though it is not currently used in clinical practice.	elevated

Differentials

Condition	Differentiating signs / symptoms	Differentiating tests
Unstable angina	• Clinical presentation may not differentiate.	• ECG may show non-specific ST-segment and T-wave changes. • Cardiac biomarkers are normal.
Non-ST-elevation myocardial infarction	• Clinical presentation may not differentiate.	• ECG may show non-specific ST-segment and T-wave changes, but does not show ST-segment elevation. • Cardiac biomarkers are elevated in both non-ST-elevation MI and STEMI.
Aortic dissection	• Patients typically present with tearing chest pain, notably between the shoulder blades. • They can be in considerable distress and haemodynamically unstable. • Peripheral pulses may be unequal or absent distally.	• CXR may show a widened mediastinum. • ECG may be unremarkable, show sinus tachycardia, or show ST-segment changes if the dissection extends proximally and involves the coronary ostium. • A CT of chest and abdomen with intravenous contrast showing the presence of a dissection flap and a true lumen and false lumen is diagnostic for aortic dissection. • A trans-oesophageal echocardiogram may also show the dissection flap with the true and false lumens.
Pulmonary embolism (PE)	• Patients classically present with acute onset of sharp stabbing chest pain that is pleuritic in nature and associated with shortness of breath. • A background of increased clotting tendency, such as known hereditary thrombophilia or connective tissue disease; known deep venous thrombosis; or previous PE increases the likelihood of the diagnosis. • Other risk factors include recent prolonged immobilisation and limb trauma.	• Patients are hypoxic with an increased arterial-alveolar gradient on the arterial blood gas. • ECG may show sinus tachycardia or right ventricular strain with prominent S wave in lead I, prominent Q in lead III, and flipped T in lead III (S1Q3T3), or can be unremarkable. • D-dimer is useful for risk stratifications. In a patient with a low probability of PE on clinical scoring, with a non-elevated d-dimer, a PE can be excluded; if elevated,

Condition	Differentiating signs / symptoms	Differentiating tests
		further work-up is required to confirm PE. • For patients with a high probability of PE on clinical scoring (i.e., PE is likely) or an abnormal D-dimer, imaging is required. The multiple-detector CT pulmonary angiography scanning of the chest is the imaging study of choice.
Pneumothorax	• Patients present with sudden onset of pleuritic chest discomfort and shortness of breath. • Tachycardia, hypotension, and cyanosis suggest a tension pneumothorax. • Known underlying medical conditions that predispose to pneumothorax, such as chronic obstructive pulmonary disease, connective tissue disease, or recent chest trauma, may support this diagnosis.	• CXR shows a visceral pleural line.
Pneumonia	• Patients usually have an insidious onset of fevers, cough (that may be productive of sputum), and shortness of breath. • Chest discomfort may be pleuritic in nature. • Examination will usually confirm pneumonic consolidation with decreased resonance, decreased air entry, and crackles over the affected lung.	• WBC count is usually elevated with neutrophilia. • CXR shows increased alveolar markings. • Blood and sputum cultures may be positive for an infective organism.
Pericarditis	• Patients can present with chest pain of varying quality that is typically better on sitting up and leaning forwards and worse with lying down. • There may be a history of recent viral syndrome and a pericardial friction rub on clinical examination.	• ECG may have diffuse ST-segment elevation that is concave up ('saddle-shaped') with PR segment depression.[141] • Cardiac biomarkers can be elevated if inflammation extends into the myocardium. • Inflammatory markers such as CRP and erythrocyte sedimentation rate may be elevated.

Condition	Differentiating signs / symptoms	Differentiating tests
		• CXR demonstrating a globular cardiac shadow is suggestive. • Echocardiogram may show a pericardial effusion or may be unremarkable.
Myocarditis	• Patients often have a recent history of influenza-like illness or underlying autoimmune condition such as systemic lupus erythematosus. • They are likely to be young and often do not have risk factors for coronary artery disease. • Myocarditis is more likely to present with symptoms of cardiac failure than with chest pain.	• ECG changes and cardiac biomarkers can mimic MI. • Inflammatory markers (erythrocyte sedimentation rate and CRP) and autoimmune assays may be elevated. • Test of choice is cardiac magnetic resonance imaging, with delayed enhancement imaging showing an epicardial or mid-myocardial involvement.
Gastro-oesophageal reflux disease	• Patients present with burning retrosternal discomfort that is relieved by antacids. • Discomfort/pain is usually non-exertional.	• Diagnosis is usually clinical. • Cardiac biomarkers, ECG, and CXR are normal. • Oesophagogastroduodenoscopy is indicated for patients with persistent or atypical symptoms and may show oesophagitis (erosions, ulcerations, strictures) or Barrett's oesophagus.
Peptic ulcer disease	• Pain is described as burning epigastric pain that occurs hours after meals or with hunger. It often wakes the patient at night and is relieved by food and antacids. • There may be a previous history of reflux or medications that can cause peptic ulcer (i.e., recent use of steroid or non-steroidal anti-inflammatory drugs).	• Endoscopy may show ulcers, erosion, or gastropathy.
Oesophageal spasm	• Patients present with squeezing retrosternal discomfort that may be relieved by glyceryl trinitrate (due to relaxation of the spasm). • Discomfort/pain is usually non-exertional.	• Cardiac biomarkers, ECG, and CXR are normal. • Oesophageal manometry or barium swallow may show evidence of dysmotility.

Condition	Differentiating signs / symptoms	Differentiating tests
Costochondritis	• Musculoskeletal chest wall discomfort that is worse with certain movement and deep breaths. • Focal tenderness over the costochondral joints may be present.	• Cardiac biomarkers, ECG, and CXR are normal.
Anxiety or panic attack	• Normal examination; however, evidence of hyperventilation is sometimes present.	• Cardiac biomarkers, ECG, and CXR are all normal.
Oesophageal rupture (Boerhaave syndrome)	• Clinical presentation may not differentiate. • There may be a history of vomiting.	• CXR shows pneumomediastinum >90% of the time. Non-specific ECG changes, including ST elevation, but without rise in cardiac biomarkers.[142] [143]
Acute cholecystitis	• On physical examination there is constant right upper quadrant pain with or without Murphy's sign (inhibition of inspiration due to pain on palpation). There may be a history of gallstones or previous episodes of biliary colic.	• Ultrasound may show a distended gallbladder and gallstones.[144] If ECG changes accompany cholecystitis, these would most likely include ST elevation.
Brugada syndrome	• More common in Asian people and men aged 30-50 years. Patients typically present after an episode of polymorphic ventricular tachycardia or a cardiac arrest.	• ECG shows saddle-shaped ST elevation in leads V1-V3. These changes are associated with complete or incomplete right bundle-branch block and T-wave inversions.
Acute stress cardiomyopathy	• Clinical features are similar to non-STEMI (NSTEMI) and may include chest pain, shortness of breath, and left ventricular wall motion abnormalities. A characteristic feature is that often the clinical state is triggered by a severe extracardiac stressor (e.g., intracranial haemorrhage, pheochromocytoma, exogenous catecholamine administration, severe emotional stress, postoperative stress, sepsis).	• Often these patients present with ECG changes, cardiac biomarker elevations, and left ventricular dysfunction on cardiac imaging that are indistinguishable from NSTEMI but on coronary angiography will have no obstructive lesion. Coronary angiography remains the definitive test for diagnosis of this condition.

Criteria

ST elevation on ECG[1]

New (or increased) and **persistent** ST-segment elevation **in at least two contiguous leads of ≥1 mm in all leads, other than leads V2-V3 where the following cut points apply**:[1]

- ≥2.5 mm in men <40 years old
- ≥2 mm in men >40 years old
- ≥1.5 mm in women regardless of age.

Criteria for acute, evolving, or recent MI[3]

Either one of the following criteria:

1. Typical rise of biomarkers of myocardial necrosis (troponin or creatine kinase-MB) with at least one of the following:
    - Ischaemic symptoms
    - Development of pathological Q waves on ECG
    - ECG changes indicative of ischaemia (ST-segment elevation or depression)
    - Coronary artery intervention (e.g., coronary angiography).
2. Pathological findings of acute MI.

Criteria for established MI[3]

Any one of the following:

1. Development of pathological Q waves on serial ECGs. The patient may or may not remember previous symptoms. Biochemical markers of myocardial necrosis may have normalised, depending on the length of time that has passed since the infarct developed.
2. Pathological findings of a healed or healing MI.
3. Cardiac magnetic resonance imaging with delayed enhancement imaging showing a classic sub-endocardial or transmural infarct in a coronary artery distribution.

DIAGNOSIS

Screening

The NHS in England offers all people aged 40 to 74 years an NHS Health Check once every 5 years, which includes a formal assessment of cardiovascular risk. NHS England has estimated this could prevent 1600 myocardial infarctions per year, saving at least 650 lives.[145]

Recommendations

Urgent

Make a **working clinical diagnosis of STEMI** and **start treatment** if the patient has signs and **symptoms of myocardial ischaemia** plus **persistent/increasing ST elevation** in two or more contiguous leads on the ECG.[2]

Do not wait for cardiac troponin levels to confirm a STEMI.

Give all patients with suspected acute coronary syndrome **a single loading dose of aspirin as soon as possible**, unless they have aspirin hypersensitivity. [75]

- Check your local protocol or discuss the patient with a senior colleague if they have hypersensitivity to aspirin.

Perform all of the following **in tandem** as soon as a clinical diagnosis of STEMI has been made.[2] [71] [75]

- Immediately assess the patient's **eligibility for coronary reperfusion therapy** (irrespective of age, ethnicity, sex, or level of consciousness).[75]
- For most patients the best option will be **primary percutaneous coronary intervention (PCI); fibrinolysis** is reserved for those without access to timely primary PCI.[2] [75]
    - If eligible, take steps to ensure reperfusion is administered as **quickly as possible**.[2] [75]
    - If not eligible, offer conservative medical management.[75]
- Gain **intravenous access** and start **continuous haemodynamic monitoring and pulse oximetry**.[2]
    - Avoid placing a cannula that obstructs access to the right radial artery (the commonest entry site for primary PCI).
- Give **pain relief**: an intravenous opioid (e.g., morphine, diamorphine) is recommended plus a concomitant intravenous anti-emetic to prevent vomiting.[82]
- Give **oxygen therapy** only if oxygen **saturation is <90%**.[2]
- Give **dual antiplatelet therapy** by adding a $P2Y_{12}$ inhibitor to aspirin.[75]
    - If the patient is having primary PCI, use prasugrel if they are not already taking an oral anticoagulant, or clopidogrel if they are already taking oral anticoagulation.[75]
- **Consider an intravenous nitrate (e.g., glyceryl trinitrate, isosorbide dinitrate)** if the patient has:[2] [82]
    - Persistent **chest pain** despite administration of sublingual (or buccal) glyceryl trinitrate
    - Sustained **hypertension**
    - Clinical and/or radiographic evidence of **congestive heart failure**.
        - **Do not give an intravenous nitrate if there is**:

- Hypotension secondary to any one of: right ventricular infarction (usually complicating an inferior or extensive anterior STEMI); severe aortic stenosis or left ventricular outflow tract obstruction; pre-existing cardiomyopathy
- Persistent hypotension secondary to another cause
- Use of a phosphodiesterase-5 inhibitor (e.g., avanafil, sildenafil, tadalafil, vardenafil) for erectile dysfunction within the last 48 hours.

- **Seek immediate specialist input** from the **interventional cardiology team**.[2]
    - If you are managing the patient at a **non-PCI capable hospital**, contact the interventional cardiology team at your designated PCI-capable hospital to discuss **immediate transfer**.

Do not give anticoagulant therapy if the patient is likely to be eligible for primary PCI. [2] [71]

- Anticoagulation will be started by the interventional cardiology team in the catheterisation laboratory.
- If the patient is having **fibrinolysis**, start **anticoagulation** at the same time.[2] [71] [75] Select enoxaparin or unfractionated heparin (unless streptokinase is used for thrombolysis, in which case choose fondaparinux).[146]
- Continue anticoagulation until revascularisation (if fibrinolysis is followed by PCI) or for the duration of hospital stay up to a maximum of 8 days.[2] [71]
- The optimal time delay between successful fibrinolysis and PCI has not been clearly defined; however, a time window for PCI of 2-24 hours after successful fibrinolysis is recommended.

In a patient who has had a **return of spontaneous circulation (ROSC) after an out-of-hospital cardiac arrest**:[2] [147] [148] [149] [150] [151]

- **Primary PCI** is the treatment of choice if there is **ST-segment elevation on the post-ROSC ECG** or **life-threatening arrhythmia**
- If there is **no ST-segment elevation on the post-ROSC ECG** then:
    - **Exclude non-coronary causes** of cardiac arrest
    - Perform **urgent echocardiography**
    - Strongly consider a referral to cardiology for **urgent angiography** if there is a high index of **suspicion of ongoing myocardial ischaemia** despite no ST-segment elevation

- When deciding whether to take a survivor of cardiac arrest (with or without ST-segment elevation) to the catheterisation laboratory for urgent angiography ± PCI:[2]
    - Consider **each case** on its **individual merits** and seek **senior advice**
    - Take account of factors associated with the cardiac arrest that will influence the chance of a **good neurological outcome**.

If a patient with STEMI has **cardiogenic shock**, seek urgent **senior support** – coronary angiography ± PCI is indicated.[2] [75]

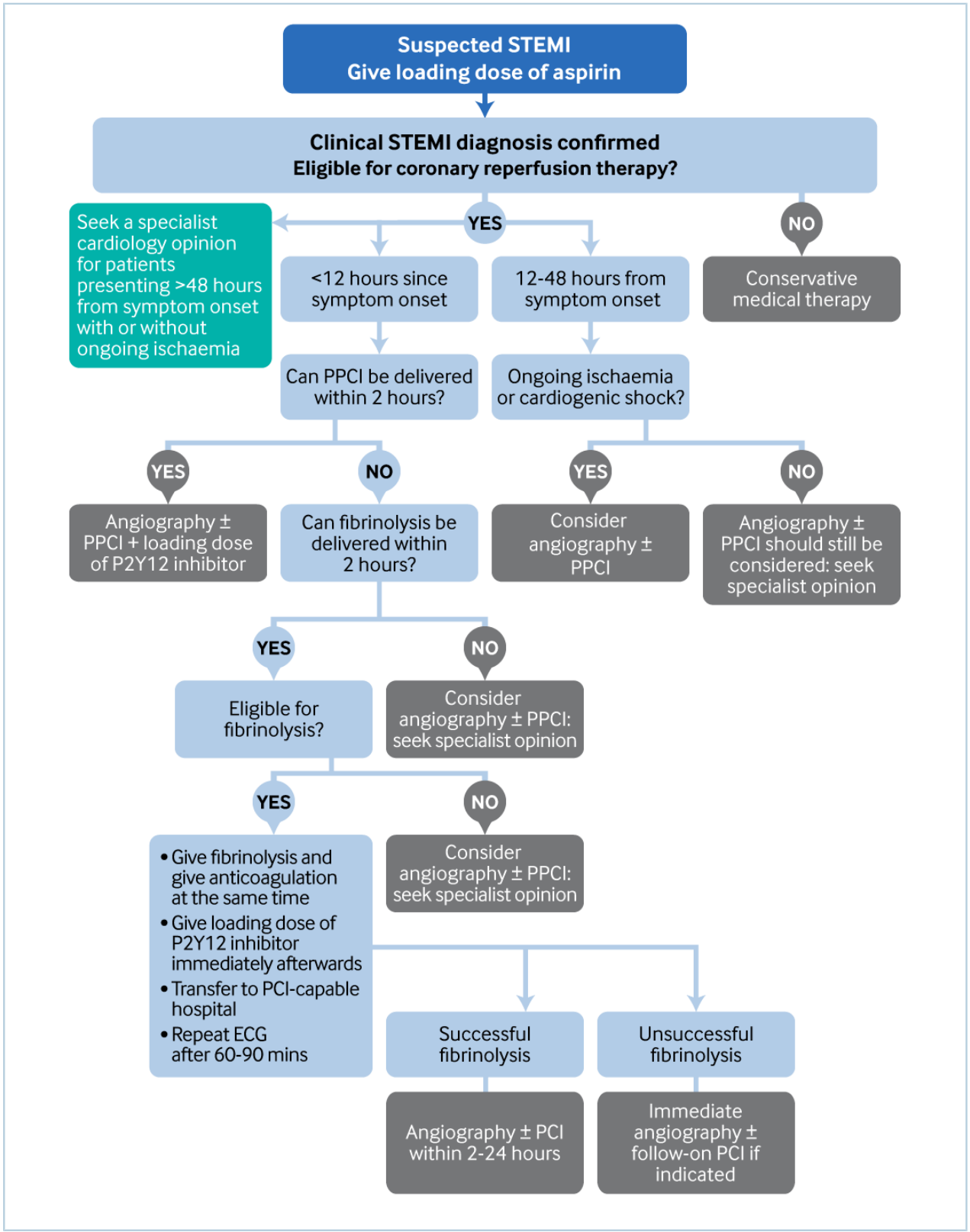
Key Recommendations

If there is a **high clinical suspicion of STEMI**or a clinical diagnosis of STEMI has been made, **seek immediate specialist input from cardiology**.

The established cornerstone of STEMI management is a **combined strategy** of:[2] [75]

- **Coronary reperfusion therapy** – primary PCI or fibrinolysis **plus**
- **Antithrombotic therapy** – with dual antiplatelet therapy and anticoagulation.
    - Dual antiplatelet therapy is started immediately and normally continued for at least 12 months.[2] [75]

Choice of coronary reperfusion therapy



Selection of the most appropriate reperfusion strategy. PPCI, primary percutaneous coronary intervention

Created by the BMJ Knowledge Centre

For most patients with STEMI, **primary PCI is the preferred reperfusion strategy** provided it can be delivered **within 120 minutes of the time when fibrinolysis could have been given**.[75] [81]

- Multiple trials have shown it has **better outcomes** (in terms of reduced mortality, reinfarction, or stroke) **and less intracranial bleeding than fibrinolysis** if administered in a **timely manner** by an experienced team.

Select primary PCI as the preferred reperfusion strategy in patients with acute STEMI, **if** both the following apply:[75] [81]

- **Symptom onset <12 hours** ago **AND**
- PCI can be delivered **as soon as possible and at the latest within 120 minutes of the time when fibrinolysis could have been given**.

Give fibrinolysis (unless contraindicated) if there is a lack of access to timely primary PCI.[2] [75] Give anticoagulation at the same time as giving fibrinolysis. Give dual antiplatelet therapy immediately afterwards.[75]

- The UK National Institute for Health and Care Excellence recommends a fibrin-specific drug such as **alteplase or tenecteplase**. It also recommends streptokinase (a non-fibrin-specific drug) as an option in some patients. Check local protocols for advice on choice of fibrinolytic drug.[152]
- Fibrinolysis can be administered as part of the **pre-hospital** management of STEMI, in which case an intravenous bolus dose of tenecteplase (or reteplase if available) is preferred as the other drugs are administered by intravenous infusion.[2] [152]
- If your patient is not at **a PCI-capable hospital, transfer them** to one immediately **after fibrinolysis** has been given.
- Assess the success of fibrinolysis with an ECG after 60-90 minutes.[75]
    - If it has failed, **offer immediate coronary angiography**, with follow-on PCI if indicated. **Do not repeat fibrinolytic therapy**.[2] [75]

If primary PCI **cannot be delivered within 120 minutes** of STEMI diagnosis, **seek a specialist cardiology opinion** to support your choice of reperfusion strategy.[2] [71] [153]

- The clinical efficacy of thrombolysis diminishes as the time from symptom onset increases.[72] [154]
- Therefore, the later the patient presents, the more consideration should be given to **transferring for primary PCI** instead, even if the delay is likely to exceed 120 minutes.[2] [74]

If your patient experiences **spontaneous resolution** of ST-segment elevation and complete symptom relief **after taking glyceryl trinitrate**:[2]

- Refer to cardiology
- Aim for early inpatient coronary angiography within 24 hours.

Seek immediate specialist advice from cardiology for any patient with a working diagnosis of **STEMI** who presents **>12 hours after symptom onset.**

- Coronary angiography ± primary PCI is recommended if there is:[2] [75]
    - Evidence of continuing myocardial ischaemia

- Haemodynamic instability or cardiogenic shock
- Life-threatening arrhythmias.

- A routine primary PCI strategy should be considered in STEMI patients presenting late (12-48 hours) after symptom onset (class IIa).[2]

If your patient has **ongoing ischaemic symptoms** suggestive of MI **BUT no ST-segment elevation** on the ECG, **primary PCI should be considered** if one or more of the following is present:[2]

- Cardiogenic shock or haemodynamic instability
- Acute heart failure presumed secondary to ongoing myocardial ischaemia
- Recurrent or refractory chest pain despite medical treatment
- Life-threatening arrhythmia
- Signs and symptoms suggestive of mechanical complications of acute MI
- Recurrent dynamic ST-segment or T-wave changes, especially intermittent ST-segment elevation.

Time targets for the management of STEMI

The benefits of reperfusion in reducing mortality and improving myocardial salvage **decline rapidly with time.** [2] [75]

- It is, therefore, vital to take every step possible to ensure the chosen reperfusion strategy (primary PCI or fibrinolysis) is delivered **as quickly as possible**.

The 2023 European Society of Cardiology acute coronary syndrome guideline sets widely accepted **maximum time-to-treatment targets as follows.** [2]

Interval	Target
First medical contact to ECG and STEMI diagnosis	**<10 minutes**
STEMI diagnosis to primary PCI	**<120 minutes** If this time target cannot be met, consider fibrinolysis
STEMI diagnosis to primary PCI if the patient's first medical contact is at a hospital	**<60 minutes** if the patient presents to or is in a PCI-capable hospital **<90 minutes** if the patient presents to or is in a non-PCI-capable hospital and needs transferring
STEMI diagnosis to administration of a bolus/infusion of a fibrinolytic drug (if primary PCI cannot be accessed within 120 minutes)	**<10 minutes**
Start of fibrinolysis to ECG assessment of its success or failure	**60-90 minutes**
If fibrinolysis is successful, time interval from starting fibrinolysis to early coronary angiography	**2-24 hours**

Long-term management of STEMI

Long-term management should include aspirin (plus a $P2Y_{12}$ inhibitor for up to 12 months as part of dual antiplatelet therapy), an ACE inhibitor, a beta-blocker, and a statin, as well as cardiac rehabilitation and modification of risk factors for cardiovascular disease.[63] [75] [155]

Full Recommendations

Treatment goals

The established cornerstone of STEMI management is a **combined strategy** of:[2] [75]

- **Coronary reperfusion therapy** – either primary percutaneous coronary intervention or fibrinolysis

AND

- **Antithrombotic therapy** – with dual antiplatelet therapy and anticoagulation.

The aim is to **restore myocardial blood flow** as quickly as possible and within guideline-mandated target times to:[2] [71] [73] [74]

- Achieve pain relief

- Limit myocardial damage/necrosis
- Reduce subsequent myocardial remodelling, which can have an adverse effect on predominantly left ventricular and overall function
- Minimise morbidity and mortality by preventing or limiting the effect of both electrical and mechanical complications of acute MI.

Initial treatment for all patients with suspected STEMI

Give all patients with suspected acute coronary syndrome **a single loading dose of aspirin as soon as possible**, unless they have aspirin hypersensitivity.[2] [75]

- Aspirin can be given orally (or via nasogastric tube if oral ingestion is not possible). An intravenous loading dose is used in some countries; however, this formulation is not available in the UK.

Check your local protocol or discuss the patient with a senior colleague if they have hypersensitivity to aspirin.

After the loading dose, continue with a lower maintenance dose of aspirin as part of ongoing dual antiplatelet therapy unless contraindicated.

Initial treatment for all patients with a clinical diagnosis of STEMI

Start treatment for STEMI **immediately** after making a clinical diagnosis – **do not wait for cardiac troponin levels** to confirm it.[2]

- Make a clinical working diagnosis of STEMI based on a combination of:
    - Signs and **symptoms** of myocardial ischaemia, plus
    - Persistent or increasing **ST elevation on the ECG**.

Perform all of the following **in tandem** as soon as a clinical diagnosis of STEMI has been made.[2] [71] [75]

- Immediately assess the patient's **eligibility for coronary reperfusion therapy.** [2] [75]
    - If eligible, take steps to ensure coronary reperfusion therapy is delivered as **quickly as possible** – see *Choice of coronary reperfusion therapy* below.
    - Do not allow the patient's age, ethnicity, or sex to influence your assessment of their suitability for reperfusion therapy.[75] Evidence suggests that women tend to receive reperfusion therapy less frequently and/or in a more delayed fashion than men, and are less likely to receive cardiac rehabilitations and secondary prevention medications.[2]
- Gain **intravenous access** and start continuous **haemodynamic monitoring and pulse oximetry.** [2]

Practical tip

When placing an intravenous cannula, avoid placing it in the right hand or right wrist area as the right radial artery is the usual route used to perform primary percutaneous coronary intervention.

- Give **pain relief.** [2] [82]
    - Titrated **intravenous opioids** (e.g., morphine, diamorphine) are the most commonly used option. Give an **intravenous anti-emetic** concomitantly to prevent vomiting.
    - Pain relief is important not just for the comfort of the patient but also because it may reduce myocardial and microvascular damage due to reduction of heart rate, cardiac workload, and oxygen consumption.[2]
- Consider an **intravenous nitrate (e.g., glyceryl trinitrate, isosorbide dinitrate).** [2]
    - Routine use of an intravenous nitrate in STEMI is not recommended as there is no evidence to support its benefits. However, an **intravenous nitrate may be useful** in patients who have:[2]
        - **Sustained hypertension**
        - Clinical and/or radiographic evidence of **congestive heart failure**
            - **Titrate the rate of infusion** according to the patient's blood pressure and wider clinical response
        - **Persistent chest pain (residual angina) despite administration of sublingual (or buccal) glyceryl trinitrate** (which the patient may have received before arriving at hospital).
    - **Do not give** an intravenous nitrate when there is **hypotension or right ventricular infarction**.
- Give oxygen therapy only if **saturations are <90%** on pulse oximetry.[2]
    - Routine supplemental oxygen is not indicated if arterial oxygen saturation (SaO_2) ≥90%.[156] [157] [158]

Evidence: Oxygen therapy in patients with acute MI

There is no evidence from randomised controlled trials (RCTs) to support the routine use of inhaled oxygen in people with acute MI without hypoxia, but guidelines vary on their specific recommendations.

There are different recommendations in guidelines on the thresholds for starting oxygen therapy. Guidelines also vary on recommended upper limits for oxygen saturation once oxygen has been started.

- The 2023 European Society of Cardiology guideline on the management of acute coronary syndromes recommends that routine oxygen is not given if the arterial oxygen saturation is ≥90%.[2] The evidence underpinning this recommendation includes the AVOID study, a Cochrane review, and the protocol for the DETO2X-AMI (all discussed below).[156] [157] [158] This guideline recommends oxygen therapy for hypoxic patients with an oxygen saturation <90% (based on limited evidence).
- One 2018 BMJ Rapid Recommendation also recommends that oxygen therapy is not initiated in patients with acute MI if the oxygen saturation is ≥90%.[159] This is based on the findings from a large systematic review and meta-analysis that liberal oxygen therapy was associated with higher mortality than conservative oxygen therapy in adults with acute illness (see below).[160]
- The UK National Institute for Health and Care Excellence guideline on chest pain of recent onset, last updated in 2016, recommends that supplemental oxygen is not routinely offered to patients with suspected acute coronary syndrome.[82] It recommends oxygen therapy if the patient has an oxygen saturation <94% and is not at risk of hypercapnic respiratory failure, aiming for a saturation of 94% to 98%. For patients with COPD who are at risk of hypercapnic respiratory failure, it recommends a target oxygen saturation of 88% to 92%, until blood gas analysis is available.[82]
- The 2016 Scottish Intercollegiate Guidelines Network (SIGN) guideline on acute coronary syndrome does not include a specific recommendation on the use of oxygen but does refer to a 2013 Cochrane review stating that it found no conclusive evidence to support the routine use of inhaled oxygen in patients with acute MI.[161][162]

There is a lack of evidence to support the routine use of oxygen in patients with acute MI when there is no hypoxia, although oxygen therapy has commonly been used as part of the initial management of patients with STEMI.

- One 2018 systematic review and meta-analysis including seven randomised trials and a total of 7702 patients with acute MI without hypoxaemia found that routine supplemental oxygen did not reduce:[163]
  - Mortality
  - Arrhythmias
  - Heart failure
  - Recurrent ischaemic events.
- The DETO2X-AMI multicentre RCT (published in 2017) compared supplemental oxygen therapy (6 L/minute) with ambient air in 6629 patients with suspected acute MI and an oxygen saturation

level of ≥90% on pulse oximetry and found no significant difference in death from any cause at 1 year (hazard ratio 0.97; 95% CI 0.79 to 1.21; P=0.80).[164]

- One 2016 Cochrane review including five RCTs compared oxygen with air in 1173 people within 24 hours of onset of suspected or proven acute MI (STEMI or non-STEMI). It found no difference in mortality, pain, or infarct size and the authors stated that they could not rule out a harmful effect.[158]
- One 2015 RCT of 441 patients with proven STEMI (the AVOID study, included in the Cochrane review above) found that patients receiving oxygen (at 8 L/minute) had similar mean peak troponin levels but significantly increased creatine kinase levels compared with those receiving air.[156] Patients randomised to receive air were given oxygen via nasal cannula (4 L/minute) or face mask (8 L/minute) if their oxygen saturation fell below 94%, to maintain a target level of 94%. Infarction size on cardiac magnetic resonance at 6 months was increased in patients with oxygen therapy compared with air.[156]

Regarding the upper limit for target oxygen saturation once oxygen has been started, evidence from a large systematic review and meta-analysis on oxygen use in acutely ill adults not at risk of hypercapnic respiratory failure (including those with MI) supports a 96% upper limit for target oxygen saturation.

- One large systematic review (published in 2018) of 25 RCTs and over 16,000 patients, including a meta-analysis, found that in adults with acute illness (including MI, but also including sepsis, critical illness, stroke, trauma, cardiac arrest, and emergency surgery), liberal oxygen therapy (broadly equivalent to a target saturation >96%) is associated with higher mortality than conservative oxygen therapy (broadly equivalent to a target saturation ≤96%).[160]
- In-hospital mortality was 11 per 1000 higher with liberal oxygen therapy versus conservative oxygen therapy (95% CI 2-22 per 1000 more). Mortality at 30 days was also higher with liberal oxygen (RR 1.14; 95% CI 1.01 to 1.29). Studies that were limited to people with chronic respiratory illness or psychiatric illness, on extracorporeal life support, receiving hyperbaric oxygen therapy, or having elective surgery were all excluded from the review.[160]

A lower target oxygen saturation of 88% to 92% is appropriate if the patient is at risk of hypercapnic respiratory failure. [82] [165]

- Give a **P2Y $_{12}$ inhibitor in addition to aspirin given in the initial phase**, as part of dual antiplatelet therapy.
  - Check your local protocol when deciding which P2Y $_{12}$ inhibitor to use and the timing of this.
  - The UK National Institute for Health and Care Excellence (NICE) recommends the following:[75]
    - If the patient is eligible for **primary percutaneous coronary intervention (PCI), start dual antiplatelet therapy during PCI**:
      - Prasugrel, in combination with aspirin, if the patient is not already taking an oral anticoagulant
        - For patients aged 75 years and over, the bleeding risk of using prasugrel needs to be weighed up against its effectiveness. If the bleeding risk from

prasugrel is a concern in these patients, ticagrelor or clopidogrel may be used as alternatives

- Clopidogrel, in combination with aspirin, if the patient is already taking an oral anticoagulant

- If the patient is having **fibrinolysis, start dual antiplatelet therapy immediately afterwards**:
  - Ticagrelor, in combination with aspirin, unless the patient has a high bleeding risk
  - Clopidogrel, in combination with aspirin, or aspirin alone, for patients with a high bleeding risk.

- **Seek immediate specialist input from the interventional cardiology team.** [2]
  - If you are managing the patient within a PCI-capable hospital, this team will be available on site. If not, call the interventional cardiology team at your designated PCI-capable hospital to discuss **immediate transfer** for coronary reperfusion therapy.[2]

Practical tip

Do not give anticoagulation therapy if the patient is likely to be eligible for primary PCI. [2] [71]
- This will be given by the interventional cardiology team in the cardiac catheterisation laboratory.

Practical tip

Administration of an intravenous nitrate should not delay transfer to the catheterisation laboratory.
- An intravenous nitrate can be given in the catheterisation laboratory by the interventional cardiology team, if needed.
- Recanalisation of the occluded infarct artery is the most effective way of relieving refractory chest pain, so the priority is to get the patient to primary PCI as quickly as possible.

Choice of coronary reperfusion therapy for different patient groups

For most patients with STEMI, **primary percutaneous coronary intervention (PCI) is the preferred reperfusion strategy.** [2] [75] [81]

- Multiple randomised controlled trials have shown that **primary PCI has better outcomes** (in terms of reducing mortality, reinfarction, or stroke) **and less intracranial bleeding than**

fibrinolysis provided it can be delivered in a timely manner by an experienced team.[166] [167] [168] [169] [170] [171]

- However, **fibrinolysis may be more appropriate when there is** a lack of access to timely PCI.[2] [75]

The most appropriate coronary reperfusion strategy will depend on:[2] [71] [74]

- Duration of ischaemic symptoms
- Persistence or resolution of ST-segment elevation on serial ECGs
- Availability of timely access to primary PCI
    - Whether a patient is first assessed in a PCI-capable or non-PCI-capable hospital
    - Estimated transfer time from a non-PCI-capable to a PCI-capable hospital
- Whether there was a pre-hospital STEMI diagnosis made by trained and fully equipped paramedic team who can administer initial pharmacotherapy
- The quality and expertise of the regional network infrastructure in place for STEMI management.

Many patients who present with STEMI are already taking long-term oral anticoagulation for various indications.[2]

- This is a **relative contraindication for fibrinolysis**.
- For any such patient, **choose a primary PCI strategy** for coronary reperfusion therapy, regardless of the anticipated time to access this intervention.

Concurrent anticoagulation and dual antiplatelet therapy is given alongside coronary reperfusion, regardless of whether primary PCI or fibrinolysis is used.

- **Seek specialist advice** if the patient has a separate indication for ongoing oral anticoagulation.

Patients presenting <12 hours from symptom onset

Primary PCI

Select primary PCI as the **preferred reperfusion strategy** for any patient who had **symptom onset <12 hours ago** and has:[2] [75] [166] [167] [168] [169] [172]

- **Ongoing signs and symptoms** of myocardial ischaemia, **AND**
- **Persistent (or increasing) ST-segment elevation** on ECG.

Take steps to ensure that primary PCI will be delivered **within 120 minutes of the time when fibrinolysis could have been given.** [75] [81]

- Primary PCI involves **immediate transfer to the catheterisation laboratory** with the intention of opening the artery with stent placement. Drug-eluting stents are recommended by the European Society of Cardiology (ESC) and National Institute for Health and Care Excellence guidelines.[2] [75]
- Many hospitals have **round-the-clock PCI capability**. If yours does not, then arrange **immediate transfer** to your **designated PCI-capable hospital**.

- If it is **not possible** to ensure primary PCI **within 120 minutes** of the time when fibrinolysis could be started, **offer fibrinolysis** (if not contraindicated) [75]
- If your patient has **STEMI with cardiogenic shock, seek urgent senior support.**Coronary angiography ± primary PCI is indicated.[2] [75]
- If the patient's coronary anatomy is unsuitable for PCI, or PCI fails, emergency coronary artery bypass graft (**CABG**) is recommended.[2]
- For more information on supportive management, see Shock .

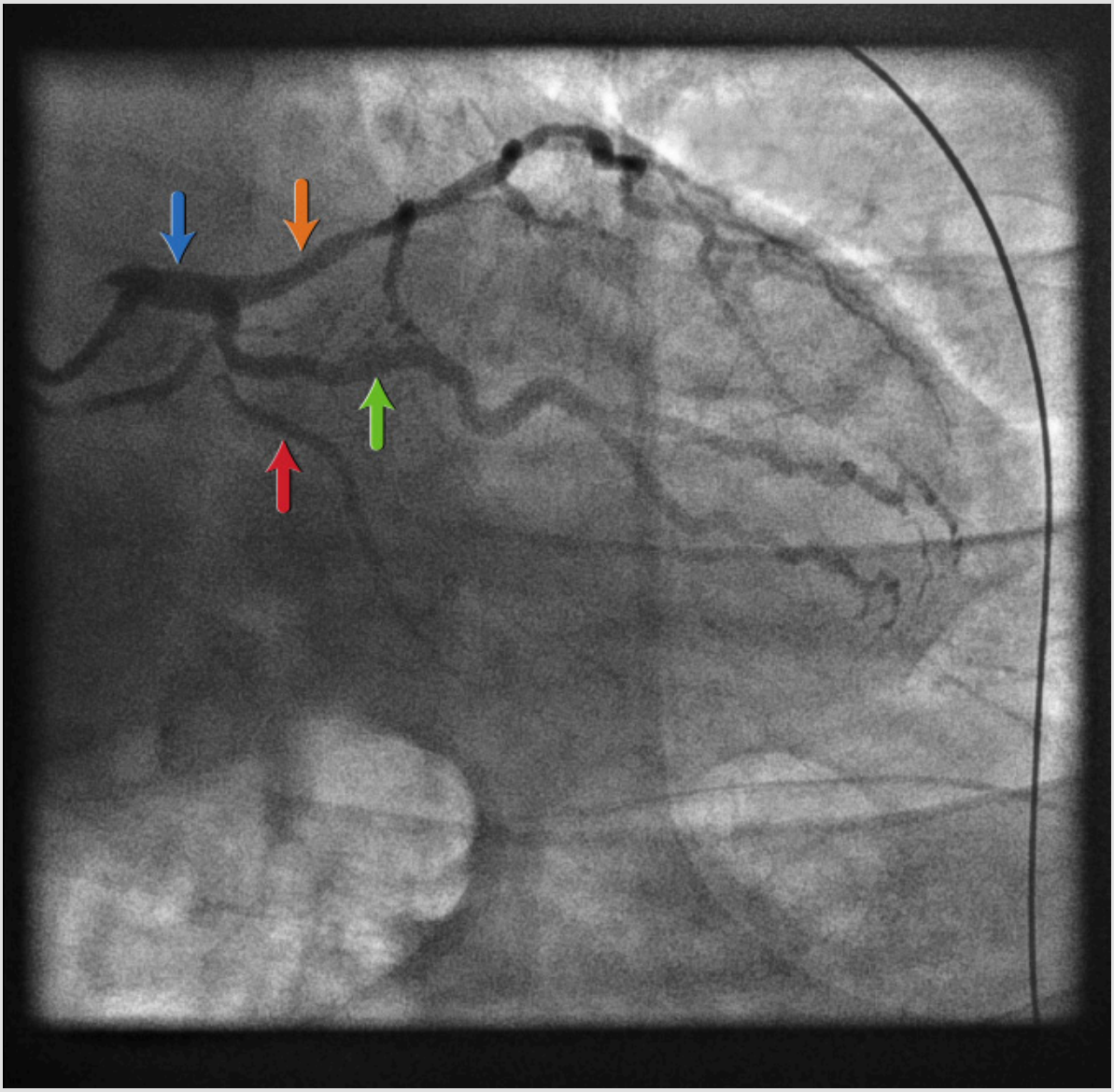
Practical tip

If your patient experiences a **spontaneous resolution of ST-segment elevation, along with complete symptom relief, after taking glyceryl trinitrate**, this is suggestive of **coronary spasm with or without associated MI.** [2]

- Refer to cardiology.
- Aim for early inpatient coronary angiography (within 24 hours).

More info: PCI angiograms – inferior STEMI case example

Left main coronary artery: right anterior oblique view



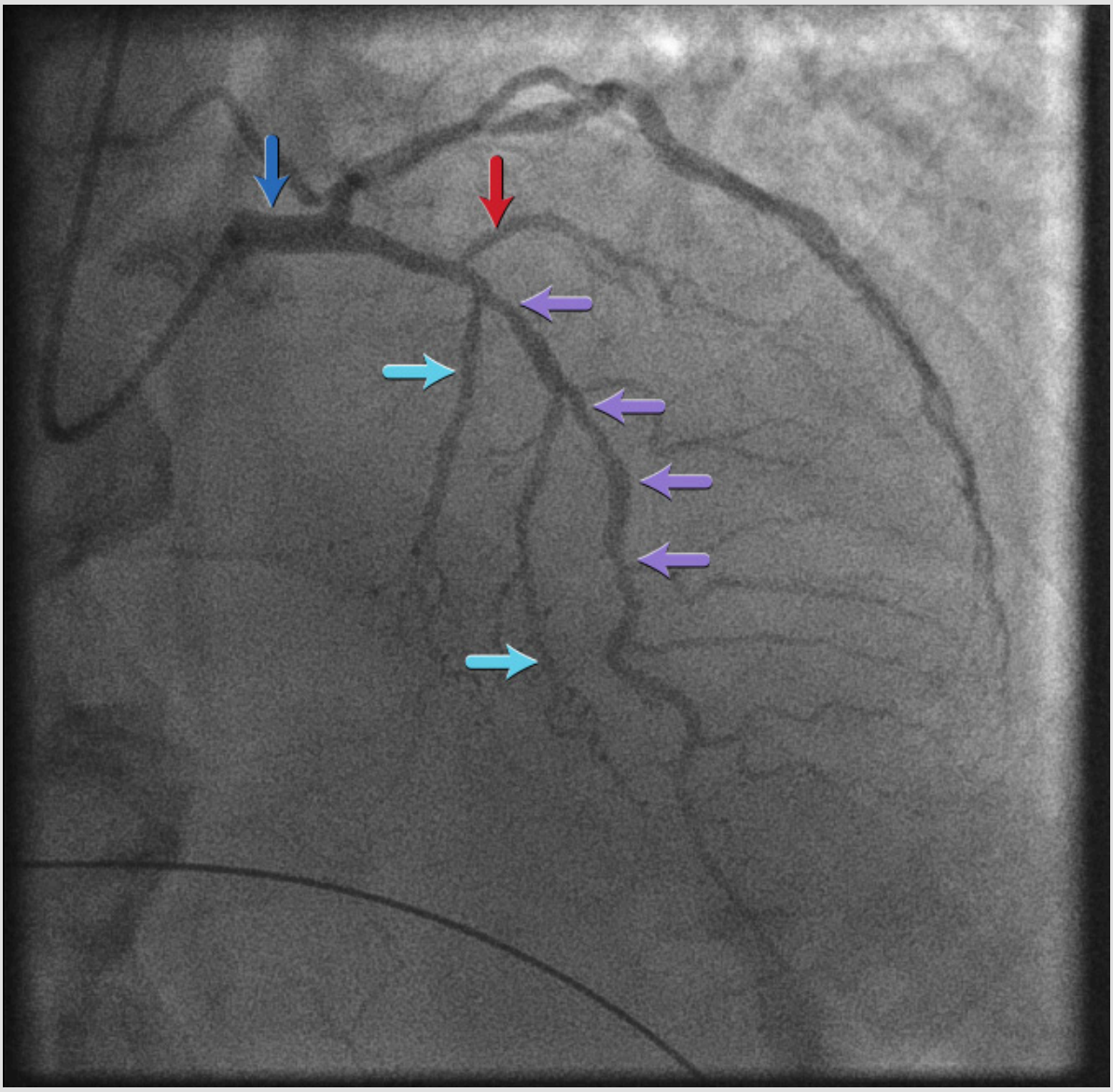
Left main coronary artery: right anterior oblique view

From the personal collection of Dr Aung Myat (used with permission)

- 70-year-old man admitted with crushing central chest pain
- Inferior ST elevation on ECG
- This would suggest a right coronary artery (RCA) occlusion so an angiogram of the left coronary artery system with a diagnostic catheter are taken first
- Bear in mind an inferior STEMI can also be associated with a left circumflex (LCx) occlusion
- This view allows you to see:
    - Left main stem (LMS) (blue arrow)
    - Proximal left anterior descending (LAD) artery (orange arrow)
    - True atrioventricular circumflex (AVCx) (red arrow), which is a small vessel in this case

- A large obtuse marginal (OM) branch artery of the LCx

Left main coronary artery: posterior anterior cranial view

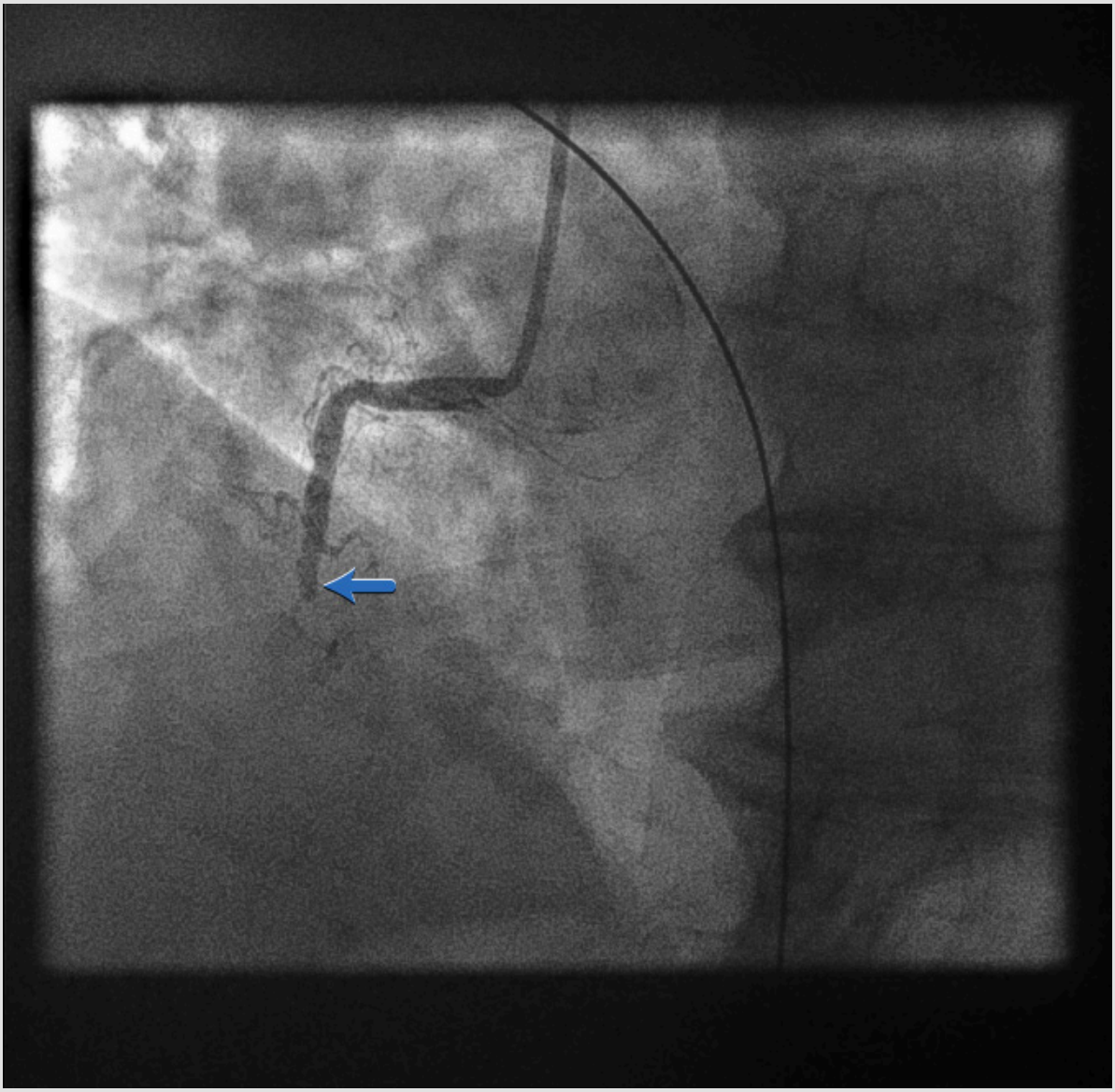


Left main coronary artery: posterior anterior cranial view

From the personal collection of Dr Aung Myat (used with permission)

- This view shows the LAD artery
- The LAD artery here is diffusely diseased throughout its mid course (purple arrows)
- Septal branches of the LAD artery are indicated by the turquoise arrows
- The red arrow corresponds to a diagonal branch of the LAD artery
- The LMS is shown by the blue arrow

Right coronary artery: left anterior oblique view



Right coronary artery: left anterior oblique view

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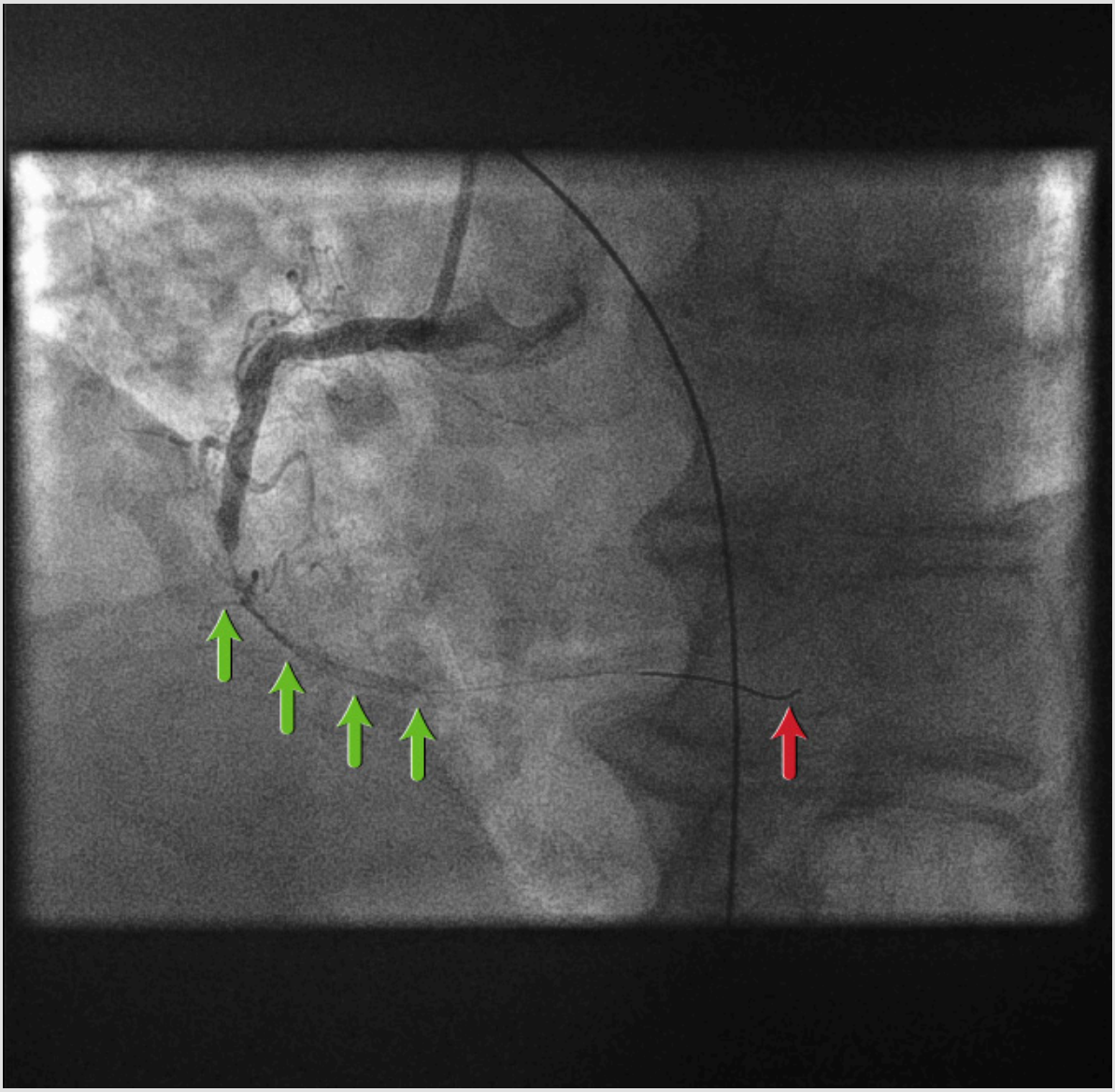
- As the initial left coronary artery angiogram showed no occlusion, we would typically assume the occlusion is in the RCA
- Therefore, a guide catheter (stiffer and larger calibre compared with a diagnostic catheter) would be used to intubate the RCA ostium
- Here the initial angiogram confirms an acute total occlusion of the mid RCA (blue arrow)

Coronary guidewire deployment



Coronary guidewire deployment

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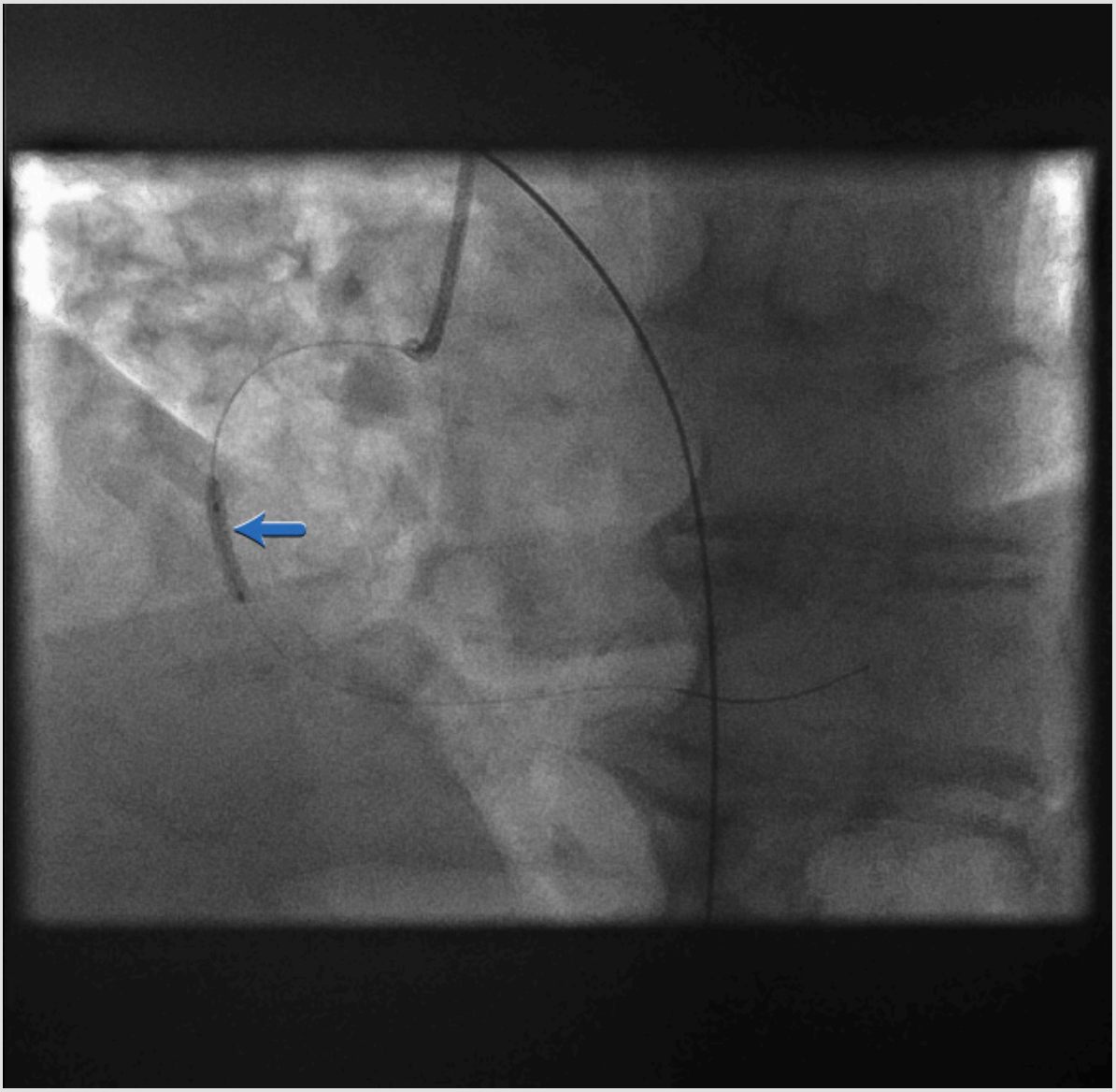
- Before introducing any equipment into a coronary artery, the patient is given a bolus dose of unfractionated heparin anticoagulation, adjusted according to weight
- A coronary guidewire is then deployed into the distal RCA (red arrow)
- Introduction of the guidewire already starts to restore coronary flow (green arrows)

First balloon dilatation

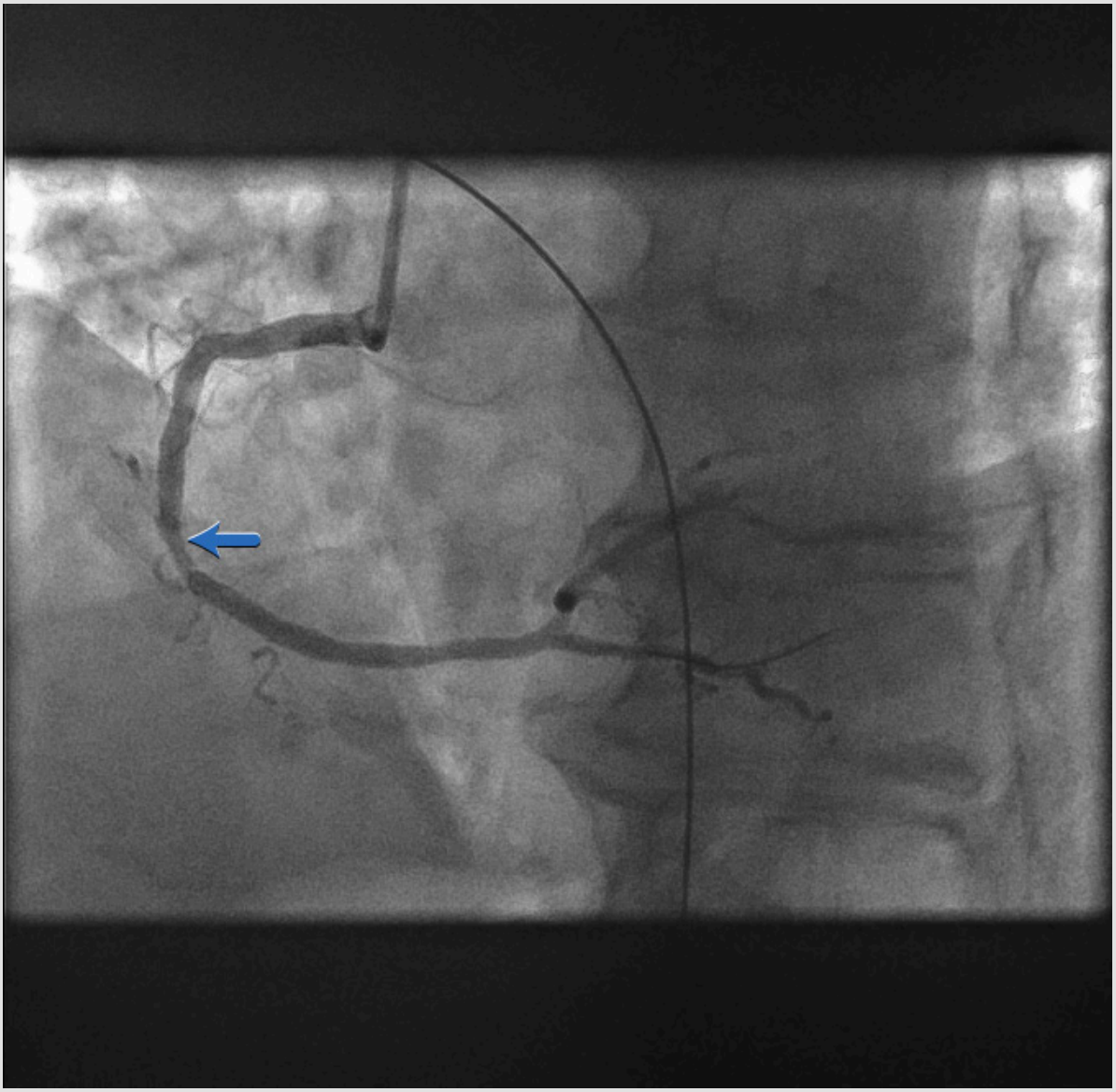


First balloon dilatation

From the personal collection of Dr Aung Myat (used with permission)

- A balloon is passed over the coronary guidewire and inflated across the point of the original acute occlusion (blue arrow)

Restoration of coronary flow

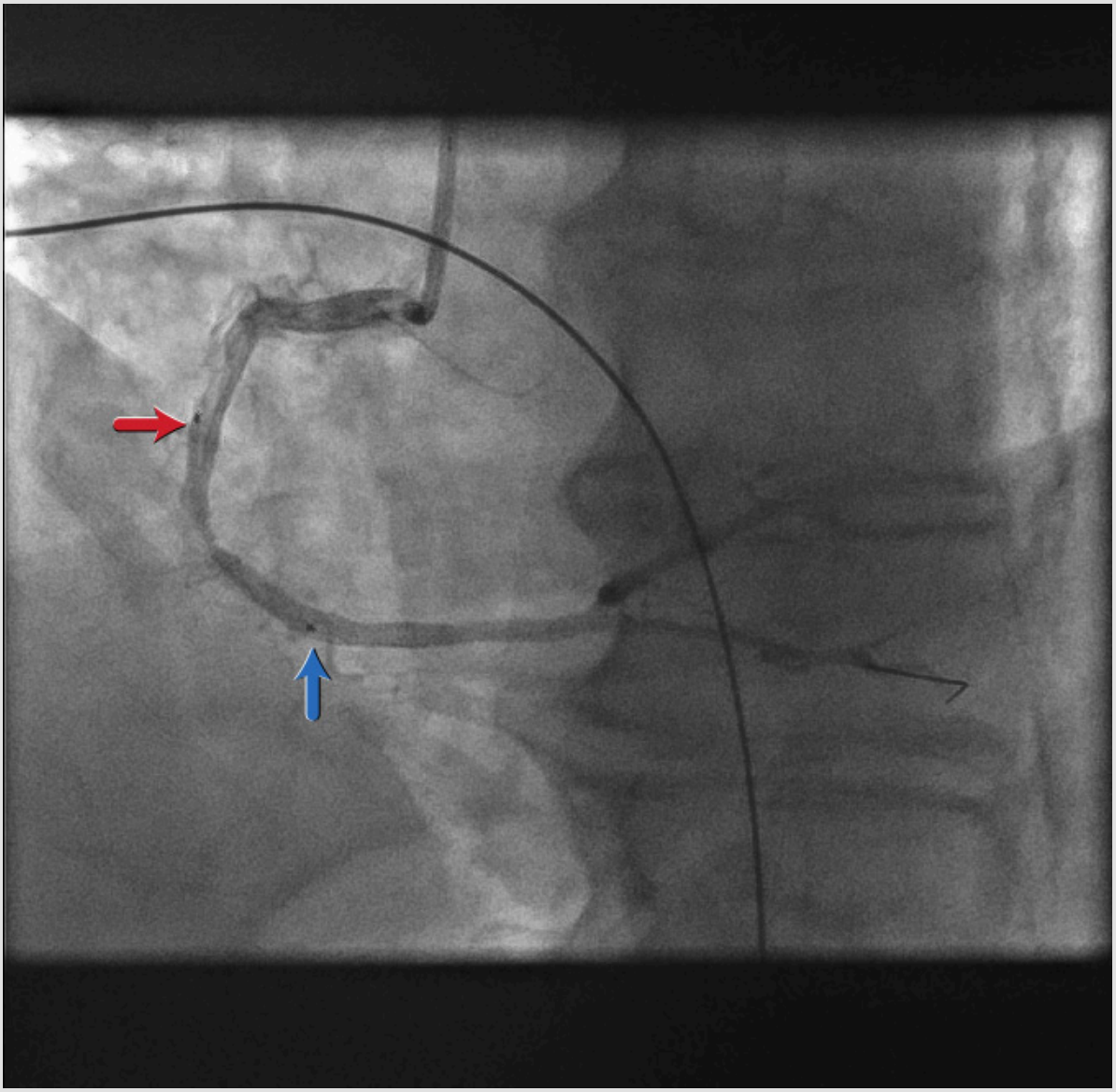


Restoration of coronary flow

From the personal collection of Dr Aung Myat (used with permission)

- The first balloon inflation restores flow to the RCA
- Now the full outline of the artery can be seen
- The area of stenosis and likely atherosclerotic plaque rupture is shown by the blue arrow

Stent positioning

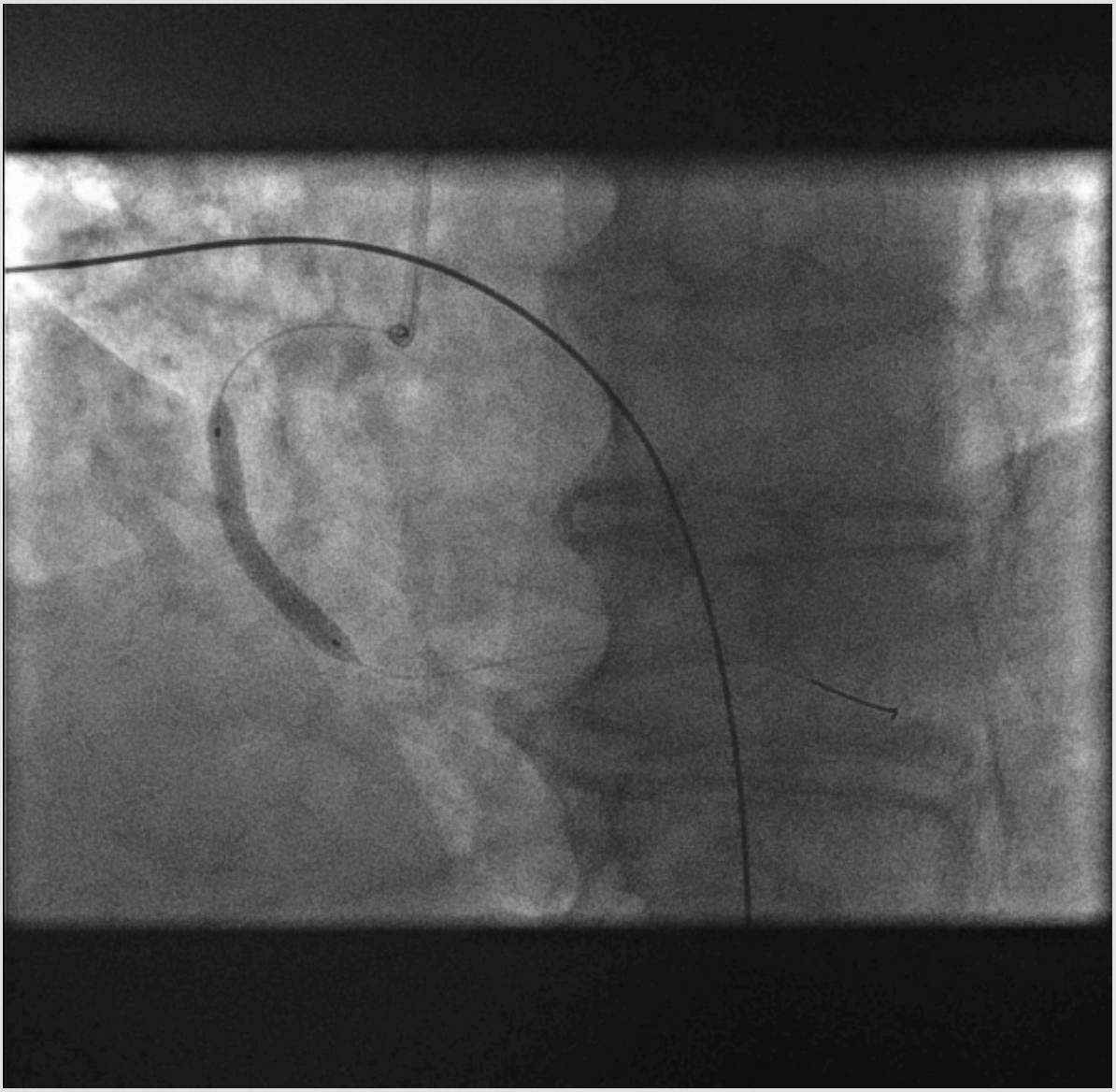


Stent positioning

From the personal collection of Dr Aung Myat (used with permission)

- The proximal (red arrow) and distal (blue arrow) stent markers are shown here
- A decision has been made to stent from what is perceived to be 'normal' artery to 'normal' artery covering the culprit lesion

Stent deployed



Stent deployed

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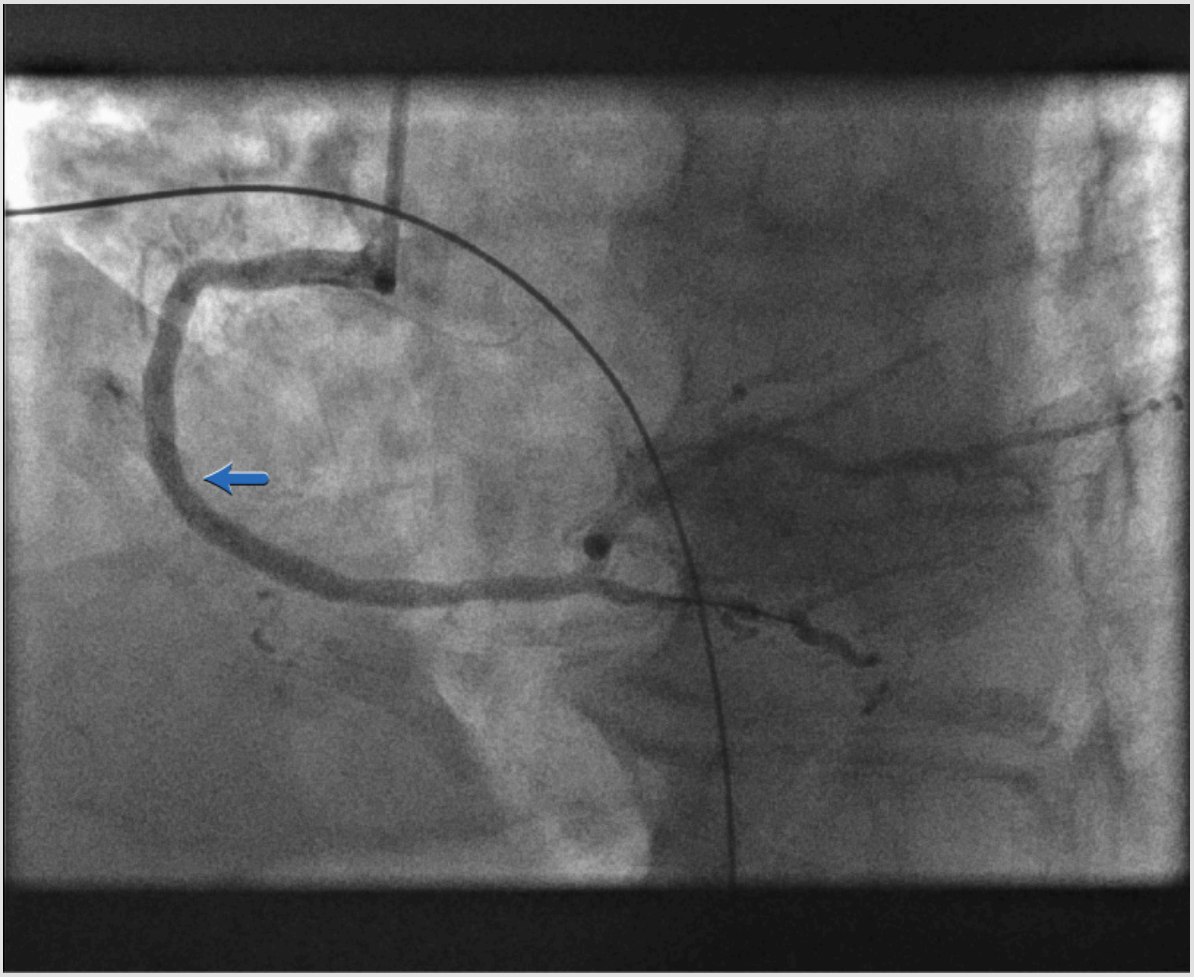
- The stent is sized according to diameter and length in millimetres
- Stent deployment transiently re-occludes the vessel, which can cause pain for the patient and haemodynamic changes

After stent deployment

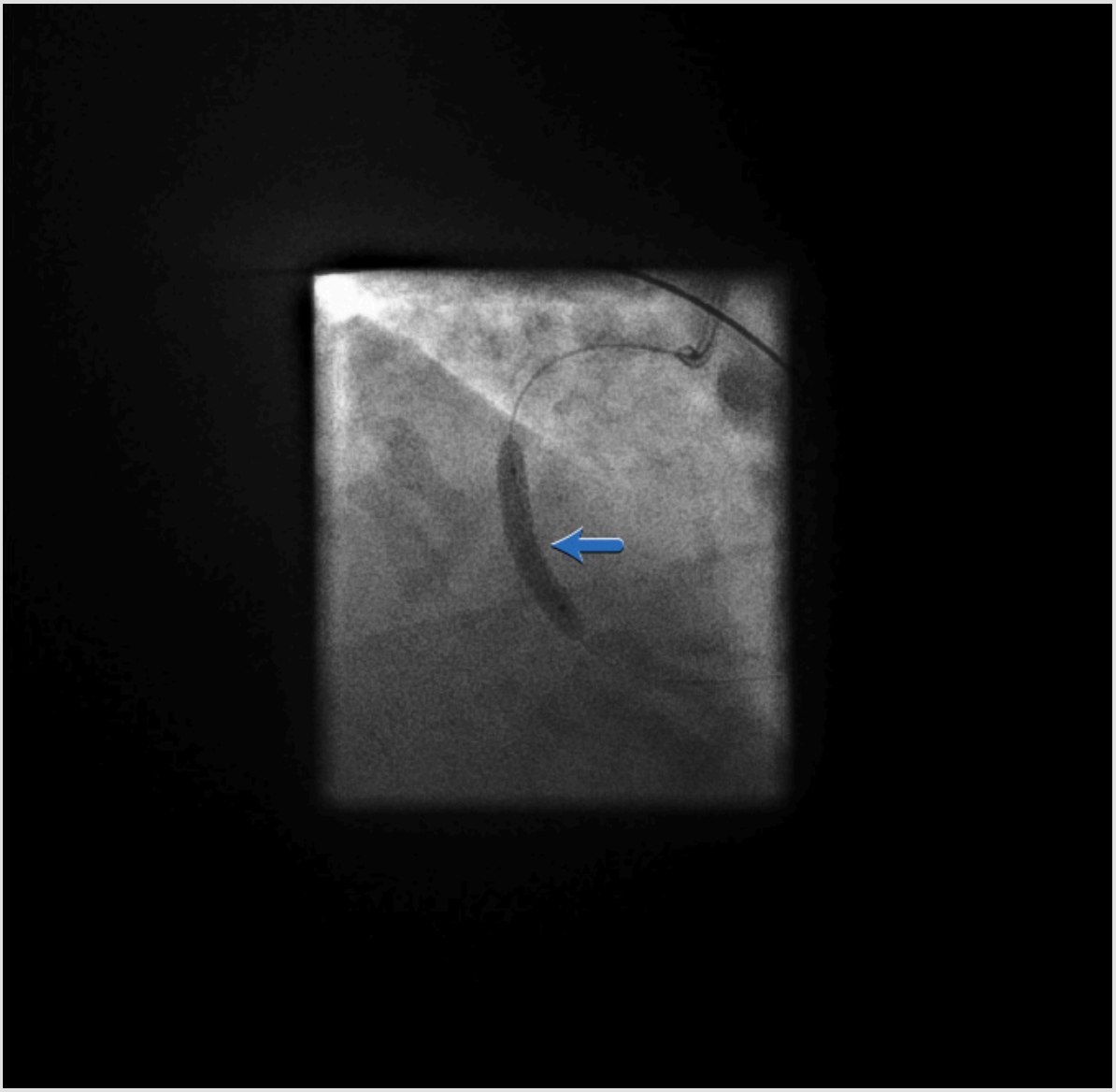


After stent deployment

From the personal collection of Dr Aung Myat (used with permission)

- The stent is now expanded within the RCA
- There remains an area of underexpanded stent as shown by the blue arrow
- Underexpansion can be seen directly using intravascular ultrasound (IVUS) or optical coherence tomography (OCT)
- Here the underexpansion assumption is based solely on angiographic appearance

Stent post-dilatation

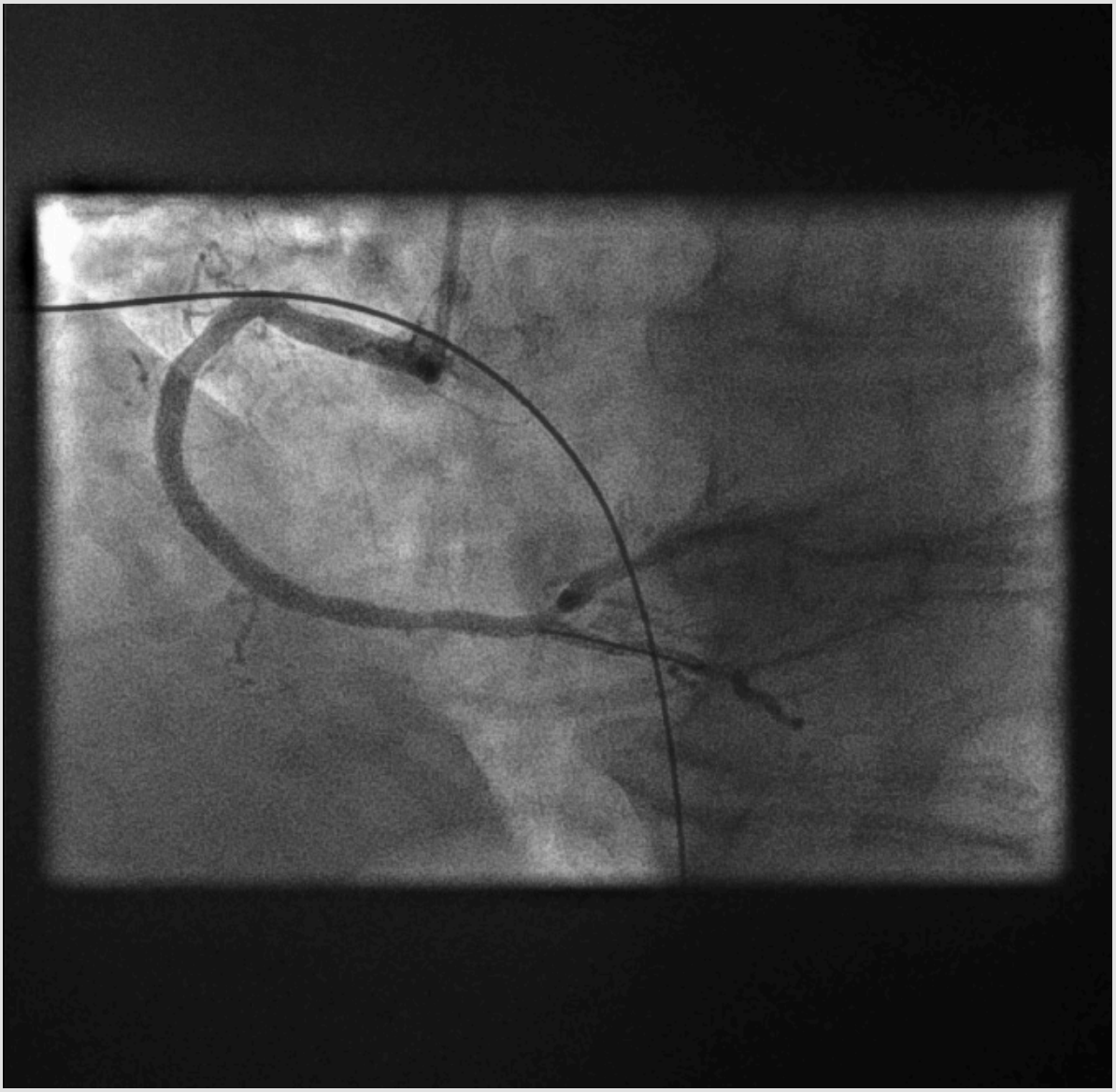


Stent post-dilatation

From the personal collection of Dr Aung Myat (used with permission)

- A post-stent balloon dilatation (blue arrow) is performed to ensure adequate stent expansion
- Underexpansion of a stent can lead to acute stent thrombosis

RCA primary angioplasty: final result



RCA primary angioplasty: final result

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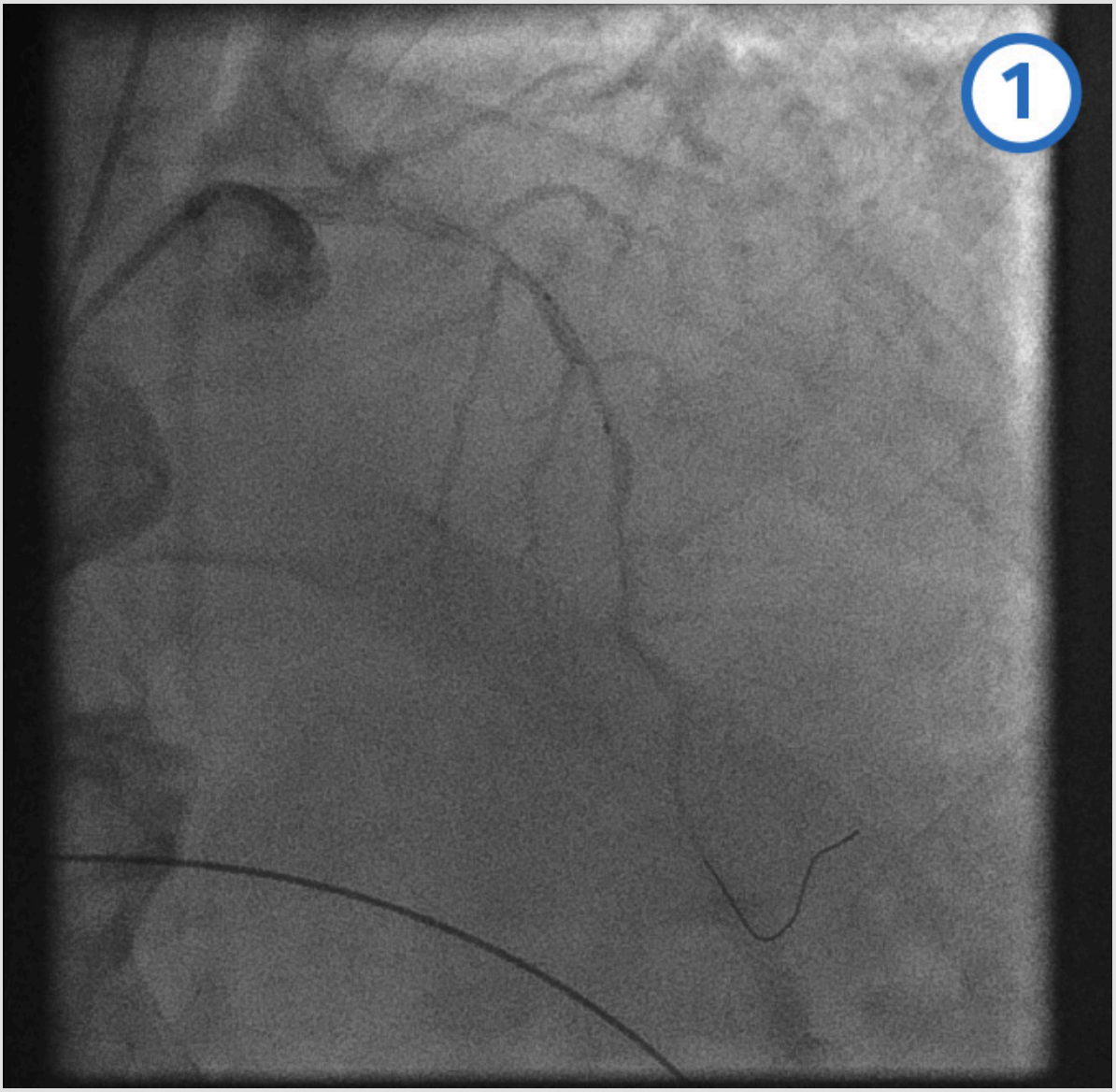
- There is an excellent angiographic result
- The stent appears fully expanded angiographically with reference to the proximal and distal vessel

Left anterior descending artery pre-dilatation



Left anterior descending artery pre-dilatation image 1

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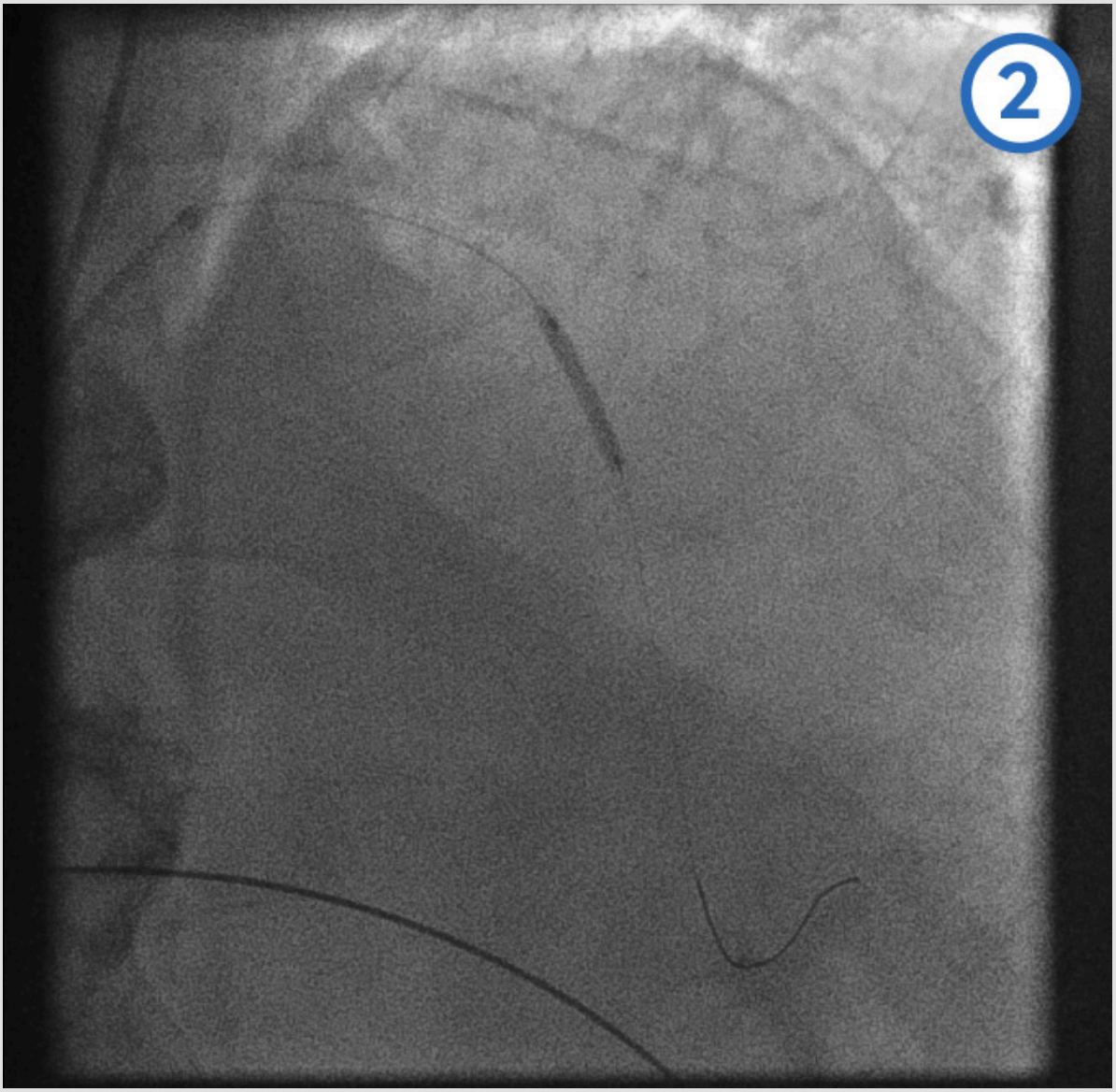

Left anterior descending artery pre-dilatation image#2

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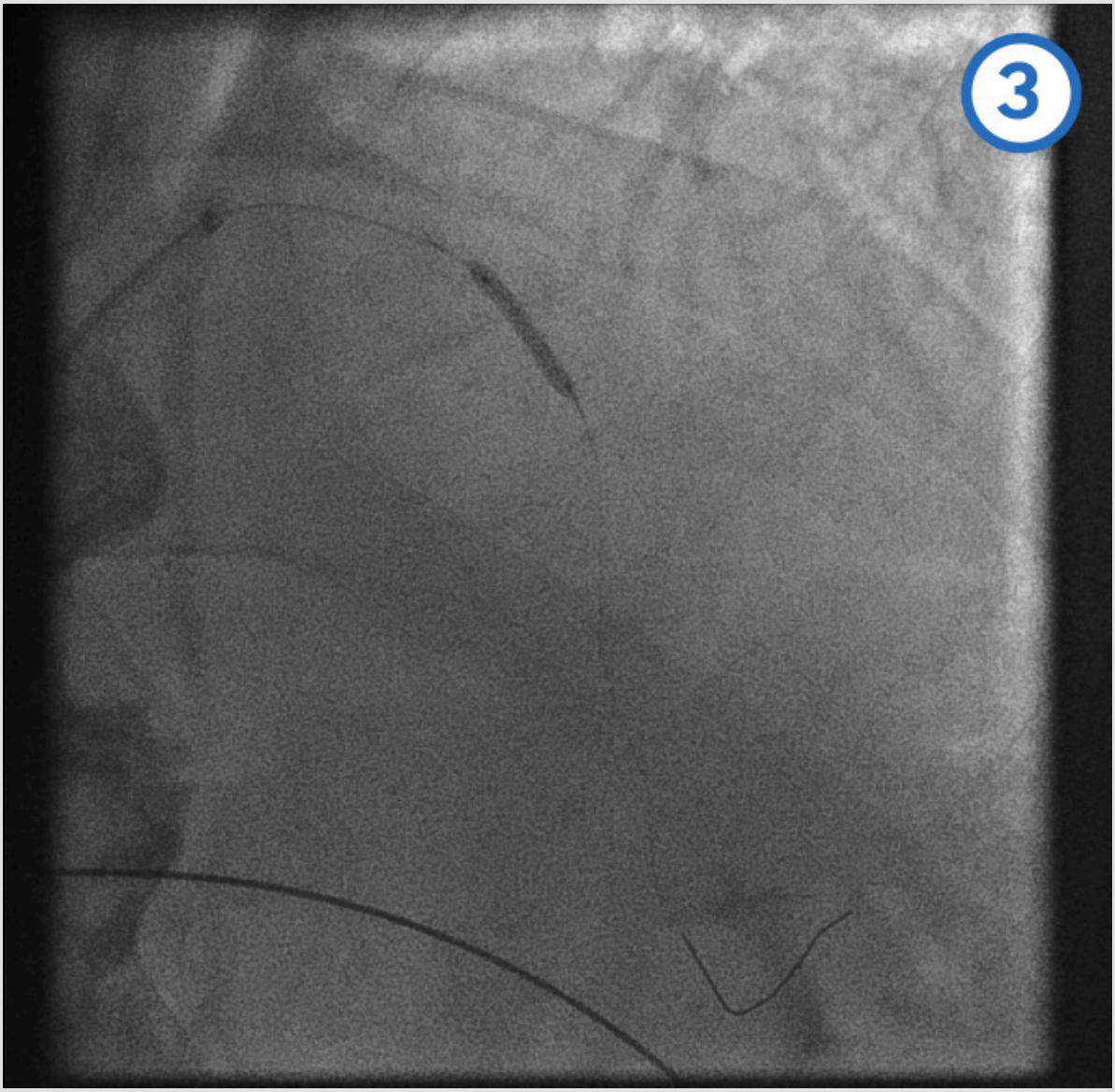

Left anterior descending artery pre-dilatation image 3

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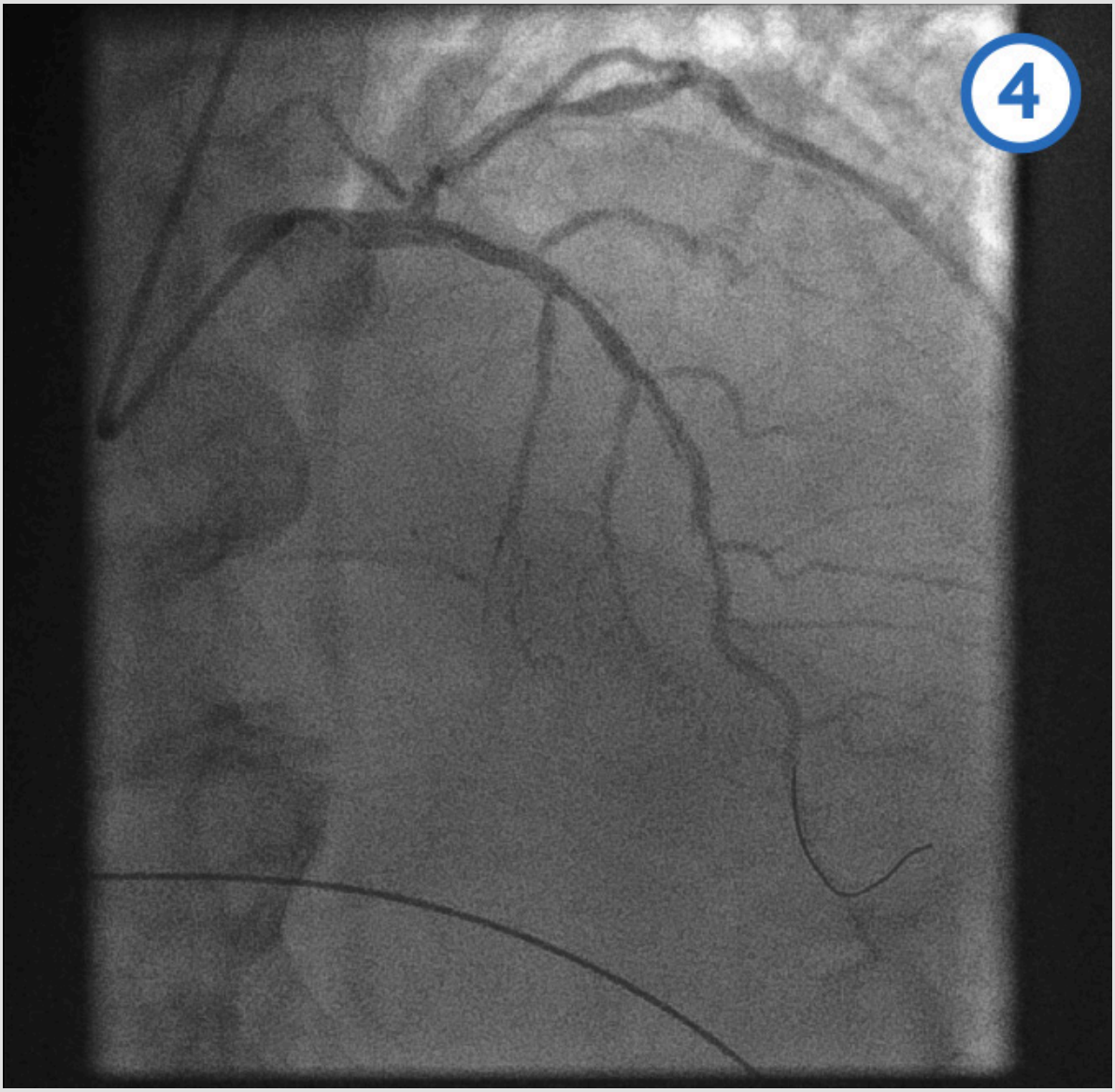

Left anterior descending artery pre-dilatation image 4

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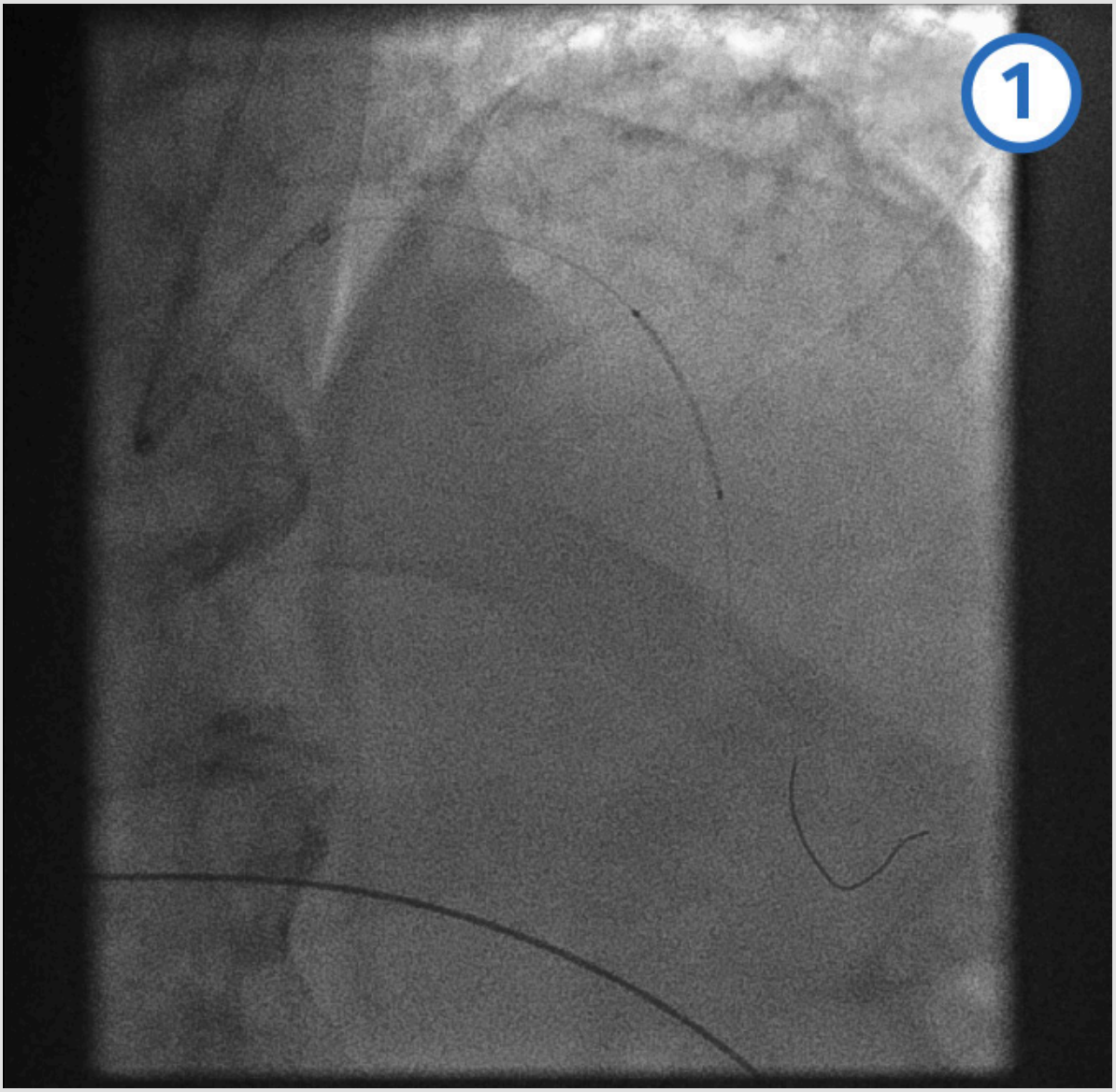
- Complete versus infarct-related artery-only (IRA) revascularisation in patients with multi-vessel disease during STEMI remains an area of open debate
- The ESC acute coronary syndrome guideline advocates IRA-only revascularisation in patients with cardiogenic shock during the index procedure, with consideration for staged PCI of non-IRA stenoses[2]
- For haemodynamically stable patients, the ESC recommends revascularisation of non-IRA lesions either during the index procedure or within 45 days[2]
- In this case the decision was made to revascularise the LAD during the index procedure
- A coronary guidewire is deployed in the distal LAD (image 1)
- The mid (image 2) and proximal (image 3) LAD is then dilated with a balloon
- Image 4 shows the LAD after the serial balloon dilatations

Mid-LAD artery stent deployment



Mid-LAD artery stent deployment#mage 1 - stent positioned in the mid-LAD artery

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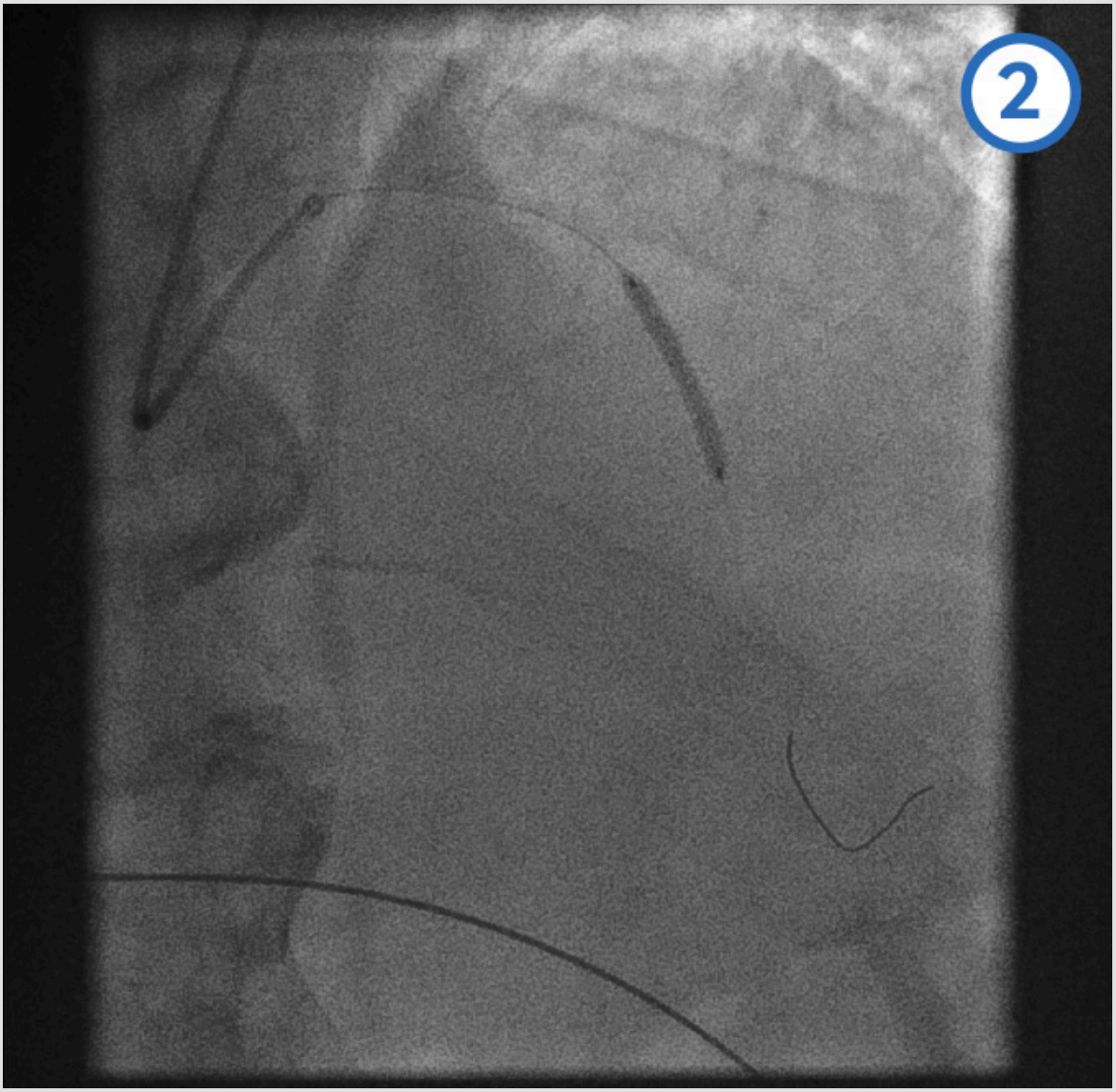

Mid-LAD artery stent deployment image 2 - stent balloon inflated

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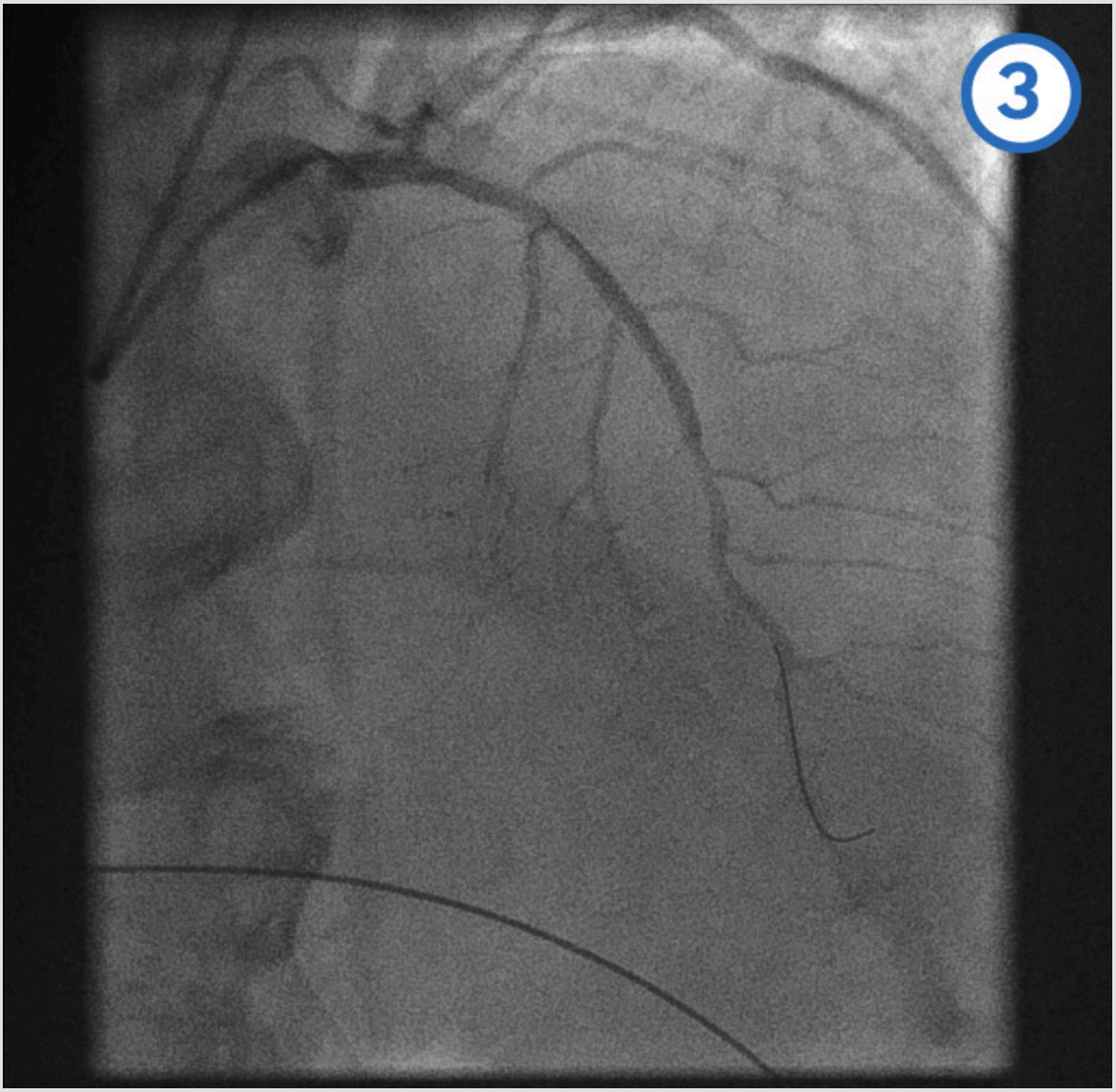

Mid-LAD artery stent deployment image 3 - post mid-LAD stent deployed

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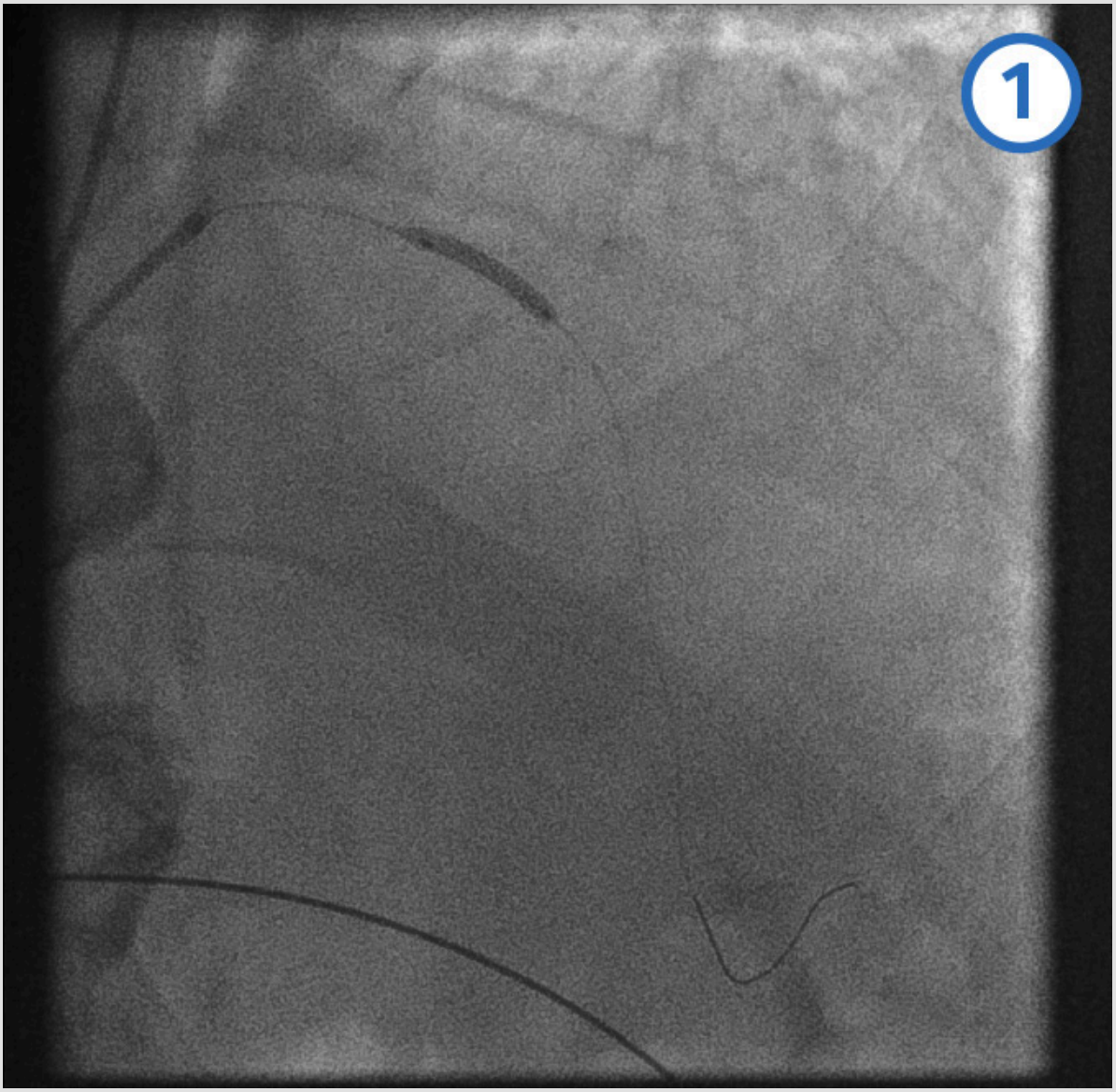
Proximal LAD stent deployment



Proximal LAD stent deployment image 1 - following the mid-LAD stent deployment the proximal vessel is pre-dilated

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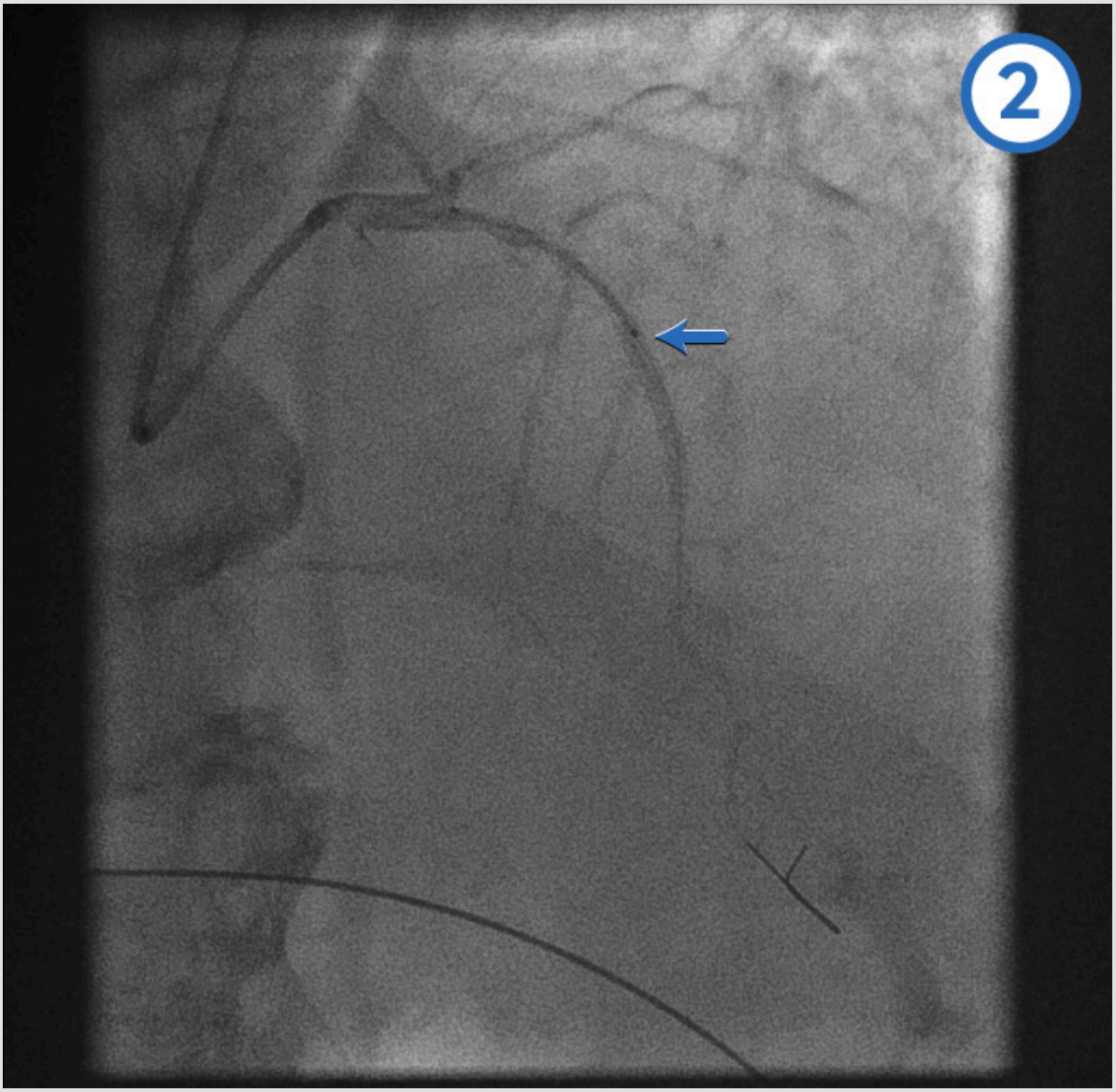

Proximal LAD stent deployment image 2 - proximal stent is positioned with the distal marker (blue arrow) overlapping/within the proximal end of the previously deployed mid-LAD stent

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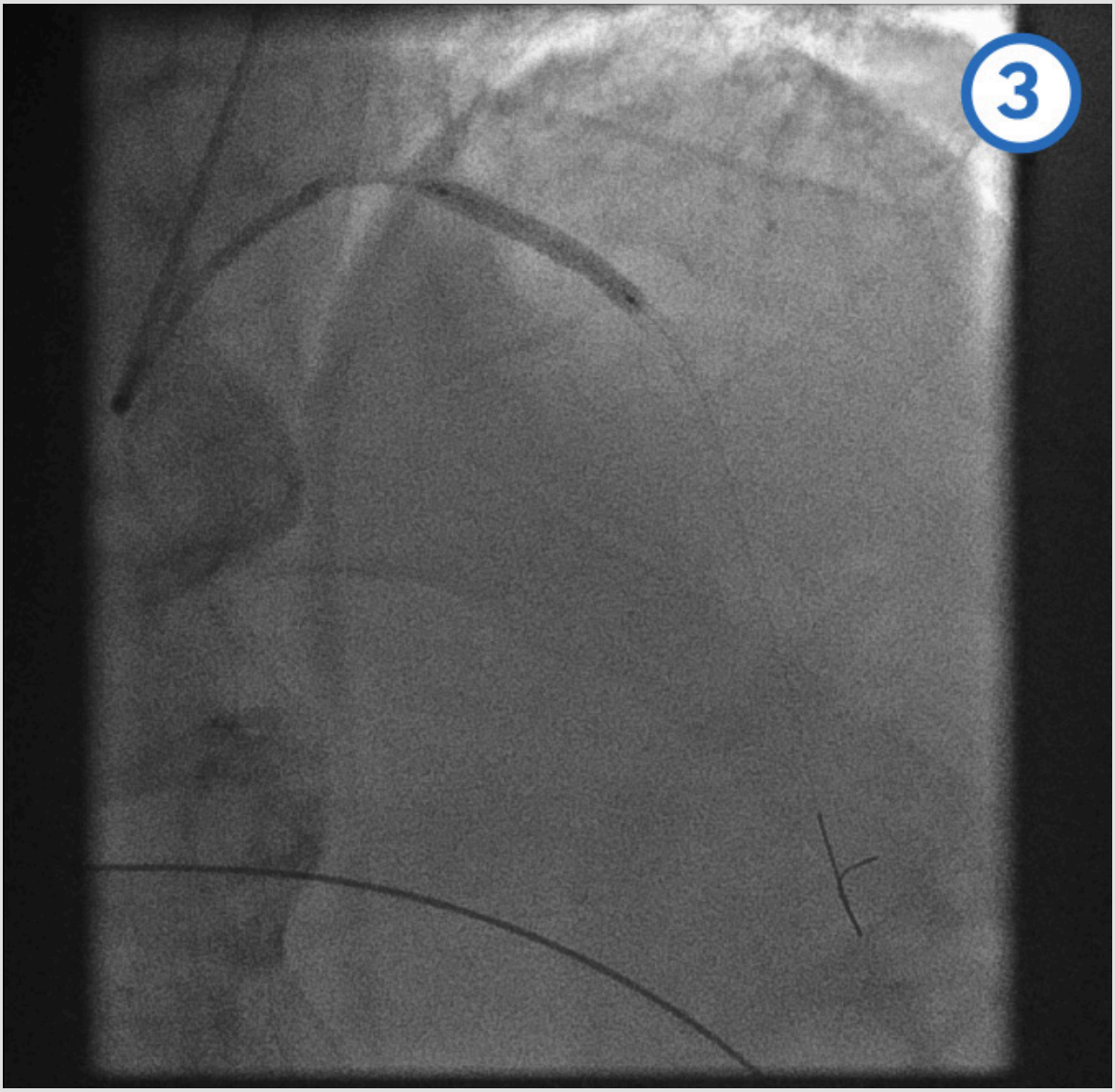

Proximal LAD stent deployment image 3 - proximal stent deployed

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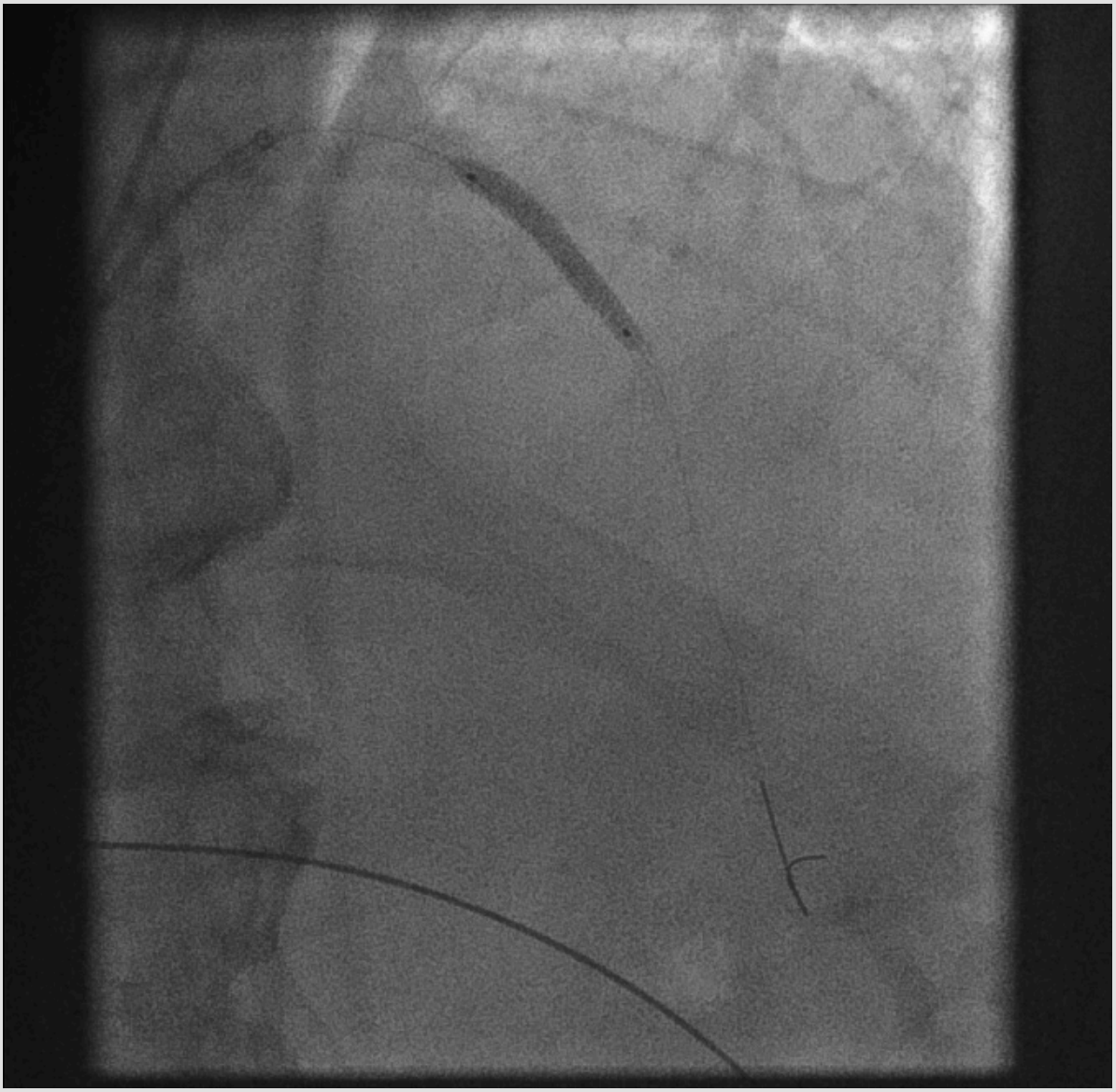
Post-dilatation of overlapping stents



Post-dilatation of overlapping stents

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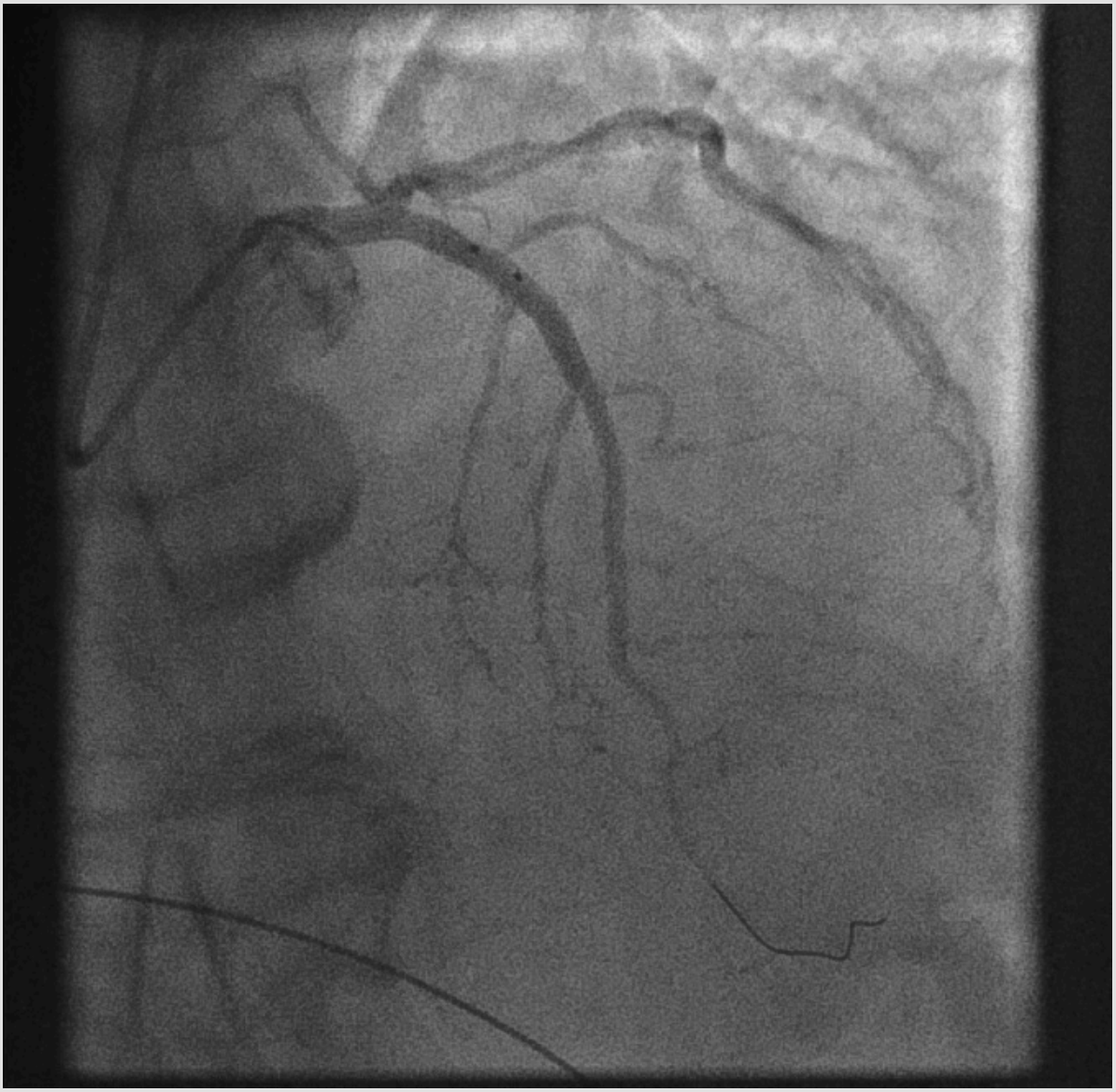
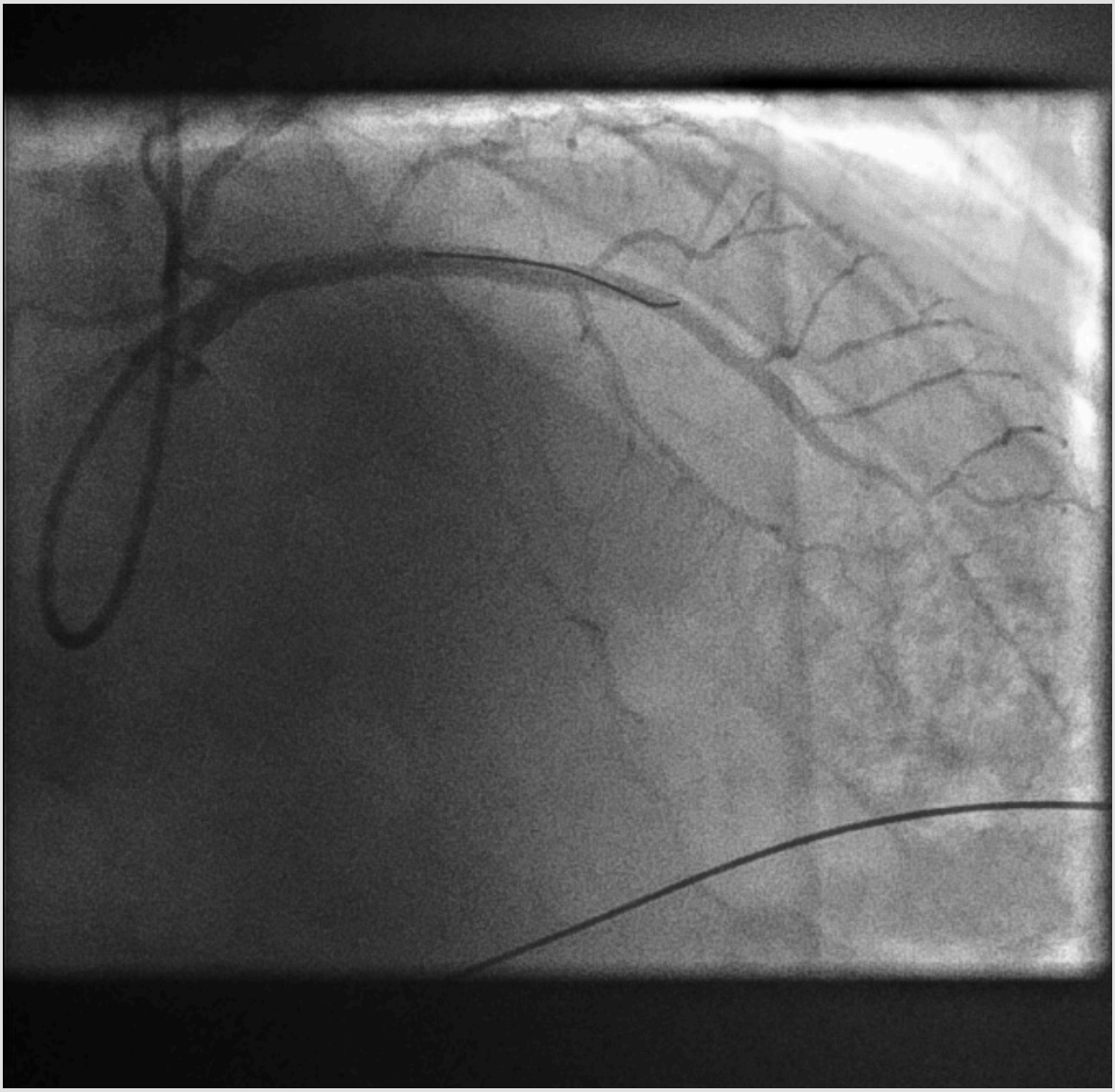
- The same balloon of the proximal stent is then used to post-dilate the overlap between the proximal and mid-LAD stents

Final LAD stenting result



Final LAD stenting result: posterior anterior cranial view

From the personal collection of Dr Aung Myat (used with permission)



Final LAD stenting result: right anterior oblique cranial view

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Conclusion:

- The patient had complete coronary revascularisation with primary angioplasty to the occluded RCA and non-IRA angioplasty to the LAD artery. The patient made an excellent recovery and was discharged home 72 hours after admission.

Fibrinolysis

Give fibrinolysis (unless contraindicated) **if primary PCI cannot be delivered within 120 minutes** of the time when fibrinolysis could be given.[75] [81]

- The ESC recommends that fibrinolysis should be given within 10 minutes of making the clinical diagnosis of STEMI.[2]

- Check your local protocols for advice on the **preferred fibrinolytic drug** and how it should be administered. See *Choice of fibrinolytic drug* below.
- **Start anticoagulation** at the same time as giving fibrinolysis and start dual antiplatelet therapy immediately afterwards.[2] [71] [75] See *Antithrombotic co-therapy given with fibrinolysis* below.

If primary PCI **cannot be delivered within 120 minutes of STEMI diagnosis, seek a specialist cardiology opinion** to support your choice of reperfusion strategy.[2] [71] [153]

- The greatest benefit of fibrinolysis is observed when given <2 hours after symptom onset. Its clinical **efficacy diminishes as time from symptom onset increases**.[72] [154]
- Therefore, the later the patient presents, the more consideration should be given to transferring for **primary PCI instead** – discuss the options with cardiology.[2] [74]

If your patient is not already at a PCI-capable hospital, transfer them to one immediately after giving fibrinolysis.[2]

- This is so that rescue PCI can be performed if fibrinolysis fails or angiography ± PCI can be arranged if fibrinolysis is effective.

Use an **ECG 60-90 minutes after administering fibrinolysis** to assess whether it has been successful.[2] [75]

- **If fibrinolysis has failed** (<50% ST-segment resolution at 60-90 minutes), offer immediate coronary angiography, with follow-on PCI if indicated. **Do not repeat fibrinolytic therapy**.[2] [75]
- **If fibrinolysis is successful** , consider angiography during the same hospital admission for patients who are clinically stable.[75]
    - The ESC guidelines recommend arranging **angiography ± PCI** of the infarct-related artery **within 2-24 hours**.[2]
    - Several randomised trials and two meta-analyses have shown that early routine angiography (with PCI if needed) after fibrinolysis reduces the rate of reinfarction and recurrent ischaemia compared with a watchful waiting strategy. The benefits of early routine PCI following fibrinolysis were seen across patient subgroups and without any increased risk of adverse events such as stroke or major bleeding.[173] [174] [175]
- If the patient has **recurrent myocardial ischaemia** after fibrinolysis, **seek immediate specialist advice from cardiology**. PCI may be indicated.[75]

The ESC guideline recommends **immediate rescue PCI if**, at any time following fibrinolysis, the patient develops **haemodynamic or electrical instability, worsening ischaemia, or persistent chest pain.** [2]

Emergency angiography ± PCI is recommended if, at any time following fibrinolysis, the patient has:

- Heart failure or cardiogenic shock[2]
- Recurrent ischaemia or evidence of re-occlusion after initially successful fibrinolysis.[2] [176] [177]

Practical tip

The ESC acute coronary syndrome guideline lists the contraindications to fibrinolytic therapy as follows:[2]

- **Absolute contraindications**
  - History of intracranial haemorrhage or stroke of unknown origin at any time
  - Ischaemic stroke in the last 6 months
  - Central nervous system damage, neoplasm, or arteriovenous malformation
  - Major trauma/surgery/head injury within the last 1 month
  - Gastrointestinal bleeding within the last 1 month
  - Bleeding disorder
  - Aortic dissection
  - Non-compressible punctures within the last 24 hours (e.g., liver biopsy, lumbar puncture)
- **Relative contraindications**
  - Transient ischaemic attack in the last 6 months
  - Oral anticoagulant therapy
  - Pregnancy or within 1 week postnatal
  - Refractory hypertension (systolic BP >180 mmHg and/or diastolic BP >110 mmHg)
  - Advanced liver disease
  - Infective endocarditis
  - Active peptic ulcer
  - Prolonged or traumatic cardiopulmonary resuscitation.

If primary PCI is not available within 120 minutes but your patient has a contraindication to fibrinolysis:

- Seek immediate advice from the interventional cardiology team
- For a patient with an absolute contraindication to fibrinolysis, primary PCI will normally be the preferred management strategy despite the delay in accessing it.

Evidence: Role of fibrinolysis in STEMI

Fibrinolytic therapy remains an important evidence-based intervention in settings where primary PCI cannot be offered in a timely manner.

Several landmark studies demonstrated the benefits of fibrinolysis in patients with STEMI (prior to the advent of primary PCI).

- The **1988 Second International Study of Infarct Survival** (ISIS-2) proved that the benefits of aspirin and fibrinolytics (e.g., streptokinase) were additive and associated with improved 10-year survival following acute MI.[178]
- The **Fibrinolytic Therapy Trialists' Collaborative Group** (1994) demonstrated highly significant benefits in saving lives and helped to establish the time window when fibrinolytic therapy is most effective.[154]
    - The study demonstrated highly significant absolute mortality reductions from fibrinolysis of about:
        - 30 per 1000 for those presenting within 0-6 hours of symptom onset
        - 20 per 1000 for those presenting 7-12 hours after symptom onset.
    - There was a statistically uncertain benefit of about 10 per 1000 for those presenting at 13-18 hours (with more randomised evidence needed in this group to assess reliably the net effects of treatment).
- The 1993 **GUSTO trial** compared different fibrinolysis strategies.[179] [180]
    - It demonstrated a benefit of 14% reduction in mortality (95% CI, 5.9% to 21.3%) for an accelerated tissue plasminogen activator (t-PA) versus streptokinase (the standard fibrinolytic treatment at the time). Alteplase (recombinant t-PA) was used in the trial.
    - The trial randomly assigned 41,021 patients with evolving acute MI to 4 different fibrinolytic strategies. The data for the primary end point of 30-day mortality for each treatment arm was as follows:
        - Streptokinase plus subcutaneous heparin = 7.2%
        - Streptokinase plus intravenous heparin = 7.4%
        - t-PA plus intravenous heparin = 6.3%
        - Streptokinase plus t-PA plus intravenous heparin = 7.0%.
    - A significant excess of haemorrhagic strokes was observed for accelerated t-PA (P = 0.03) and for the combination strategy (P <0.001), as compared with streptokinase only.

The 2013 STREAM trial provided further support for fibrinolysis as an appropriate reperfusion strategy for STEMI when primary PCI cannot be delivered in a timely manner – especially if followed by routine early angiography ± PCI (a 'pharmaco-invasive strategy'). [171]

- The STREAM trial was conducted at a time when primary PCI was established as the standard of care for STEMI.
- In the trial, 1892 STEMI patients presenting within 3 hours of symptom onset but unable to have primary PCI within 1 hour were randomly assigned to:
    - Primary PCI or
    - Pre-hospital bolus tenecteplase (with a half-dose used in patients ≥75 years) plus clopidogrel plus enoxaparin before transport to a PCI-capable hospital
        - Patients had emergency angiography if fibrinolysis failed
        - If fibrinolysis was successful, patients had routine angiography within 6-24 hours after randomisation.
- There was no significant difference between the primary PCI and fibrinolysis groups in the primary composite end point of death, shock, congestive heart failure, or reinfarction within 30 days.
    - More intracranial haemorrhages occurred in the fibrinolysis group.
- **The ESC guideline recommendation is that fibrinolysis should be given within 10 minutes of STEMI diagnosis.** [2]

The extent to which the delay-to-PCI time diminishes the advantages of primary PCI over fibrinolysis has been widely debated, and more research is needed.

- It is clear that if delay to treatment is similar, primary PCI is superior to fibrinolysis in reducing mortality, reinfarction, or stroke.[2]
    - However, fibrinolysis can be administered expeditiously and the longer the delay to primary PCI, the lower the benefits over fibrinolysis. The exact cut-off point at which the delay to access PCI makes fibrinolysis the better reperfusion strategy is based on weak evidence.

Multivariate meta-analyses provide further evidence to support a pharmaco-invasive strategy, suggesting it is safer and more effective than facilitated PCI or fibrinolysis, where primary PCI is not available in a timely fashion.[181]

Patients presenting >12 hours from symptom onset

Seek immediate specialist advice from cardiology to discuss management options for any STEMI patient who presents >12 hours after symptom onset.

- Coronary angiography ± primary PCI is recommended if there is:
    - Evidence of continuing myocardial ischaemia or cardiogenic shock[75]

- Haemodynamic instability or life-threatening arrhythmias.[2]

If your patient has ongoing ischaemic symptoms suggestive of MI but no ongoing ST-segment elevation on the ECG, primary PCI should be considered if one or more of the following is present:[2]

- Cardiogenic shock or haemodynamic instability
- Acute heart failure presumed secondary to ongoing myocardial ischaemia
- Recurrent or refractory chest pain despite medical treatment
- Cardiac arrest or life-threatening arrhythmia
- Signs and symptoms suggestive of mechanical complications of acute MI
- Recurrent dynamic ST-segment or T-wave changes, especially intermittent ST-segment elevation.

Discuss urgently with cardiology any patient who presents >12 hours after symptom onset but has no ongoing symptoms.[2] [182]

- Routine primary PCI strategy should still be considered in patients presenting between 12 and 48 hours after symptom onset. However, if the time since symptom onset is >48 hours and the patient is now asymptomatic, routine PCI of an occluded infarct-related artery is not recommended.[2]
- Fibrinolysis is not indicated.

Survivors of out-of-hospital cardiac arrest

Primary PCI is the treatment of choice for any patient in whom a **return of spontaneous circulation (ROSC)** is achieved **after cardiac arrest and who has**:[2] [147] [148] [149] [150] [151]

- ST-segment elevation on their post-ROSC ECG.

In a patient who has been resuscitated from cardiac arrest but has **no ST-segment elevation** on their post-ROSC ECG:[2]

- **Exclude non-coronary causes** of cardiac arrest:
  - Non-cardiogenic shock
  - Respiratory failure
  - Cerebrovascular event
  - Pulmonary embolism
  - Intoxication/poisoning
- Perform **urgent echocardiography** [2]
  - Assess left and right ventricular function
  - Identify other mechanical complications of acute MI
- Consider referral to cardiology for **urgent angiography** if there is a **high index of suspicion of ongoing myocardial ischaemia** despite no ST-segment elevation, for example:
  - Chest pain prior to cardiac arrest

- Presence of cardiac risk factors
- Established coronary artery disease or previous myocardial infarction
- Ambiguous or abnormal post-ROSC ECG.

To make a decision on whether to take a survivor of cardiac arrest (with or without ST-segment elevation) to the catheterisation laboratory for urgent angiography ± PCI:[2]

- Consider each case on its **individual merits** and **seek senior advice**
- Take account of factors associated with a **greater chance of a good neurological outcome**, including:[183]
  - A witnessed cardiac arrest
  - Immediate bystander cardiopulmonary resuscitation and/or early arrival of emergency medical services (<10 minutes)
  - An initial shockable rhythm
  - Sustained ROSC achieved within 20 minutes.
- The European Society of Cardiology recommends that, in general, patients with ROSC and persistent ST-segment elevation should undergo angiography ± PCI. Although there is a lack of dedicated trials, registry reports suggest good outcomes for PCI in these patients, particularly for those who are non-comatose at initial assessment.

See Cardiac arrest .

Target times for administering coronary reperfusion

The benefits of reperfusion in reducing mortality and improving myocardial salvage **decline rapidly with time**.[2] [75]

- It is, therefore, vital to take every step possible to ensure the chosen reperfusion strategy (primary percutaneous coronary intervention [PCI] or fibrinolysis) is delivered as quickly as possible.

The 2023 European Society of Cardiology acute coronary syndrome guideline sets widely accepted maximum time-to-treatment targets as follows:[2]

Interval	Target
First medical contact to ECG and STEMI diagnosis	**<10 minutes**
STEMI diagnosis to primary PCI	**<120 minutes** If this time target cannot be met, consider fibrinolysis
STEMI diagnosis to primary PCI if the patient's first medical contact is at a hospital	**<60 minutes** if the patient presents to or is in a PCI-capable hospital **<90 minutes** if the patient presents to or is in a non-PCI-capable hospital and needs transferring
STEMI diagnosis to administration of a bolus/infusion of a fibrinolytic drug (if primary PCI cannot be accessed within 120 minutes)	**<10 minutes**
Start of fibrinolysis to ECG assessment of its success or failure	**60-90 minutes**
If fibrinolysis is successful, time interval from starting fibrinolysis to early coronary angiography	**2-24 hours**

Antithrombotic co-therapy for primary PCI

Choice of antiplatelet therapy for patients undergoing primary PCI

Give any patient who will undergo primary percutaneous coronary intervention (PCI) dual antiplatelet therapy with **a P2Y $_{12}$ inhibitor plus aspirin**.[75]

- After the initial aspirin loading dose, reduce the aspirin to a lower daily maintenance dose.
- Check your local protocol when deciding which P2Y $_{12}$ inhibitor to use, the timing of this, and the recommended initial loading and ongoing daily doses.

The UK National Institute for Health and Care Excellence (NICE) recommends the following:[75]

- Prasugrel, in combination with aspirin, if the patient is not already taking an oral anticoagulant
    - For patients aged 75 years and over, the bleeding risk of using prasugrel needs to be weighed up against its effectiveness. If the bleeding risk from prasugrel is a concern in these patients, ticagrelor or clopidogrel may be used as alternatives

- Clopidogrel, in combination with aspirin, if the patient is taking an oral anticoagulant.

The European Society of Cardiology guideline recommends prasugrel or ticagrelor (in combination with aspirin) for patients undergoing primary PCI. Prasugrel should be considered in preference to ticagrelor, but clopidogrel is only indicated when neither of these drugs is available or they are contraindicated.[2]

Cangrelor is a reversible intravenous $P2Y_{12}$ inhibitor that can be considered if the patient is unable to ingest an oral drug.[2] NICE has yet to make any recommendation on the use of cangrelor.

Always follow your local protocol for $P2Y_{12}$ inhibitor selection and timing.

Practical tip

Do not use ticagrelor in patients who:

- Have active bleeding or a history of intracranial haemorrhage.

Prasugrel is:

- **Contraindicated** in patients with active bleeding or a history of previous stroke or transient ischaemic attack
- **Not generally recommended** in patients aged ≥75 years or those with body weight <60 kg[2] [75]
    - If prasugrel is recommended by the interventional cardiology team for these patient groups, then a lower maintenance dose is advised.[2] [184]

Do not give a glycoprotein IIb/IIIa inhibitor or fibrinolytics prior to primary PCI. [75]

- Glycoprotein IIb/IIIa inhibitors (e.g., eptifibatide, tirofiban, abciximab) may be used by the interventional cardiology team during the PCI procedure to tackle a high thrombus burden or no-reflow phenomenon. Abciximab is not currently available in the UK, and shortages in other countries have been reported.

Evidence: Choice of P2Y $_{12}$ inhibitor for patients undergoing PCI

Third-generation P2Y $_{12}$ inhibitors prasugrel and ticagrelor are preferred to clopidogrel as part of a dual antiplatelet therapy regimen with aspirin for the treatment of STEMI in patients undergoing primary PCI who are not already on oral anticoagulation.

In November 2020 the UK National Institute for Health and Care Excellence (NICE) updated its acute coronary syndromes guidance. This included a network meta-analysis of dual antiplatelet therapy that found prasugrel and ticagrelor were more effective than clopidogrel at both 30 days and 1 year. Although there was some uncertainty at 30 days, prasugrel was more effective than ticagrelor at 1 year, especially for reducing all-cause mortality and reinfarction.[75] These results were driven by 3 large trials, as follows.

- In the TRITON-TIMI 38 trial, 13,608 acute coronary syndrome (ACS) patients awaiting PCI were randomised to loading and maintenance doses of prasugrel versus clopidogrel.
    - There was a significant reduction in favour of prasugrel in the combined primary end point of cardiovascular mortality, non-fatal MI, or non-fatal stroke (9.9% vs. 12.1%; hazard ratio [HR] 0.81, 95% CI 0.73 to 0.90; P <0.001).[185]
- The PLATelet inhibition and patient Outcomes (PLATO) trial randomised 18,624 patients admitted to hospital with an ACS (with or without ST-segment elevation) to loading and maintenance doses of ticagrelor versus clopidogrel.
    - There was a significant reduction in favour of ticagrelor in the 12-month primary efficacy end point of cardiovascular death, MI, or stroke (9.8% vs. 11.7%; HR 0.84, 95% CI 0.77 to 0.92; P <0.001).
    - Of note, the ticagrelor group also had significantly lower all-cause mortality (4.5% vs. 5.9%, P <0.001).[186]
- The ISAR-REACT 5 trial, in which 4018 ACS patients awaiting PCI were randomised to ticagrelor versus prasugrel.
    - There was a significant reduction in favour of prasugrel in the 12-month primary efficacy end point of all-cause mortality, MI, or stroke (9.3% with ticagrelor vs. 6.9% with prasugrel; HR 1.36, 95% CI 1.09 to 1.70; P = 0.006).[187]

The faster onset of action and greater antiplatelet potency of prasugrel and ticagrelor (compared with clopidogrel) means they carry a greater risk of bleeding. Therefore, clopidogrel is preferred in people already taking an oral anticoagulant.

- In the TRITON-TIMI trial, the prasugrel group had a higher risk of life-threatening bleeding versus the clopidogrel group (1.4% vs. 0.9%; P = 0.01).[185]
- In the PLATO trial, ticagrelor was associated with a higher rate of major bleeding unrelated to coronary artery bypass grafting compared with clopidogrel (4.5% vs. 3.8%).[186]
- In the ISAR-REACT 5 trial there was no significant difference in major bleeding between ticagrelor versus prasugrel (HR 1.12, 95% CI 0.83 to 1.51).[187]

The evidence is less certain for people aged 75 years and over and a reduced dose of prasugrel or alternative treatment should be considered on an individual basis.

- NICE noted that the TRITON-TIMI trial adverse events, including major bleeding, were more common with prasugrel than with clopidogrel in people aged 75 years and over. Subsequent trials used a reduced dose of prasugrel in this age group and did not find an increased risk of bleeding.
    - NICE also stated that more research is required comparing the efficacy of prasugrel, ticagrelor, and clopidogrel in people aged 75 years and over.[75]

The European Society of Cardiology guideline recommends prasugrel or ticagrelor (with preference for prasugrel) in combination with aspirin for patients undergoing primary PCI, with clopidogrel only indicated when neither of these drugs is available or they are contraindicated.[2]

Choice of anticoagulant therapy for patients undergoing primary PCI

Anticoagulation is routinely given during primary PCI.

- This will be **started by the interventional cardiology team** in the cardiac catheterisation laboratory.
    - Therefore, **do not start anticoagulation** if your patient is likely to be eligible for primary PCI.

Unfractionated heparin is recommended first line for intravenous anticoagulation.[2] [75] Alternatives include:

- Bivalirudin (a direct thrombin inhibitor) for patients with a history of heparin-induced thrombocytopenia
- Enoxaparin.

Fondaparinux is **not recommended** as an adjunctive anticoagulant during primary PCI.[2]

- It was associated with catheter thrombosis at the time of primary PCI in the OASIS-6 trial.[188]

Routine post-procedural anticoagulation is not required after primary PCI unless there is a separate indication for it, for example:[188]

- Full-dose anticoagulation:
    - Atrial fibrillation
    - Mechanical heart valve
    - Left ventricular thrombus
- Prophylactic-dose anticoagulation:

- Patients at high risk of venous thromboembolism.

Choice of fibrinolytic drug

If the delay to primary percutaneous coronary intervention (PCI) means fibrinolysis is selected as the most appropriate reperfusion strategy, **check your local protocols regarding the choice of fibrinolytic drug.**

The UK National Institute for Health and Care Excellence recommends a fibrin-specific drug such as **alteplase or tenecteplase**.[152] It also recommends **streptokinase** (a non-fibrin-specific drug) as an option in some patients.[152]

The European Society of Cardiology guideline recommends the same drugs plus **reteplase** (no longer available in the UK).[2] According to these guidelines, you should consider a half-dose of tenecteplase if the patient is aged 75 years or older.[2]

When choosing a fibrinolytic drug take account of:[152]

- Balancing the benefit and harm (e.g., stroke risk) of each drug for the individual patient
- The importance of avoiding giving streptokinase to a patient who has received it in the past (patients treated with streptokinase may develop antibodies that neutralise the drug if repeat treatment is given)
- Your local hospital protocol for reducing delays in the administration of thrombolysis.

To shorten the time to treatment, **fibrinolysis can be administered as part of the pre-hospital management** of STEMI.[2] [152]

- This may be part of the emergency care treatment pathway where geographical considerations mean the anticipated time to primary PCI will be >120 minutes.
- In such cases, **a bolus dose of tenecteplase** (**or reteplase** if available) **is preferred** for the sake of practicality as the other fibrinolytic drug options are administered by intravenous infusion.

Transfer to a PCI-capable hospital is indicated in all patients **immediately after administration** of fibrinolysis.

Antithrombotic co-therapy given with fibrinolysis

Antithrombotic co-therapy with fibrinolysis consists of a combination of:

- Dual antiplatelet therapy with a $P2Y_{12}$ inhibitor, in addition to aspirin given in the initial phase
- Parenteral anticoagulation.

Give any patient having fibrinolysis anticoagulation **at the same time** as giving fibrinolysis and **start dual antiplatelet therapy immediately afterwards** (see below). [75]

The choice of parenteral anticoagulation can be: [2]

- **Enoxaparin** – an intravenous bolus dose initially, followed by subcutaneous dosing in patients <75 years of age (patients ≥75 years of age are given subcutaneous dosing only). The European Society of Cardiology (ESC) guideline recommends this as the first-choice anticoagulant for patients undergoing fibrinolysis[2] [189] [190] [191]
- **Unfractionated heparin** – a weight-adjusted intravenous bolus followed by an intravenous infusion, recommended by ESC when enoxaparin is not available
- **Fondaparinux (only if streptokinase is used for fibrinolysis)** – an intravenous dose initially, followed by the first subcutaneous dose given 24 hours later.[192]

Ensure anticoagulation co-therapy for any patient undergoing fibrinolysis continues:[2] [71]

- Up to the point of coronary revascularisation with percutaneous coronary intervention (PCI; if performed), or
- For the duration of the hospital stay to a maximum of 8 days in total if revascularisation is not performed.[2]

If a pharmaco-invasive strategy is adopted (fibrinolysis followed by angiography ± PCI), then one regimen that is supported by robust data is composed of:[2] [171] [176] [193] [194]

- Fibrinolysis with intravenous tenecteplase, plus
- Oral aspirin and clopidogrel, plus
- Intravenous enoxaparin at the time of fibrinolysis, plus
- Subcutaneous enoxaparin until the time of revascularisation by PCI.

Give the patient **dual antiplatelet therapy with a $P2Y_{12}$ inhibitor plus aspirin immediately after giving fibrinolysis**.[75]

- After the initial aspirin loading dose, reduce the aspirin to a lower daily maintenance dose.
- Check your local protocol when deciding which $P2Y_{12}$ inhibitor to use, the timing of this, and the recommended initial loading and ongoing daily doses.

The UK National Institute for Health and Care Excellence recommends the following:[75]

- Ticagrelor, in combination with aspirin, unless the patient has a high bleeding risk
- Clopidogrel, in combination with aspirin, or aspirin alone for patients with a high bleeding risk.

The ESC guideline recommends clopidogrel (in combination with aspirin) as the preferred agent.[2]

Evidence: Choice of P2Y $_{12}$ inhibitor for patients not undergoing PCI

Ticagrelor, a third-generation P2Y $_{12}$ inhibitor, is preferred to clopidogrel as part of a dual antiplatelet therapy regimen with aspirin for the treatment of STEMI in patients who are not undergoing PCI.

In November 2020 the UK National Institute for Health and Care Excellence (NICE) updated its acute coronary syndromes guidance. This included a network meta-analysis of dual antiplatelet therapy.[75]

- As prasugrel is only licensed for patients undergoing primary PCI the guideline committee considered the evidence for ticagrelor versus clopidogrel for patients not undergoing PCI.
- Most of the evidence was from patients with unstable angina or non-STEMI, as the majority of people with STEMI are managed with PCI; however, this mostly indirect population was felt to be appropriate due to similarities in the basic pathophysiology and the consistent benefits of ticagrelor over clopidogrel in all patients not undergoing PCI.
- Ticagrelor reduced reinfarction at 30 days and 1 year. There was some uncertainty around mortality at 30 days but ticagrelor reduced all-cause and cardiac mortality at 1 year.
    - Ticagrelor also reduced the need for revascularisation at 30 days and 1 year, and stent thrombosis events at 1 year.
    - Overall, the evidence did not show a clinically important harm with ticagrelor over clopidogrel in terms of bleeding (major or minor) at 30 days or 1 year.
    - There was a possible increased risk of stroke with ticagrelor compared with clopidogrel, but the absolute difference in number of events was small and the confidence intervals were wide.
    - Breathing adverse events (e.g., shortness of breath) were more common with ticagrelor at 30 days and 1 year. However, the guideline committee considered these to be mostly reversible.
- NICE reported a cost-effectiveness analysis study that showed in medically managed people ticagrelor was cost-effective compared with clopidogrel.

The faster onset of action and greater antiplatelet potency of ticagrelor compared with clopidogrel means it may carry a greater risk of bleeding.

- In the PLATelet inhibition and patient Outcomes (PLATO) trial, ticagrelor was associated with a higher rate of major bleeding unrelated to coronary artery bypass grafting compared with clopidogrel (4.5% vs. 3.8%).[186]
- The NICE guideline committee noted that most other studies excluded older people or other groups of people at higher risk of bleeding, and was concerned about the trade off between benefit and harm in these subgroups.
    - Therefore, the committee agreed that clopidogrel plus aspirin or aspirin alone may be more appropriate for people with a higher risk of bleeding.[75]

Patients not receiving immediate coronary reperfusion therapy

Offer conservative medical management to any patient who is ineligible for any reperfusion therapy.[75]

Ensure the patient still receives dual antiplatelet therapy, anticoagulation, and all appropriate secondary prevention therapies.[2] [75]

Check your local protocol when deciding which $P2Y_{12}$ inhibitor to use and the timing of this.

The UK National Institute for Health and Care Excellence recommends the following options for dual antiplatelet therapy in patients who are not treated with percutaneous coronary intervention:[75]

- Ticagrelor, in combination with aspirin, unless the patient has a high bleeding risk
- Clopidogrel, in combination with aspirin, or aspirin alone for patients with a high bleeding risk.

Arrange echocardiography assessment of left ventricular ejection fraction for all STEMI patients.[75]

Long-term management

Ensure all patients are given the following (while taking into account any contraindications).

- Dual antiplatelet therapy with aspirin and a $P2Y_{12}$ inhibitor (unless the patient has a separate indication for anticoagulation - seek specialist advice).[75] **Continue the $P2Y_{12}$ inhibitor** used in the acute phase for up to 12 months (unless contraindicated).[75] The National Institute for Health and Care Excellence (NICE) in the UK recommends:[75]
    - For patients who had undergone percutaneous coronary intervention (PCI):
        - Prasugrel in those not taking an oral anticoagulant. However, follow your local protocol. Consider ticagrelor or clopidogrel in patients aged 75 years or older if the bleeding risk from prasugrel is a concern.
        - Clopidogrel in those taking an oral anticoagulant.
    - For patients who had fibrinolysis:
        - Ticagrelor unless the patient has a high bleeding risk
        - Clopidogrel or aspirin alone if the patient has a high bleeding risk.
- The European Society of Cardiology recommends 12 months of dual antiplatelet therapy as the default strategy, although alternate regimes can be considered in certain circumstances depending on bleeding and ischaemic risks:[2] [195]
    - Single antiplatelet therapy (preferably with a $P2Y_{12}$ receptor inhibitor, e.g., ticagrelor) for patients who are event-free **after 3-6 months** of dual antiplatelet therapy, and who are not at high ischaemic risk[196] [197] [198] [199] [200] [201]
    - Aspirin or $P2Y_{12}$ receptor inhibitor monotherapy **after 1 month** of dual antiplatelet therapy in patients with high bleeding risk.[202]

- De-escalation of antiplatelet therapy in the first 30 days is not recommended. However, beyond 30 days after an acute coronary syndrome (ACS), de-escalation of $P2Y_{12}$ receptor inhibitor therapy may be considered as an alternative strategy in those at high bleeding risk.[2] Theoretically, by identifying patients who are unlikely to have a response to clopidogrel by genetic/platelet function testing, there is potential for personalised antiplatelet therapy.[203] However, it is unclear whether de-escalation guided by platelet function testing or genetic testing improves clinical management and outcomes, and such a strategy based on platelet function testing or genetic testing should be prospectively tested in patients who may benefit from de-escalating antithrombotic therapy.[2]

Continue aspirin indefinitely unless the patient has hypersensitivity.

Practical tip

Evidence on the selection and duration of antiplatelet therapy following coronary revascularisation is evolving rapidly, with studies showing potential benefits from different strategies.[204] [205]
In the UK, you should check the patient's clinical report for an individualised plan written by the interventional cardiology team. This should specify what antiplatelet therapy is advised for long-term management, based on current evidence, individual patient factors, and the specific interventions undertaken for each patient.

More info: Antiplatelet therapy for patients with a separate indication for anticoagulation

Seek specialist advice when deciding the duration and type (dual or single) of antiplatelet therapy in the 12 months after STEMI for patients with a separate indication for anticoagulation (e.g., in patients with ongoing atrial fibrillation).

The National Institute for Health and Care Excellence (NICE) in the UK recommends taking account of all of the following when deciding about the duration and type (dual or single) of antiplatelet therapy in patients with a separate indication for anticoagulation:[75]

- Bleeding risk
- Thromboembolic risk
- Cardiovascular risk
- Patient's wishes.

Be aware that long-term continuation of aspirin, clopidogrel, and oral anticoagulation (triple therapy) significantly increases bleeding risk.

For patients **already on anticoagulation** who **had PCI**:[75]

- Continue anticoagulation and clopidogrel for up to 12 months
- If the patient is taking a direct oral anticoagulant, adjust the dose according to bleeding risk, thromboembolic risk, and cardiovascular risk.

For patients with a **new indication for anticoagulation** who **had PCI**:[75]

- Offer clopidogrel (to replace prasugrel or ticagrelor) for up to 12 months and an oral anticoagulant licensed for the indication, which best matches the patient's bleeding risk, thromboembolic risk, cardiovascular risk, and wishes.

For patients already on anticoagulation, or those with a new indication, who did **not have PCI**:[75]

- Continue anticoagulation and, unless there is a high risk of bleeding, consider continuing aspirin (or clopidogrel for patients with contraindication for aspirin) for up to 12 months.

Do not routinely offer prasugrel or ticagrelor in combination with anticoagulation in patients with a separate indication for anticoagulation.[75]

- An ACE inhibitor
  - Start this as soon as the patient is haemodynamically stable and continue it indefinitely. Complete upwards dose titration within 4-6 weeks of hospital discharge.[75]
  - Offer an angiotensin-II receptor antagonist as an alternative if the patient is intolerant to an ACE inhibitor.[75]
  - Measure renal function, serum electrolytes, and blood pressure before starting an ACE inhibitor or angiotensin-II receptor antagonist.[75] In practice, if the patient has abnormal renal function or blood pressure, start with a low dose and titrate this carefully with close monitoring.

- A beta-blocker
    - Start this as soon as the patient is haemodynamically stable.[75]
    - Continue the beta-blocker for at least 12 months if the patient does not have reduced left ventricular ejection fraction (LVEF).[75] [206]
    - Continue the beta-blocker indefinitely if the patient has reduced LVEF.[75]
    - Evidence shows that a beta-blocker may reduce the short-term risk of a reinfarction and the long-term risk of all-cause mortality and cardiovascular mortality in patients with acute myocardial infarction.[155] [207] [206] [208]

- High-intensity statin therapy.[45] [63] [75]
    - Non-adherence to statin therapy and failure to achieve lipid targets is associated with an increased cardiovascular mortality following acute MI.[209] Patients should be counselled on the importance of medication adherence.
    - Target low-density lipoprotein (LDL)-cholesterol is <1.4 mmol/L (<55 mg/dL) and a ≥50% LDL-cholesterol reduction from baseline. If these targets are not achieved on maximal statin therapy, add ezetimibe.[2] [63]
    - A proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitor monoclonal antibody (e.g., evolocumab, alirocumab) may be added to statin and ezetimibe therapy if LDL-cholesterol targets are not achieved despite maximal statin and ezetimibe therapy.[2] [63] [210] [211] [212] Treatment can be started during ACS admission or at outpatient follow-up 4-6 weeks later.

Give an aldosterone antagonist (e.g., eplerenone, spironolactone) to any patient with signs or symptoms of heart failure and reduced LVEF.[75]

- Start this within 3-14 days of a STEMI and preferably after starting an ACE inhibitor.[75]

Give a sodium-glucose cotransporter-2 (SGLT2) inhibitor (e.g., dapagliflozin, empagliflozin) to patients with heart failure when they are clinically stable, regardless of their LVEF.[213] [214] [215]

Calcium-channel blockers and potassium-channel activators

- Do not routinely give calcium-channel blockers.[75]
- Consider a non-dihydropyridine calcium-channel blocker, such as verapamil, in a patient without pulmonary congestion or reduced LVEF who has a contraindication to beta-blockers (or when these need to be discontinued).[75]
- Do not offer nicorandil (a potassium-channel activator).

Offer cardiac rehabilitation to all patients. This should include an exercise component, health education, stress management, and psychological and social support. Advise all patients on lifestyle changes such as:[2] [75]

- Changes to diet
- Reduction of alcohol consumption
- Smoking cessation
- Weight management

- Physical exercise
- Reduced sedentary time.

Patients without any pre-existing risk factors for cardiovascular disease are at increased risk of early mortality; even patients who are deemed low risk require prompt initiation of evidence-based pharmacotherapy post ACS.[216]

Treatment algorithm overview

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Acute (summary)

suspected or clinical diagnosis of STEMI (symptoms of myocardial ischaemia + ST elevation on ECG)

	1st	aspirin
	plus	assess eligibility for coronary reperfusion therapy
	plus	analgesia
	plus	anti-emetic
	consider	oxygen
	consider	intravenous nitrate
<12 hours since symptom onset: primary PCI available within 120 minutes	plus	primary PCI
	plus	P2Y12 inhibitor
	plus	parenteral anticoagulation
	consider	glycoprotein IIb/IIIa inhibitor
<12 hours since symptom onset: primary PCI NOT available within 120 minutes of the time when fibrinolysis could be given AND patient is eligible for fibrinolysis	plus	fibrinolysis
	plus	parenteral anticoagulation
	plus	P2Y12 inhibitor
	plus	angiography with or without PCI
<12 hours since symptom onset: primary PCI NOT available within 120 minutes AND patient has absolute contraindication to fibrinolysis	plus	consult interventional cardiology team to discuss primary PCI
	plus	P2Y12 inhibitor
>12 hours since symptom onset: with ongoing	plus	primary PCI

Acute (summary)

evidence of myocardial ischaemia (symptoms and/or signs on ECG)		
	plus	P2Y12 inhibitor
	plus	parenteral anticoagulation
	consider	glycoprotein IIb/IIIa inhibitor
>12 hours since symptom onset: no ongoing evidence of myocardial ischaemia (no symptoms and/or signs on ECG)	plus	seek immediate cardiology advice to discuss management options
	plus	P2Y12 inhibitor

Ongoing (summary)

post-STEMI

1st	continue dual antiplatelet therapy
plus	start or continue beta-blocker or non-dihydropyridine calcium-channel blocker
plus	start or continue ACE inhibitor or angiotensin-II receptor antagonist
plus	statin
consider	ezetimibe
consider	proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitor
consider	aldosterone antagonist
consider	sodium-glucose co-transporter-2 (SGLT2) inhibitor
plus	cardiac rehabilitation

Treatment algorithm

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Acute

suspected or clinical diagnosis of STEMI (symptoms of myocardial ischaemia + ST elevation on ECG)

1st **aspirin**

Primary options

» aspirin: 300 mg orally (chewed or dispersed in water) as a loading dose, followed by 75-100 mg once daily thereafter
The loading and maintenance dose varies across guidelines and locations; check local protocols for further guidance on dose.

» Give all patients with suspected STEMI a single loading dose of aspirin as soon as possible, unless they have aspirin hypersensitivity.[2] [75]

- Aspirin can be given orally (or via nasogastric tube if oral ingestion is not possible). An intravenous loading dose is used in some countries; however, this formulation is not available in the UK.

»Check your local protocol or discuss the patient with a senior colleague if they have hypersensitivity to aspirin.

» Start treatment for STEMI **immediately** after making a clinical working diagnosis - **do not wait for cardiac troponin levels to confirm it.** [2]

» After the loading dose, continue with a lower maintenance dose of aspirin as part of ongoing dual antiplatelet therapy unless contraindicated.

plus **assess eligibility for coronary reperfusion therapy**

Treatment recommended for ALL patients in selected patient group

» Immediately assess the patient's **eligibility for coronary reperfusion therapy** as soon as a clinical diagnosis of STEMI has been made.[2] [75]

Acute

- If eligible, take steps to ensure coronary reperfusion therapy (primary percutaneous coronary intervention or fibrinolysis) is delivered as **quickly as possible**. If not eligible, offer conservative medical management.[75]
- Do not allow the patient's age, ethnicity, or sex to influence your assessment of their suitability for reperfusion therapy.[75] Evidence suggests that women tend to receive reperfusion therapy less frequently and/or in a more delayed fashion than men, and are less likely to receive cardiac rehabilitations and secondary prevention medications.[2]

» For most patients with STEMI, **primary percutaneous coronary intervention (PCI) is the preferred reperfusion strategy** provided it can be delivered within 120 minutes of the time when fibrinolysis could have been given.[2] [75] [81]

- Multiple randomised controlled trials have shown that **primary PCI has better outcomes** (in terms of reducing mortality, reinfarction, or stroke) **and less intracranial bleeding than fibrinolysis** provided it can be delivered in a timely manner by an experienced team.[2][166] [167] [168] [169] [170] [171]
- However, **fibrinolysis may be more appropriate when there is** a lack of access to timely PCI.[2] [75]

Acute

Practical tip

The European Society of Cardiology (ESC) **acute coronary syndrome** guideline lists the contraindications to fibrinolytic therapy as follows:[2]

- **Absolute contraindications**
  - History of intracranial haemorrhage or stroke of unknown origin at any time
  - Ischaemic stroke in the last 6 months
  - Central nervous system damage, neoplasm, or arteriovenous malformation
  - Major trauma/surgery/head injury within the last 1 month
  - Gastrointestinal bleeding within the last 1 month
  - Bleeding disorder
  - Aortic dissection
  - Non-compressible punctures within the last 24 hours (e.g., liver biopsy, lumbar puncture)
- **Relative contraindications**
  - Transient ischaemic attack in the last 6 months
  - Oral anticoagulant therapy
  - Pregnancy or within 1 week postnatal
  - Refractory hypertension (systolic BP >180 mmHg and/or diastolic BP >110 mmHg)
  - Advanced liver disease
  - Infective endocarditis
  - Active peptic ulcer
  - Prolonged or traumatic cardiopulmonary resuscitation.

If primary PCI is not available within 120 minutes but your patient has a contraindication to fibrinolysis:

- Seek immediate advice from the interventional cardiology team
- For a patient with an absolute contraindication to fibrinolysis, primary PCI will normally be the preferred management strategy despite the delay in accessing it.

Acute

» **Seek immediate specialist input** from the **interventional cardiology team**.[2]

- If you are managing the patient within a PCI-capable hospital, this team will be available on site. If not, call the interventional cardiology team at your designated PCI-capable hospital to discuss **immediate transfer** for coronary reperfusion therapy.
- Also take the earliest opportunity to gain **intravenous access** and start **continuous haemodynamic monitoring** and **pulse oximetry**.[2]

Practical tip

When placing an intravenous cannula, avoid placing it in the right hand or right wrist area as the right radial artery is the usual route used to perform primary PCI.

» Concurrent anticoagulation and dual antiplatelet therapy is given alongside coronary reperfusion, regardless of whether primary PCI or fibrinolysis is used. The benefits of reperfusion in reducing mortality and improving myocardial salvage decline rapidly with time.[2] [75]

- It is, therefore, vital to take every step possible to ensure the chosen reperfusion strategy (primary PCI or fibrinolysis) is delivered as quickly as possible.

Practical tip

Do not give anticoagulation therapy if the patient is likely to be eligible for primary PCI. [2] [71]

- This will be given by the interventional cardiology team in the cardiac catheterisation laboratory.

» **The ESC acute coronary syndrome guideline has set widely accepted maximum time-to-treatment targets as follows:** [2]

Acute

Interval	Target
First medical contact to ECG and STEMI diagnosis	**<10 minutes**
STEMI diagnosis to primary PCI	**<120 minutes** If this time target cannot be met, consider fibrinolysis
STEMI diagnosis to primary PCI if the patient's first medical contact is at a hospital	**<60 minutes** if the patient presents to or is in a PCI-capable hospital **<90 minutes** if the patient presents to or is in a non-PCI-capable hospital and needs transferring
STEMI diagnosis to administration of a bolus/infusion of a fibrinolytic drug (if primary PCI cannot be accessed within 120 minutes)	**<10 minutes**
Start of fibrinolysis to ECG assessment of its success or failure	**60-90 minutes**
If fibrinolysis is successful, time interval from starting fibrinolysis to early coronary angiography	**2-24 hours**

» The most appropriate coronary reperfusion strategy will depend on:[2] [71] [74]

- Duration of ischaemic symptoms
- Persistence or resolution of ST-segment elevation on serial ECGs
- Availability of timely access to primary PCI

Acute

- Whether a patient is first assessed in a PCI-capable or non-PCI-capable hospital
- Estimated transfer time from a non-PCI-capable to a PCI-capable hospital

- Whether there was a pre-hospital STEMI diagnosis made by trained and fully equipped paramedic team who can administer initial pharmacotherapy
- The quality and expertise of the regional network infrastructure in place for STEMI management.

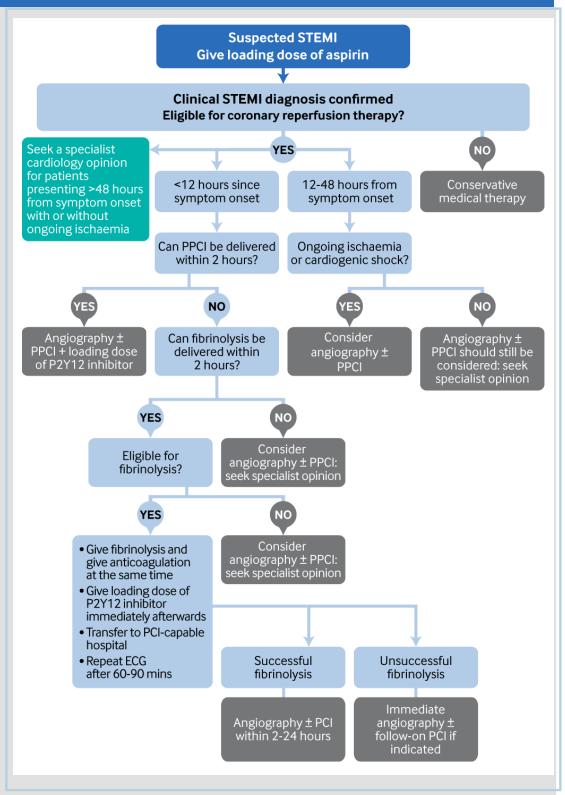
» Many patients who present with STEMI are already taking long-term oral anticoagulation for various indications.[2]

- This is a **relative contraindication** for **fibrinolysis**.
- For any such patient, **choose a primary PCI strategy** for coronary reperfusion therapy, regardless of the anticipated time to access this intervention.[2]

More info: Summary flowchart – choice of coronary reperfusion strategy

The flowchart below summarises the choice of coronary reperfusion strategy according to time since the patient's symptom onset and the anticipated time to access primary PCI.[2] [75]

Acute



Selection of the most appropriate reperfusion strategy. PPCI, primary percutaneous coronary intervention
Created by the BMJ Knowledge Centre

plus **analgesia**

Treatment recommended for ALL patients in selected patient group

Primary options

» morphine sulfate: 2.5 to 10 mg intravenously initially, followed by 2.5 to 10 mg if required (at a rate of 1-2 mg/minute)
The lower end of the dose range is recommended in elderly and frail patients.

OR

» diamorphine: 2.5 to 5 mg intravenously initially, followed by 1.25 to 5 mg if required (at a rate of 1-2 mg/minute)
The lower end of the dose range is recommended in elderly and frail patients.

» **Give pain relief.** [2] [82]

- Titrated **intravenous opioids** (e.g., morphine or diamorphine) are the most commonly used option for analgesia.

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- Pain relief is important not just for the comfort of the patient but also because it may reduce myocardial and microvascular damage due to reduction of heart rate, cardiac workload, and oxygen consumption.[2]

plus **anti-emetic**

Treatment recommended for ALL patients in selected patient group

Primary options

» ondansetron: 4-8 mg intravenously as a single dose

OR

» metoclopramide: body weight <60 kg: up to 500 micrograms/kg/day intravenously given in 3 divided doses; body weight ≥60 kg: 10 mg intravenously up to three times daily

OR

» cyclizine: 50 mg intravenously three times daily

» Give an anti-emetic concomitantly with the opioid analgesic to prevent the patient vomiting the oral loading dose of dual antiplatelet therapy.

consider **oxygen**

Treatment recommended for SOME patients in selected patient group

» Give oxygen therapy only if **saturations are <90%** on pulse oximetry.[2]

- Routine supplemental oxygen is not indicated if arterial oxygen saturation (SaO_2) ≥90%.[156] [157] [158]

consider **intravenous nitrate**

Treatment recommended for SOME patients in selected patient group

Primary options

» glyceryl trinitrate: 15-20 micrograms/minute intravenous infusion initially, adjust dose according to response, maximum 400 micrograms/minute

OR

Acute

» isosorbide dinitrate: 2-10 mg/hour intravenous infusion initially, adjust dose according to response, maximum 20 mg/hour

» Routine use of an intravenous nitrate in STEMI is not recommended as there is no evidence to support its benefits.[2]

- In practice, a sublingual (or buccal) dose is often given after a STEMI diagnosis has been made (and sometimes before the patient arrives at hospital).

Practical tip

If your patient experiences a spontaneous resolution of ST-segment elevation, along with complete symptom relief, after taking glyceryl trinitrate, this is suggestive of coronary spasm with or without associated MI. [2]

- Refer to cardiology.
- Aim for early coronary angiography (within 24 hours).

» **Consider an intravenous nitrate** if the patient has:[2] [82]

- **Sustained hypertension**
- Clinical and/or radiographic evidence of **congestive heart failure**
- **Persistent chest pain (residual angina) despite administration of sublingual (or buccal) glyceryl trinitrate** (which the patient may have received before arriving at hospital).

» **Titrate the rate of infusion** according to the patient's blood pressure and wider clinical response.

» **Do not give an intravenous nitrate when there is hypotension or right ventricular infarction.**

Acute

Practical tip

Administration of intravenous nitrate should not delay transfer to the catheterisation laboratory.

- An intravenous nitrate can be given in the catheterisation laboratory by the interventional cardiology team, if needed.
- Recanalisation of the occluded infarct artery is the most effective way of relieving refractory chest pain so the priority is to get the patient to primary percutaneous coronary intervention as quickly as possible.

<12 hours since symptom onset: primary PCI available within 120 minutes

plus

primary PCI

Treatment recommended for ALL patients in selected patient group

» Select primary percutaneous coronary intervention (PCI) as the **preferred reperfusion strategy** for any patient who had **symptom onset <12 hours ago and has**:[2] [75] [81] [166] [167] [168] [169] [172]

- **Ongoing signs and symptoms** of myocardial ischaemia, **AND**
- **Persistent (or increasing) ST-segment elevation** on ECG.

» Take steps to ensure that primary PCI will be delivered **within 120 minutes of the time when fibrinolysis could have been given.** [75] [81]

» Primary PCI involves **immediate transfer to the catheterisation laboratory** with the intention of opening the artery with stent placement. Drug-eluting stents are recommended by the European Society of Cardiology and the UK National Institute for Health and Care Excellence.[2] [75]

- Many hospitals have **round-the-clock PCI capability**. If yours does not, then arrange **immediate transfer** to your **designated PCI-capable hospital**.
- If it is **not possible** to ensure primary PCI **within 120 minutes, offer fibrinolysis** (if not contraindicated).[75]
- If your patient has **STEMI with cardiogenic shock, seek urgent**

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senior support. Coronary angiography ± primary PCI is indicated.[2] [75]

- If the patient's coronary anatomy is unsuitable for PCI, or PCI fails, emergency coronary artery bypass graft (**CABG**) is recommended.[2]
- For more information on supportive management, see Shock.

plus **P2Y12 inhibitor**

Treatment recommended for ALL patients in selected patient group

Primary options

» prasugrel: <75 years of age and body weight <60 kg: 60 mg orally as a loading dose, followed by 5 mg once daily thereafter; <75 years of age and body weight ≥60 kg: 60 mg orally as a loading dose, followed by 10 mg once daily thereafter; ≥75 years of age: 60 mg orally as a loading dose, followed by 5 mg once daily thereafter

OR

» clopidogrel: <75 years of age: 300-600 mg orally as a loading dose, followed by 75 mg once daily thereafter; ≥75 years of age: 75 mg orally once daily
The licensed loading dose in the UK is 300 mg. The ESC guideline recommends a higher loading dose of 600 mg for patients proceeding to PCI. While this higher dose is not licensed in the UK, it is widely used in practice.[2]

OR

» ticagrelor: 180 mg orally as a loading dose, followed by 90 mg twice daily thereafter

» Give any patient who will undergo primary percutaneous coronary intervention (PCI) dual antiplatelet therapy with **a P2Y $_{12}$ inhibitor plus aspirin**.[75]

- After the initial aspirin loading dose, reduce the aspirin to a lower daily maintenance dose.
- Check your local protocol when deciding which P2Y $_{12}$ inhibitor to use, the timing of this and the recommended initial loading and ongoing daily doses.

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» The UK National Institute for Health and Care Excellence (NICE) recommends the following for any patient undergoing PCI:[75]

- Prasugrel, in combination with aspirin, if the patient is not already taking an oral anticoagulant
  - For patients aged 75 years and older, the bleeding risk of using prasugrel needs to be weighed up against its effectiveness. If the bleeding risk from prasugrel is a concern in these patients, ticagrelor or clopidogrel may be used as alternatives.
- Clopidogrel, in combination with aspirin, if the patient is already taking an oral anticoagulant.

» The European Society of Cardiology guideline recommends prasugrel or ticagrelor (in combination with aspirin) for patients undergoing primary PCI. Prasugrel should be considered in preference to ticagrelor, but clopidogrel is only indicated when neither of these drugs is available or they are contraindicated.[2]

» Cangrelor is a reversible intravenous $P2Y_{12}$ inhibitor that can be considered if the patient is unable to ingest an oral drug.[2] NICE has yet to make any recommendation on the use of cangrelor.

» Always follow your local protocol for $P2Y_{12}$ inhibitor selection and timing.

plus **parenteral anticoagulation**

Treatment recommended for ALL patients in selected patient group

Primary options

» heparin: no glycoprotein IIb/IIIa inhibitor use planned: 70-100 units/kg intravenous bolus; glycoprotein IIb/IIIa inhibitor use planned: 50-70 units/kg intravenous bolus
Monitor activated clotting time (ACT) during procedure. Further doses may be required to maintain ACT.

OR

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» enoxaparin: 0.5 mg/kg intravenous bolus
Some centres may continue with subcutaneous dosing after this initial intravenous dose; consult local protocols for further guidance.

OR

» bivalirudin: 0.75 mg/kg intravenous bolus initially, followed by 1.75 mg/kg/hour intravenous infusion during procedure and for up to 4 hours after procedure, reduce to 0.25 mg/kg/hour for a further 4-12 hours if necessary

» Anticoagulation is routinely given during primary percutaneous coronary intervention (PCI).

- This will be **started by the interventional cardiology team** in the cardiac catheterisation laboratory.
  - Therefore, **do not start anticoagulation** if your patient is likely to be eligible for primary PCI.

» Unfractionated heparin is recommended first line for intravenous anticoagulation.[2] [75] Alternatives include:

- Bivalirudin (a direct thrombin inhibitor) for patients with a history of heparin-induced thrombocytopenia
- Enoxaparin.

» Fondaparinux is **not recommended** as an adjunctive anticoagulant during primary PCI.[2]

- It was associated with catheter thrombosis at the time of primary PCI in the OASIS-6 trial.[188]

» **Routine post-procedural anticoagulation is not required** after primary PCI unless there is a separate indication for it, for example:[2]

- Full-dose anticoagulation:
  - Atrial fibrillation
  - Mechanical heart valve

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- Left ventricular thrombus

- Prophylactic-dose anticoagulation:
  - Patients at high risk of venous thromboembolism

- Consult local protocols for full- and prophylactic-dose regimens for these indications. The doses presented here are the doses used during primary PCI only.

consider **glycoprotein IIb/IIIa inhibitor**

Treatment recommended for SOME patients in selected patient group

Primary options

» eptifibatide: consult specialist for guidance on dose

OR

» tirofiban: consult specialist for guidance on dose

» A glycoprotein IIb/IIIa inhibitor (e.g., eptifibatide, tirofiban, abciximab) may be used by the interventional cardiology team during the percutaneous coronary intervention procedure.[2] Abciximab is not currently available in the UK, and shortages in other countries have been reported.

- This can help to tackle a high thrombus burden or no-reflow phenomenon.

<12 hours since symptom onset: primary PCI NOT available within 120 minutes of the time when fibrinolysis could be given AND patient is eligible for fibrinolysis

plus **fibrinolysis**

Treatment recommended for ALL patients in selected patient group

Primary options

» tenecteplase: body weight <60 kg: 30 mg (6000 units) intravenous bolus as a single dose; body weight 60 to <70 kg: 35 mg (7000 units) intravenous bolus as a single dose; body weight 70 to <80 kg: 40 mg (8000 units) intravenous bolus as a single dose; body weight 80 to <90 kg: 45 mg (9000 units) intravenous bolus as a single dose;

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body weight ≥90 kg: 50 mg (10,000 units) intravenous bolus as a single dose
Dose should be halved in patients ≥75 years of age due to a higher bleeding risk in these patients. Tenecteplase is only licensed in the UK for use within 6 hours of symptom onset.

OR

» alteplase: accelerated regimen (if started within 6 hours of symptom onset): 15 mg intravenous bolus, followed by 0.75 mg/kg (maximum 50 mg/dose) intravenous infusion over 30 minutes, then 0.5 mg/kg (maximum 35 mg/dose) intravenous infusion over 60 minutes, maximum 100 mg total dose over 90 minutes; regular regimen (if started within 6-12 hours of symptom onset): 10 mg intravenous bolus, followed by 50 mg intravenous infusion over 60 minutes, then 10 mg intravenous infusion over 30 minutes for four infusions, maximum 100 mg total dose over 3 hours and maximum 1.5 mg/kg in patients with body weight <65 kg
The ESC guideline recommends the accelerated regimen as the preferred regimen.[2]

Secondary options

» streptokinase: 1.5 million units intravenous infusion given over 30-60 minutes

» Give fibrinolysis (unless contraindicated) **if primary percutaneous coronary intervention (PCI) cannot be delivered within 120 minutes** of the time when fibrinolysis could be given.[75] [81]

» **Start anticoagulation** at the same time as giving fibrinolysis and start dual antiplatelet therapy immediately afterwards.[2] [71] [75]

» Check your local protocols regarding the **choice of fibrinolytic drug.**

- The UK National Institute for Health and Care Excellence recommends a fibrin-specific drug such as **alteplase or tenecteplase**.[152] It also recommends **streptokinase** (a non-fibrin-specific drug) as an option in some patients.[152]
- The European Society of Cardiology (ESC) guideline recommends the same fibrin-specific drugs plus **reteplase** (no longer available in the UK).[2] According

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to these guidelines, consider a half-dose of tenecteplase if the patient is aged 75 years or older.[2]

- When choosing a fibrinolytic drug take account of:[152]
  - Balancing the benefit and harm (e.g., stroke risk) of each drug for the individual patient
  - The importance of avoiding giving streptokinase to a patient who has received it in the past (patients treated with streptokinase may develop antibodies that neutralise the drug if repeat treatment is given)
  - Your local hospital protocol for reducing delays in the administration of thrombolysis.

» To shorten the time to treatment, **fibrinolysis can be administered as part of the pre-hospital management** of STEMI.[2] [152]

- This may be part of the emergency care treatment pathway where geographical considerations mean the anticipated time to primary PCI will be >120 minutes.
- In such cases, a **bolus dose of tenecteplase** (**or reteplase** if available) **is preferred** for the sake of practicality as the other fibrinolytic drug options are administered by intravenous infusion.

» **If your patient is not already at a PCI-capable hospital, transfer them to one immediately after giving fibrinolysis.** [2]

- This is so that rescue PCI can be performed if fibrinolysis fails or angiography ± PCI can be arranged if fibrinolysis is effective.

» If primary PCI **cannot be delivered within 120 minutes of STEMI diagnosis, seek a specialist cardiology opinion** to support your choice of reperfusion strategy.[2] [71] [153]

- The greatest benefit of fibrinolysis is observed when given <2 hours after

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symptom onset. Its clinical **efficacy diminishes as time from symptom onset increases**.[72] [154]

- Therefore, the later the patient presents (particularly >2-3 hours), the more consideration should be given to transferring for **primary PCI instead** – discuss the options with cardiology.[2] [74]

Acute

Practical tip

The ESC acute coronary syndrome guideline lists the contraindications to fibrinolytic therapy as follows:[2]

- **Absolute contraindications**
  - History of intracranial haemorrhage or stroke of unknown origin at any time
  - Ischaemic stroke in the last 6 months
  - Central nervous system damage, neoplasm, or arteriovenous malformation
  - Major trauma/surgery/head injury within the last 1 month
  - Gastrointestinal bleeding within the last 1 month
  - Bleeding disorder
  - Aortic dissection
  - Non-compressible punctures within the last 24 hours (e.g., liver biopsy, lumbar puncture)
- **Relative contraindications**
  - Transient ischaemic attack in the last 6 months
  - Oral anticoagulant therapy
  - Pregnancy or within 1 week postnatal
  - Refractory hypertension (systolic BP >180 mmHg and/or diastolic BP >110 mmHg)
  - Advanced liver disease
  - Infective endocarditis
  - Active peptic ulcer
  - Prolonged or traumatic cardiopulmonary resuscitation.

Evidence: Role of fibrinolysis in STEMI

Fibrinolytic therapy remains an important evidence-based intervention in settings

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where primary PCI cannot be offered in a timely manner.

Several landmark studies demonstrated the benefits of fibrinolysis in patients with STEMI (prior to the advent of primary PCI).

- The **1988 Second International Study of Infarct Survival**(ISIS-2) proved that the benefits of aspirin and fibrinolytics (e.g. streptokinase) were additive and associated with improved 10-year survival following acute MI.[178]
- The **Fibrinolytic Therapy Trialists' Collaborative Group** (1994) demonstrated highly significant benefits in saving lives and helped to establish the time window when fibrinolytic therapy is most effective.[154]
    - The study demonstrated highly significant absolute mortality reductions from fibrinolysis of about:
        - 30 per 1000 for those presenting within 0-6 hours of symptom onset
        - 20 per 1000 for those presenting 7-12 hours after symptom onset.
    - There was a statistically uncertain benefit of about 10 per 1000 for those presenting at 13-18 hours (with more randomised evidence needed in this group to assess reliably the net effects of treatment).
- The 1993 **GUSTO trial** compared different fibrinolysis strategies.[179] [180]
    - It demonstrated a benefit of 14% reduction in mortality (95% CI, 5.9% to 21.3%) for accelerated tissue plasminogen activator

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(t-PA) versus streptokinase (the standard fibrinolytic treatment at the time). Alteplase (recombinant t-PA) was used in the trial.

- The trial randomly assigned 41,021 patients with evolving acute MI to 4 different fibrinolytic strategies. The data for the primary end point of 30-day mortality for each treatment arm was as follows:
  - Streptokinase plus subcutaneous heparin = 7.2%
  - Streptokinase plus intravenous heparin = 7.4%
  - t-PA plus intravenous heparin = 6.3%
  - Streptokinase plus t-PA plus intravenous heparin = 7.0%.
- A significant excess of haemorrhagic strokes was observed for accelerated t-PA (P = 0.03) and for the combination strategy (P <0.001), as compared with streptokinase only.

The 2013 STREAM trial provided further support for fibrinolysis as an appropriate reperfusion strategy for STEMI when primary PCI cannot be delivered in a timely manner – especially if followed by routine early angiography ± PCI (a 'pharmaco-invasive strategy'). [171]

- The STREAM trial was conducted at a time when primary PCI was established as the standard of care for STEMI.

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- In the trial, 1892 STEMI patients presenting within 3 hours of symptom onset but unable to have primary PCI within 1 hour were randomly assigned to:
    - Primary PCI or
    - Pre-hospital bolus tenecteplase (with a half-dose used in patients ≥75 years) plus clopidogrel plus enoxaparin before transport to a PCI-capable hospital
        - Patients had emergency angiography if fibrinolysis failed
        - If fibrinolysis was successful, patients had routine angiography within 6-24 hours after randomisation.
- There was no significant difference between the primary PCI and fibrinolysis groups in the primary composite end point of death, shock, congestive heart failure, or reinfarction within 30 days.
    - More intracranial haemorrhages occurred in the fibrinolysis group.
- **The ESC guideline recommendation is that fibrinolysis should be given within 10 minutes of STEMI diagnosis.** [2]

The extent to which the delay-to-PCI time diminishes the advantages of primary PCI over fibrinolysis has been widely debated and more research is needed. [2]

- It is clear that if delay to treatment is similar, primary PCI is superior

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to fibrinolysis in reducing mortality, reinfarction, or stroke.[2]

- However, fibrinolysis can be administered expeditiously and the longer the delay to primary PCI, the lower the benefits over fibrinolysis. The exact cut-off point at which the delay to access PCI makes fibrinolysis the better reperfusion strategy is based on weak evidence.

Multivariate meta-analyses provide further evidence to support a pharmaco-invasive strategy, suggesting it is safer and more effective than facilitated PCI or fibrinolysis, where primary PCI is not available in a timely fashion.[181]

plus **parenteral anticoagulation**

Treatment recommended for ALL patients in selected patient group

Primary options

» enoxaparin: <75 years of age: 30 mg intravenous bolus, followed by 1 mg/kg subcutaneously every 12 hours until revascularisation or hospital discharge for up to 8 days, maximum 100 mg/dose for each of the first 2 subcutaneous doses; ≥75 years of age: 0.75 mg/kg subcutaneously every 12 hours until revascularisation or hospital discharge for up to 8 days, maximum 75 mg/dose for each of the first 2 subcutaneous doses

OR

» heparin: 60 units/kg (maximum 4000 units) intravenous bolus, followed by 12 units/kg/hour (maximum 1000 units/hour) intravenous infusion for 24-48 hours; adjust dose according to aPTT

Secondary options

» fondaparinux: 2.5 mg intravenously once daily on the first day, followed by 2.5 mg subcutaneously once daily for up to 8 days (or hospital discharge if sooner)

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Only use fondaparinux when streptokinase is used for fibrinolysis.

» **Start anticoagulation** at the same time as fibrinolysis.[2] [71] [75]

» The choice of parenteral anticoagulation can be any one of:[2]

- **Enoxaparin** – the European Society of Cardiology (ESC) guideline recommends this as the first-choice anticoagulant for patients undergoing fibrinolysis[2] [189] [190] [191]
- **Unfractionated heparin** – a weight-adjusted intravenous bolus followed by an intravenous infusion, recommended by ESC when enoxaparin is not available
- **Fondaparinux (only if streptokinase is used for fibrinolysis)** – an intravenous dose initially, followed by the first subcutaneous dose given 24 hours later.[192]

» Ensure anticoagulation for any patient undergoing fibrinolysis continues:[2] [71]

- Up to the point of coronary revascularisation with percutaneous coronary intervention (if performed), or
- For the duration of hospital stay to a maximum of 8 days in total if revascularisation is not performed.[2]

plus **P2Y12 inhibitor**

Treatment recommended for ALL patients in selected patient group

Primary options

» ticagrelor: 180 mg orally as a loading dose, followed by 90 mg twice daily thereafter

OR

» clopidogrel: <75 years of age: 300 mg orally as a loading dose, followed by 75 mg once daily thereafter; ≥75 years of age: 75 mg orally once daily

» Give the patient **dual antiplatelet therapy with a P2Y $_{12}$ inhibitor plus aspirin immediately after giving fibrinolysis**.[75]

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- After the initial aspirin loading dose, reduce the aspirin to a lower daily maintenance dose.
- Check your local protocol when deciding which P2Y $_{12}$ inhibitor to use, the timing of this and the recommended initial loading and ongoing daily doses.

» The UK National Institute for Health and Care Excellence recommends the following:[75]

- Ticagrelor, in combination with aspirin, unless the patient has a high bleeding risk
- Clopidogrel, in combination with aspirin, or aspirin alone for patients with a high bleeding risk.

» The European Society of Cardiology guideline recommends clopidogrel (in combination with aspirin) as the preferred agent.[2]

plus **angiography with or without PCI**

Treatment recommended for ALL patients in selected patient group

» Use an **ECG 60-90 minutes after administering fibrinolysis** to assess whether it has been successful.[2] [75]

» **If fibrinolysis has failed** (<50% ST-segment resolution at 60-90 minutes), offer immediate coronary angiography, with follow-on percutaneous coronary intervention (PCI) if indicated.[2] [75] **Do not repeat fibrinolytic therapy.** [2] [75]

» If the patient has recurrent myocardial ischaemia after fibrinolysis, **seek immediate specialist advice from cardiology**. PCI may be indicated.[75]

» **If fibrinolysis is successful**, consider angiography during the same hospital admission for patients who are clinically stable.[75]

- The European Society of Cardiology (ESC) guidelines recommend arranging **angiography ± PCI** of the infarct-related artery **within 2-24 hours**.[2]
- Several randomised trials and two meta-analyses have shown that early routine angiography (with PCI if needed) after fibrinolysis reduces the rate of reinfarction and recurrent ischaemia compared with a watchful waiting strategy. The

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benefits of early routine PCI following fibrinolysis were seen across patient subgroups and without any increased risk of adverse events such as stroke or major bleeding.[173] [174] [175]

» The ESC guideline recommends **immediate rescue PCI** if, at any time following fibrinolysis, the patient develops **haemodynamic or electrical instability, worsening ischaemia, or persistent chest pain**.[2]

» **Emergency angiography ± PCI** is recommended if, at any time following fibrinolysis, the patient has:

- Heart failure or cardiogenic shock[2]
- Recurrent ischaemia or evidence of re-occlusion after initially successful fibrinolysis.[2] [176] [177]

<12 hours since symptom onset: primary PCI NOT available within 120 minutes AND patient has absolute contraindication to fibrinolysis

plus

consult interventional cardiology team to discuss primary PCI

Treatment recommended for ALL patients in selected patient group

» If primary percutaneous coronary intervention (PCI) is not available within 120 minutes but your patient has a contraindication to fibrinolysis:

- Seek **immediate advice from the interventional cardiology team**
- For a patient with an absolute contraindication to fibrinolysis, **primary PCI will normally be the preferred management strategy** despite the delay in accessing it.

Acute

Practical tip

The European Society of Cardiology acute coronary syndrome guideline lists the contraindications to fibrinolytic therapy as follows:[2]

- **Absolute contraindications**
  - History of intracranial haemorrhage or stroke of unknown origin at any time
  - Ischaemic stroke in the last 6 months
  - Central nervous system damage, neoplasm, or arteriovenous malformation
  - Major trauma/surgery/head injury within the last 1 month
  - Gastrointestinal bleeding within the last 1 month
  - Bleeding disorder
  - Aortic dissection
  - Non-compressible punctures within the last 24 hours (e.g., liver biopsy, lumbar puncture)
- **Relative contraindications**
  - Transient ischaemic attack in the last 6 months
  - Oral anticoagulant therapy
  - Pregnancy or within 1 week postnatal
  - Refractory hypertension (systolic BP >180 mmHg and/or diastolic BP >110 mmHg)
  - Advanced liver disease
  - Infective endocarditis
  - Active peptic ulcer
  - Prolonged or traumatic cardiopulmonary resuscitation.

» If primary PCI is undertaken, the interventional cardiology team will **start parenteral anticoagulation in the catheterisation laboratory.**

Acute

» Arrange echocardiography assessment of left ventricular ejection fraction for all STEMI patients.[75]

plus **P2Y12 inhibitor**

Treatment recommended for ALL patients in selected patient group

Primary options

» prasugrel: <75 years of age and body weight <60 kg: 60 mg orally as a loading dose, followed by 5 mg once daily thereafter; <75 years of age and body weight ≥60 kg: 60 mg orally as a loading dose, followed by 10 mg once daily thereafter; ≥75 years of age: 60 mg orally as a loading dose, followed by 5 mg once daily thereafter

OR

» clopidogrel: <75 years of age: 300-600 mg orally as a loading dose, followed by 75 mg once daily thereafter; ≥75 years of age: 75 mg orally once daily
The licensed loading dose in the UK is 300 mg. The ESC guideline recommends a higher loading dose of 600 mg for patients proceeding to PCI. While this higher dose is not licensed in the UK, it is widely used in practice.[2]

OR

» ticagrelor: 180 mg orally as a loading dose, followed by 90 mg twice daily thereafter

» Give the patient **dual antiplatelet therapy with a P2Y $_{12}$ inhibitor plus aspirin**.[75]

- After the initial aspirin loading dose, reduce the aspirin to a lower daily maintenance dose.
- Check your local protocol when deciding which P2Y $_{12}$ inhibitor to use, the timing of this and the recommended initial loading and ongoing daily doses.

» The UK National Institute for Health and Care Excellence (NICE) recommends the following:[75]

- If the patient is eligible for primary percutaneous coronary intervention (PCI):

Acute

- Prasugrel, in combination with aspirin, if they are not already taking an oral anticoagulant
  - For patients aged 75 years and older, the bleeding risk of using prasugrel needs to be weighed up against its effectiveness. If the bleeding risk from prasugrel is a concern in these patients, ticagrelor or clopidogrel may be used as alternatives.
- Clopidogrel, in combination with aspirin, if they are taking an oral anticoagulant.

- If the patient is not having primary PCI:
  - Ticagrelor, in combination with aspirin, unless the patient has a high bleeding risk
  - Clopidogrel, in combination with aspirin, or aspirin alone, for patients with a high bleeding risk.

» The European Society of Cardiology guideline recommends prasugrel or ticagrelor (in combination with aspirin) for patients undergoing primary PCI. Prasugrel should be considered in preference to ticagrelor, but clopidogrel is only indicated when neither of these drugs is available or they are contraindicated.[2]

» Cangrelor is a reversible intravenous $P2Y_{12}$ inhibitor that can be considered if the patient is unable to ingest an oral drug.[2] NICE has yet to make any recommendation on the use of cangrelor.

» Always follow your local protocol for $P2Y_{12}$ inhibitor selection and timing.

>12 hours since symptom onset: with ongoing evidence of myocardial ischaemia (symptoms and/or signs on ECG) **plus** **primary PCI**

Treatment recommended for ALL patients in selected patient group

MANAGEMENT

Acute

» Seek immediate specialist advice from cardiology to discuss management options for any STEMI patient who presents **>12 hours after symptom onset.**

- Coronary angiography ± primary percutaneous coronary intervention (PCI) is recommended if there is:
  - Evidence of continuing myocardial ischaemia or cardiogenic shock[75]
  - Haemodynamic instability or life-threatening arrhythmias.[2]

» If your patient has **ongoing ischaemic symptoms** suggestive of MI **but no ongoing ST-segment elevation** on the ECG, **primary PCI** should be considered if **one or more** of the following is present:[2]

- Cardiogenic shock or haemodynamic instability
- Acute heart failure presumed secondary to ongoing myocardial ischaemia
- Recurrent or refractory chest pain despite medical treatment
- Cardiac arrest or life-threatening arrhythmia
- Signs and symptoms suggestive of mechanical complications of acute MI
- Recurrent dynamic ST-segment or T-wave changes, especially intermittent ST-segment elevation.

plus **P2Y12 inhibitor**

Treatment recommended for ALL patients in selected patient group

Primary options

» prasugrel: <75 years of age and body weight <60 kg: 60 mg orally as a loading dose, followed by 5 mg once daily thereafter; <75 years of age and body weight ≥60 kg: 60 mg orally as a loading dose, followed by 10 mg once daily thereafter; ≥75 years of age: 60 mg orally as a loading dose, followed by 5 mg once daily thereafter

OR

Acute

» clopidogrel: <75 years of age: 300-600 mg orally as a loading dose, followed by 75 mg once daily thereafter; ≥75 years of age: 75 mg orally once daily
The licensed loading dose in the UK is 300 mg. The ESC guideline recommends a higher loading dose of 600 mg for patients proceeding to PCI. While this higher dose is not licensed in the UK, it is widely used in practice.[2]

OR

» ticagrelor: 180 mg orally as a loading dose, followed by 90 mg twice daily thereafter

» Give any patient who will undergo primary percutaneous coronary intervention (PCI) dual antiplatelet therapy with **a P2Y $_{12}$ inhibitor plus aspirin**.[75]

- After the initial aspirin loading dose, reduce the aspirin to a lower daily maintenance dose.
- Check your local protocol when deciding which P2Y $_{12}$ inhibitor to use, the timing of this and the recommended initial loading and ongoing daily doses.

» The UK National Institute for Health and Care Excellence (NICE) recommends the following for any patient undergoing PCI:[75]

- Prasugrel, in combination with aspirin, if they are not already taking an oral anticoagulant
  - For patients aged 75 years and older, the bleeding risk of using prasugrel needs to be weighed up against its effectiveness. If the bleeding risk from prasugrel is a concern in these patients, ticagrelor or clopidogrel may be used as alternatives.
- Clopidogrel, in combination with aspirin, if the patient is taking an oral anticoagulant.

» The European Society of Cardiology recommends prasugrel or ticagrelor (in combination with aspirin) for patients undergoing

Acute

primary PCI. Prasugrel should be considered in preference to ticagrelor, but clopidogrel is only indicated when neither of these drugs is available or they are contraindicated.[2]

» Cangrelor is a reversible intravenous $P2Y_{12}$ inhibitor that can be considered if the patient is unable to ingest an oral drug.[2] NICE has yet to make any recommendation on the use of cangrelor.

» Always follow your local protocol for $P2Y_{12}$ inhibitor selection and timing.

plus **parenteral anticoagulation**

Treatment recommended for ALL patients in selected patient group

Primary options

» heparin: no glycoprotein IIb/IIIa inhibitor use planned: 70-100 units/kg intravenous bolus; glycoprotein IIb/IIIa inhibitor use planned: 50-70 units/kg intravenous bolus
Monitor activated clotting time (ACT) during procedure. Further doses may be required to maintain ACT.

OR

» enoxaparin: 0.5 mg/kg intravenous bolus
Some centres may continue with subcutaneous dosing after this initial intravenous dose; consult local protocols for further guidance.

OR

» bivalirudin: 0.75 mg/kg intravenous bolus initially, followed by 1.75 mg/kg/hour intravenous infusion during procedure and for up to 4 hours after procedure, reduce to 0.25 mg/kg/hour for a further 4-12 hours if necessary

» Anticoagulation is routinely given during primary percutaneous coronary intervention (PCI).

- This will be **started by the interventional cardiology team** in the cardiac catheterisation laboratory.

Acute

- Therefore, **do not start anticoagulation** if your patient is likely to be eligible for primary PCI.

» Unfractionated heparin is recommended first line for intravenous anticoagulation.[2] [75] Alternatives include:

- Bivalirudin (a direct thrombin inhibitor) for patients with a history of heparin-induced thrombocytopenia
- Enoxaparin.

» Fondaparinux is **not recommended** as an adjunctive anticoagulant during primary PCI.[2]

- It was associated with catheter thrombosis at the time of primary PCI in the OASIS-6 trial.[188]

» **Routine post-procedural anticoagulation is not required** after primary PCI unless there is a separate indication for it, for example:[2]

- Full-dose anticoagulation:
  - Atrial fibrillation
  - Mechanical heart valve
  - Left ventricular thrombus
- Prophylactic-dose anticoagulation:
  - Patients at high risk of venous thromboembolism
- Consult local protocols for full- and prophylactic-dose regimens for these indications. The doses presented here are the doses used during primary PCI only.

consider **glycoprotein IIb/IIIa inhibitor**

Treatment recommended for SOME patients in selected patient group

Primary options

» eptifibatide: consult specialist for guidance on dose

OR

MANAGEMENT

Acute

» tirofiban: consult specialist for guidance on dose

» A glycoprotein IIb/IIIa inhibitor (e.g., eptifibatide, tirofiban, abciximab) may be used by the interventional cardiology team during the percutaneous coronary intervention procedure.[2] Abciximab is not currently available in the UK, and shortages in other countries have been reported.

- This can help to tackle a high thrombus burden or no-reflow phenomenon.

>12 hours since symptom onset: no ongoing evidence of myocardial ischaemia (no symptoms and/or signs on ECG)

plus **seek immediate cardiology advice to discuss management options**

Treatment recommended for ALL patients in selected patient group

» **Discuss urgently** with cardiology any patient who presents **>12 hours** after symptom onset but has **no ongoing symptoms.** [2] [182]

- Routine primary percutaneous coronary intervention (PCI) strategy should still be considered in patients presenting between 12 and 48 hours after symptom onset. However, if the time since symptom onset is >48 hours and the patient is now asymptomatic, routine PCI of an occluded infarct-related artery is not recommended. [2]
- Fibrinolysis is not indicated.

plus **P2Y12 inhibitor**

Treatment recommended for ALL patients in selected patient group

Primary options

» prasugrel: <75 years of age and body weight <60 kg: 60 mg orally as a loading dose, followed by 5 mg once daily thereafter; <75 years of age and body weight ≥60 kg: 60 mg orally as a loading dose, followed by 10 mg once daily thereafter; ≥75 years of age: 60 mg orally as a loading dose, followed by 5 mg once daily thereafter

OR

Acute

» clopidogrel: <75 years of age: 300-600 mg orally as a loading dose, followed by 75 mg once daily thereafter; ≥75 years of age: 75 mg orally once daily
The licensed loading dose in the UK is 300 mg. The ESC guideline recommends a higher loading dose of 600 mg for patients proceeding to PCI. While this higher dose is not licensed in the UK, it is widely used in practice.[2]

OR

» ticagrelor: 180 mg orally as a loading dose, followed by 90 mg twice daily thereafter

» Give the patient dual antiplatelet therapy with **a P2Y $_{12}$ inhibitor plus aspirin**.[75]

- After the initial aspirin loading dose, reduce the aspirin to a lower daily maintenance dose.
- Check your local protocol when deciding which P2Y $_{12}$ inhibitor to use, the timing of this and the recommended initial loading and ongoing daily doses.

» The UK National Institute for Health and Care Excellence (NICE) recommends the following:[75]

- If the patient is eligible for primary percutaneous coronary intervention (PCI):
  - Prasugrel, in combination with aspirin, if they are not already taking an oral anticoagulant
    - For patients aged 75 years and older, the bleeding risk of using prasugrel needs to be weighed up against its effectiveness. If the bleeding risk from prasugrel is a concern in these patients, ticagrelor or clopidogrel may be used as alternatives.

Acute

- Clopidogrel, in combination with aspirin, if the patient is taking an oral anticoagulant.

- If the patient is not having primary PCI:
  - Ticagrelor, in combination with aspirin, unless the patient has a high bleeding risk
  - Clopidogrel, in combination with aspirin, or aspirin alone, for patients with a high bleeding risk.

» The European Society of Cardiology guideline recommends clopidogrel (in combination with aspirin) as the preferred agent.[2]

Ongoing

post-STEMI

1st **continue dual antiplatelet therapy**

Primary options

» aspirin: 75-100 mg once daily thereafter

--AND--

» prasugrel: <75 years of age and body weight <60 kg: 5 mg orally once daily; <75 years of age and body weight ≥60 kg: 10 mg orally once daily; ≥75 years of age: 5 mg orally once daily

-or-

» ticagrelor: 90 mg orally twice daily

-or-

» clopidogrel: 75 mg orally once daily

Secondary options

» clopidogrel: 75 mg orally once daily

» Ensure all patients are given dual antiplatelet therapy with aspirin and a P2Y $_{12}$ inhibitor (while taking into account any contraindications; seek specialist advice if the patient has a separate indication for anticoagulation. See more info panel below).[75]

» **Continue the P2Y $_{12}$ inhibitor** used in the acute phase for up to 12 months (unless contraindicated).[75] The National Institute for Health and Care Excellence (NICE) in the UK recommends:[75]

- For patients who had undergone percutaneous coronary intervention (PCI):
  - Prasugrel in those not taking an oral anticoagulant. However, follow your local protocol. At present, many hospitals in the UK use ticagrelor rather than prasugrel in these patients. This new guidance by NICE will require a change in current practice in the UK. Consider ticagrelor or clopidogrel in patients aged 75 years or older if the bleeding risk from prasugrel is a concern
  - Clopidogrel in those taking an oral anticoagulant.

Ongoing

- For patients who had fibrinolysis:
  - Ticagrelor unless the patient has a high bleeding risk
  - Clopidogrel or aspirin alone if the patient has a high bleeding risk.

» The European Society of Cardiology recommends 12 months of dual antiplatelet therapy as the default strategy, although alternate regimes can be considered in certain circumstances depending on bleeding and ischaemic risks:[2] [195]

- Single antiplatelet therapy (preferably with a P2Y $_{12}$ receptor inhibitor, e.g., ticagrelor) for patients who are event-free **after 3-6 months**of dual antiplatelet therapy and who are not high ischaemic risk[196] [197] [198] [199] [200] [201]
- Aspirin or P2Y $_{12}$ receptor inhibitor monotherapy after 1 month of dual antiplatelet therapy in patients with high bleeding risk.[202]

» De-escalation of antiplatelet therapy in the first 30 days is not recommended. However, beyond 30 days after an acute coronary syndrome, de-escalation of P2Y $_{12}$ receptor inhibitor therapy may be considered as an alternative strategy in those at high bleeding risk.[2] Theoretically, by identifying patients who are unlikely to have a response to clopidogrel by genetic/platelet function testing, there is potential for personalised antiplatelet therapy.[203] However, it is unclear whether de-escalation guided by platelet function testing or genetic testing improves clinical management and outcomes, and such a strategy based on platelet function testing or genetic testing should be prospectively tested in patients who may benefit from de-escalating antithrombotic therapy.[2]

» **Continue aspirin** indefinitely unless the patient has hypersensitivity.

Ongoing

Practical tip

Evidence on the selection and duration of antiplatelet therapy following coronary revascularisation is evolving rapidly, with studies showing potential benefits from different strategies.[204] [205]
In the UK, you should check the patient's clinical report for an individualised plan written by the interventional cardiology team. This should specify what antiplatelet therapy is advised for long-term management, based on current evidence, individual patient factors, and the specific interventions undertaken for each patient.

More info: Antiplatelet therapy for patients with a separate indication for anticoagulation

Seek specialist advice when deciding the duration and type (dual or single) of antiplatelet therapy in the 12 months after STEMI for patients with a separate indication for anticoagulation (e.g., in patients with ongoing atrial fibrillation).

The National Institute for Health and Care Excellence (NICE) in the UK recommends taking account of all of the following when deciding about the duration and type (dual or single) of antiplatelet therapy in patients with a separate indication for anticoagulation:[75]

- Bleeding risk
- Thromboembolic risk
- Cardiovascular risk
- Patient's wishes.

Be aware that long-term continuation of aspirin, clopidogrel, and oral anticoagulation (triple therapy) significantly increases bleeding risk.

For patients **already on anticoagulation** who **had PCI**:[75]

- Continue anticoagulation and clopidogrel for up to 12 months
- If the patient is taking a direct oral anticoagulant, adjust the dose according to bleeding

Ongoing

risk, thromboembolic risk, and cardiovascular risk.

For patients with a **new indication for anticoagulation** who **had PCI**:[75]

- Offer clopidogrel (to replace prasugrel or ticagrelor) for up to 12 months and an oral anticoagulant licensed for the indication, which best matches the patient's bleeding risk, thromboembolic risk, cardiovascular risk, and wishes.

For patients already on anticoagulation, or those with a new indication, who did **not have PCI**:[75]

- Continue anticoagulation and, unless there is a high risk of bleeding, consider continuing aspirin (or clopidogrel for patients with contraindication for aspirin) for up to 12 months.

Do not routinely offer prasugrel or ticagrelor in combination with anticoagulation in patients with a separate indication for anticoagulation.[75]

plus **start or continue beta-blocker or non-dihydropyridine calcium-channel blocker**

Treatment recommended for ALL patients in selected patient group

Primary options

» bisoprolol: 1.25 mg orally once daily initially for 1 week, increase gradually according to response, maximum 10 mg/day

OR

» carvedilol: 3.125 mg orally twice daily initially, increase gradually according to response, maximum 50 mg/day (body weight <85 kg) or 100 mg/day (body weight >85 kg)

Secondary options

» verapamil: 240 mg orally (modified-release) in the morning and 120 mg in the evening; or

Ongoing

120 mg orally (modified-release) three times daily

» Ensure all patients are given a beta-blocker (while taking into account any contraindications).[75]

- Start this as soon as the patient is haemodynamically stable.[75]
- Continue the beta-blocker for at least 12 months if the patient does not have reduced left ventricular ejection fraction (LVEF).[75] [206]

» Continue the beta-blocker indefinitely if the patient has reduced LVEF.[75]

» Do not routinely give calcium-channel blockers after STEMI.[75]

- Consider a non-dihydropyridine calcium-channel blocker, such as verapamil, in a patient without pulmonary congestion or reduced LVEF who has a contraindication to beta-blockers (or when these need to be discontinued).[75]

» Do not offer nicorandil (a potassium-channel activator) after STEMI.[75]

plus **start or continue ACE inhibitor or angiotensin-II receptor antagonist**

Treatment recommended for ALL patients in selected patient group

Primary options

» enalapril: 2.5 mg orally once daily initially, increase gradually according to response, usual dose 10-20 mg twice daily, maximum 40 mg/day

OR

» ramipril: 2.5 mg orally twice daily for 3 days, increase gradually according to response, maximum 10 mg/day

OR

» lisinopril: 2.5 to 5 mg orally once daily initially, increase gradually according to response, maximum 10 mg/day

Secondary options

MANAGEMENT

Ongoing

» valsartan: 20 mg orally twice daily initially, increase gradually according to response, maximum 320 mg/day

OR

» losartan: 12.5 mg orally once daily initially, increase gradually according to response, maximum 150 mg/day

OR

» candesartan: 4 mg orally once daily initially, increase gradually according to response, maximum 32 mg/day

» Ensure all patients are given an ACE inhibitor (while taking into account any contraindications).

- Start this as soon as the patient is haemodynamically stable and continue it indefinitely. Complete upwards dose titration within 4-6 weeks of hospital discharge.[75]
- Offer an angiotensin-II receptor antagonist as an alternative if the patient is intolerant to an ACE inhibitor.[75]
- Measure renal function, serum electrolytes, and blood pressure before starting an ACE inhibitor or angiotensin-II receptor antagonist.[75] In practice, if the patient has abnormal renal function or blood pressure, start with a low dose and titrate this carefully with close monitoring.

plus **statin**

Treatment recommended for ALL patients in selected patient group

Primary options

» atorvastatin: 40-80 mg orally once daily

OR

» rosuvastatin: 20-40 mg orally once daily

» Ensure all patients are given a high-intensity statin (while taking into account any contraindications).[45] [63] [75]

consider **ezetimibe**

Treatment recommended for SOME patients in selected patient group

Primary options

Ongoing

» ezetimibe: 10 mg orally once daily

» If low-density lipoprotein (LDL)-cholesterol targets are not achieved with maximal statin therapy (LDL-cholesterol is <1.4 mmol/L [<55 mg/dL] and a ≥50% LDL-cholesterol reduction from baseline), add ezetimibe.[2] [63]

consider **proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitor**

Treatment recommended for SOME patients in selected patient group

Primary options

» evolocumab: 140 mg subcutaneously every 2 weeks; or 420 mg subcutaneously once monthly

OR

» alirocumab: 75-150 mg subcutaneously every 2 weeks; or 300 mg subcutaneously every 4 weeks

» A PCSK9 inhibitor monoclonal antibody may be added to statin and ezetimibe therapy if LDL-cholesterol targets are not achieved despite maximal statin and ezetimibe therapy.[2] [63] [210] [211] [212] Treatment can be started during acute coronary syndrome admission or at outpatient follow-up 4-6 weeks later.

consider **aldosterone antagonist**

Treatment recommended for SOME patients in selected patient group

Primary options

» eplerenone: 25 mg orally once daily initially, increase gradually according to response, maximum 50 mg/day

OR

» spironolactone: 25 mg orally once daily initially, increase gradually according to response, maximum 50 mg/day

» Give an aldosterone antagonist to any patient with signs or symptoms of heart failure and reduced left ventricular ejection fraction.[75]

- Start this within 3-14 days of a STEMI and preferably after starting an ACE inhibitor.[75]

Ongoing

consider **sodium-glucose co-transporter-2 (SGLT2) inhibitor**

Treatment recommended for SOME patients in selected patient group

Primary options

» dapagliflozin: 10 mg orally once daily

OR

» empagliflozin: 10 mg orally once daily

» Give an SGLT2 inhibitor (e.g., dapagliflozin, empagliflozin) to patients with heart failure when they are clinically stable, regardless of their left ventricular ejection fraction.[213] [214] [215]

plus **cardiac rehabilitation**

Treatment recommended for ALL patients in selected patient group

» Offer cardiac rehabilitation to all patients. This should include an exercise component, health education, stress management, and psychological and social support. Advise all patients on lifestyle changes such as:[2] [75]

- Changes to diet
- Reduction of alcohol consumption
- Smoking cessation
- Weight management
- Physical exercise
- Reduced sedentary time.

Emerging

Factor Xa inhibitors

These are being studied for acute coronary syndromes.[217] Rivaroxaban is a factor Xa inhibitor that has been shown to reduce cardiovascular events when given in addition to dual antiplatelet therapy to stabilised STEMI patients, but increases bleeding events.[218] One review and meta-analysis found that direct oral anticoagulants in combination with antiplatelet therapy were associated with a reduced risk of ischaemic events at the cost of an increase in major bleedings compared with antiplatelet therapy alone in patients with STEMI.[219]

Factor IX inhibitors

Pegnivacogin, a factor IX inhibitor that is reversible with anivamersen, has been shown to reduce the incidence of ischaemic events in patients with acute coronary syndrome (ACS) compared with intravenous heparin, when given during coronary interventions.[220] [221] [222]

Factor XI inhibitors

Multiple factor XI inhibitors are under investigation for various indications.[223] Asundexian, a small molecule factor XIa inhibitor, has been investigated specifically for ACS.[223]

L-carnitine

One meta-analysis of 13 placebo-controlled trials found L-carnitine to significantly reduce all-cause mortality, ventricular arrhythmias, and angina symptoms in patients experiencing acute myocardial infarction. Further evaluation is needed.[224]

Colchicine

Several randomised controlled trials have investigated the role of the anti-inflammatory agent colchicine in both chronic and ACS.[2] [225] Use of colchicine for secondary prevention was shown to significantly reduce composite cardiovascular end points (including cardiovascular death, myocardial infarction, and stroke) in two meta-analyses of trials investigating patients with a previous acute coronary event.[225] [226] [227] Research suggests the beneficial effect is greater with early, in-hospital initiation of treatment.[228] Patients should be investigated for liver and/or kidney disease prior to commencing colchicine.[229] The European Society of Cardiology recommends that low-dose colchicine may be considered for long-term management of patients with ACS on the basis of its anti-inflammatory properties, particularly if other risk factors are insufficiently controlled or recurrent cardiovascular events occur under optimal therapy.[2]

Dalcetrapib

Dalcetrapib, a cholesterol ester transfer protein inhibitor (CETP), has been shown to reduce the incidence of new-onset diabetes in patients with recent ACS.[230]

Polypill therapy

One pill that combines routine post-ACS medications (e.g., aspirin, an ACE inhibitor such as ramipril, and a statin such as atorvastatin) has been shown to improve treatment compliance and consequently reduce risk of adverse cardiovascular events post-ACS.[231]

Semaglutide

The glucagon-like peptide-1 (GLP-1) receptor agonist semaglutide has been shown to reduce the risk of death from cardiovascular causes in overweight patients with pre-existing cardiovascular disease.[232] [233]

Primary prevention

Use a systematic approach among people aged 40 years and above to identify those at high risk of cardiovascular disease (CVD) and most likely to benefit from preventive actions.[63] [64]

- National Institute for Health and Care Excellence (NICE) guidance recommends the use of the QRISK3 tool to assess 10-year CVD risk for primary prevention in individuals aged between 25 and 84 years.[63] [QRISK3]
- The European Society of Cardiology (ESC) guideline on CVD prevention recommends use of country-specific SCORE2 risk charts.[64]

Most guidelines recommend initial risk assessment using 10-year CVD risk calculators.[63] [64] [65] Consider use of a lifetime risk estimation tool to inform subsequent discussion and shared decision-making, and to motivate lifestyle changes; NICE suggests considering this in people with a 10-year QRISK3 score less than 10%, and in people under age 40 years who have CVD risk factors.[63] [64] [QRISK3-lifetime]

CVD risk tools may underestimate risk in certain groups of people (e.g., people with severe mental illness; those with autoimmune disorders and other systemic inflammatory disorders; people who have recently stopped smoking; people taking medications for HIV, immunosuppressants, or treatments for CVD risk factors).[63] Some groups of patients are unsuitable for calculator-based risk assessment so check the guidance for each tool carefully. For example, NICE recommends that all patients with type 1 diabetes, patients with an estimated glomerular filtration rate of <60 mL/min/1.73 m^2, and patients with familial hypercholesterolaemia should be considered at high risk of CVD without using a risk assessment tool.[63]

Risk assessment becomes more challenging with advancing age. Coronary artery calcification increases every decade over the age of 40, and increases rapidly in women post menopause.[66] However, CVD-free survival dissociates from overall survival with increasing age, and there is evidence that the QRISK3 tool overstates CVD risk in older people.[64] [67] In view of this ongoing uncertainty, guideline organisations differ in their approach to age-specific recommendations:

- NICE recommends that any person aged 85 years or older should be considered high risk based on age alone (especially if they smoke and/or have high blood pressure).[63]
- The ESC recommends progressively higher thresholds for treatment of risk factors with advancing age.[64]

Encourage individuals of all ages to adopt a healthy lifestyle and take steps to modify any major risk factors for CVD regardless of their 10-year or lifetime risk score, and before offering medicines to treat risk factors.[63] [64]

- Advise smokers to stop and offer support to help them do so.
    - For a smoker, cessation is the single most important step that can be taken to reduce heart-related and all-cause death.
    - Even low levels of smoking increase risk of CVD; this includes exposure to secondhand smoke.[7] [15][18]
    - Various support programmes, medicines, and alternative therapies are available; a combination of behavioural interventions plus pharmacotherapy is recommended.[18] [65]
- Encourage a cardioprotective diet, for example replacing consumption of saturated fats (e.g., meat, dairy products) with unsaturated fats (e.g., fish, fruits, vegetables, and nuts).
- Offer advice on ways of increasing activity levels to the recommended minimum (150 minutes of moderate intensity or 75 minutes of vigorous intensity aerobic physical activity each week).[68]
- Advise weight loss if the person is living with obesity or overweight.

- Advise avoidance of excess alcohol consumption.
- Advise on further dietary and lifestyle measures and/or treatment to control other modifiable risk factors (e.g., low salt diet for control of high blood pressure, with addition of antihypertensive medication if indicated).

Offer a statin to any individual whose CVD risk score puts them at increased risk of CVD.

- NICE recommends to offer atorvastatin for primary prevention to anyone whose 10-year CVD risk is estimated to be 10% or higher, using the QRISK3 tool (after modification of lifestyle and management of risk factors).[63]
- The ESC recommendations for lipid-lowering treatment follow age-band-specific 10-year risk thresholds, with higher treatment thresholds recommended for older people.[64]
- Check local and national guidelines; treatment recommendations differ depending on underlying population risk and practical considerations. For example, the Scottish Intercollegiate Guidelines Network (SIGN) concluded that a 10-year CVD risk of 20% or higher is a more appropriate threshold for offering statin treatment for primary prevention in the Scottish population, after their analysis found 95% of all individuals aged 60 to 64 in Scotland to be eligible for treatment under a 10% or higher 10-year risk threshold.[69]

Aspirin is not recommended for the primary prevention of CVD because the increased risk of major bleeding is considered to outweigh any potential benefits.[63] [64]

Secondary prevention

Recommended pharmacotherapy for secondary prevention is covered in Management recommendations .

Offer cardiac rehabilitation to all patients. This should include an exercise component, health education, stress management, and psychological and social support. Advise all patients on lifestyle changes such as:[2] [75]

- Changes to diet
- Reduction of alcohol consumption
- Smoking cessation
- Weight management
- Physical exercise
- Reduced sedentary time.

The most important preventive actions involve combined dietary and lifestyle changes, as outlined in the Primary prevention section.

Patients should switch to a heart-healthy diet. If overweight, patients should lose weight and maintain a healthy body weight.[2] In overweight/obese patients weight loss is associated with an improvement in the constituent elements of the metabolic syndrome, as well as a reduction in the pathological aspects implicated in coronary artery disease: endothelial dysfunction and inflammation.[257] Patients should consume a diet rich in vegetables and fruits. Patients should be advised to choose wholegrain, high-fibre foods and to eat fish, especially oily fish, at least twice a week. Excess sugars, trans-fats, salt, and foods with excess cholesterol should be limited.

For a smoker, cessation is the single most crucial step that can be taken to reduce heart-related and all-cause death.[18] This includes avoiding second-hand smoke. Many different types of support are available, and smoking cessation service referrals can be made via a cardiac rehabilitation programme. Data from the EVITA (Evaluation of Varenicline in Smoking Cessation for patients post Acute Coronary Syndrome) trial suggest that pharmacotherapy with varenicline started in hospital at the time of an acute coronary

syndrome may be efficacious for smoking cessation; however, further studies to assess safety end points are needed.[258]

Cardiorespiratory fitness is a strong predictor of outcome following an acute myocardial infarction.[2] Patients should be encouraged to partake in regular aerobic and resistance exercise in addition to the exercise programme of cardiac rehabilitation; in patients with CAD, there is a direct correlation between the volume of moderate to vigorous physical activity and reduction in cardiovascular risk and mortality.[2] [38] Advice about exercise should be offered as part of a cardiac rehabilitation programme.[75] It is recommended that patients engage in ≥30 minutes of moderate-intensity physical activity on most, and preferably all, days of the week. Likewise, patients should engage in multiple short bouts of physical activity daily, such as walking the dog or taking the stairs instead of the lift.

Sedentary behaviour is an independent risk factor for all-cause mortality and the European Society of Cardiology recommends advising reduced sedentary time.[2]

Family members can be very helpful and should become involved along with other support systems to help remind patients of, and to reinforce, lifestyle changes. Patients should use the resources that are available (e.g., written materials, the Internet, educational classes, regular counselling) and be in close communication with healthcare providers.

Patient discussions

Patients should schedule a follow-up appointment with their doctor in 1-2 weeks. Patients should be given prescriptions and detailed discharge instructions including a list of medicines to take (e.g., dual antiplatelet therapy, ACE inhibitor, beta-blocker, statin). These instructions should inform the patient what to do if they experience any recurrent signs or symptoms and should include restrictions on physical activity. Before discharge, patients should also receive instruction and prescriptions for any additional testing that is needed by the physician. If one is available, patients should enter a cardiac rehabilitation programme. Cardiac rehabilitation is a structured programme that provides myocardial infarction survivors with the tools, motivation, and support needed to change behaviour and increase chance of survival. Typically, cardiac rehabilitation programmes use group therapy to supervise and promote beneficial exercise, as well as to provide emotional support.

Patients should return to the nearest accident and emergency department or call their physician if they develop recurring chest pain or discomfort, shortness of breath, sweating, gastrointestinal symptoms, lightheadedness, palpitations, or other symptoms suggesting another myocardial infarction or heart condition.

FOLLOW UP

Monitoring

Monitoring

Survivors of acute MI should be closely followed up to ensure adequate modification of risk factors and optimisation of (and adherence to) pharmacotherapy for secondary prevention, as well as to monitor for the development of post MI complications and/or residual angina symptoms. Patients should be evaluated within 2 to 3 weeks of discharge and evaluated periodically based on the extent of myocardial damage and patient condition.

Initiating risk-factor modification and aggressive medical management prior to discharge has been associated with increased patient adherence. All patients should continue the optimal medical regimen indefinitely. This includes dual antiplatelet therapy with aspirin plus ticagrelor, prasugrel, or clopidogrel (for at least 1 year); beta-blockers; statins; and ACE inhibitors (especially in patients with decreased ejection fraction).

Ejection fraction is assessed by echocardiography during the index hospital admission, possibly 3 months after the acute episode (depending on physician discretion and/or institutional protocols) and then periodically thereafter, depending on left ventricular (LV) function and symptoms.[131] Patients with ejection fraction <35% at 3 months' follow-up should be referred to an electrophysiologist for consideration of an implantable cardioverter defibrillator (ICD), as there is a high risk for arrhythmias in this population. Patients who develop diminished LV function and congestive heart failure should be followed up and managed appropriately.

Patients with a history of MI are at increased risk for recurrent infarction. They should be evaluated by stress testing or cardiac catheterisation if symptoms develop. Routine periodic ischaemic evaluations with stress echocardiography or myocardial perfusion studies are controversial.

Complications

Complications	Timeframe	Likelihood
sinus bradycardia, first-degree heart block, and type I second-degree heart block	**short term**	**medium**
Usually develop with occlusion of the right coronary artery, and are caused by infarction of the atrioventricular node and the conduction system above the His bundle or by increased vagal tone. These arrhythmias are usually benign and transient and do not require any treatment. If the heart rate goes below 50 bpm and the patient is symptomatic, give intravenous atropine. Temporary pacing may be required if heart failure, syncope or angina develop.		
complete heart block with anterior MI	**short term**	**medium**
Can develop acutely in patients with anterior infarcts and may lead to ventricular asystole. Complete heart block in anterior infarcts occurs because of infarction and necrosis of the bundle branches in the septum. Trans-cutaneous or, preferably, trans-venous pacing should be carried out in these patients immediately after conduction defects are noticed. Permanent pacing is recommended when high-degree atrioventricular block does not resolve within a waiting period of at least 5 days after MI.		
recurrent chest pain	**short term**	**medium**
Chest pain may occur after percutaneous coronary intervention.[251] Angina-like symptoms should be assessed with ECG changes for escalation of medical therapy, and repeat angiography considered.[71]		
type II second-degree heart block with anterior MI	**short term**	**low**
Usually occurs from a defect in the conduction system below the His bundle and can rapidly progress into complete heart block in patients with anterior MI. Trans-cutaneous or more preferably trans-venous cardiac pacing should be carried out in these patients immediately after conduction defects are noted.		
complete heart block with inferior MI	**short term**	**low**
Can develop in patients with inferior infarcts caused by vagal stimulation. Usually transient or resolved with atropine. Temporary pacing may be required if heart failure, syncope, or angina develop. Permanent pacing is rarely required.		

FOLLOW UP

Complications	Timeframe	Likelihood
acute mitral regurgitation	**short term**	**low**

Acute mitral regurgitation from papillary muscle rupture is a rare but serious complication of acute MI, with in-hospital mortality rates of 10% to 40%.[252] Inferior MI can cause rupture of the posteromedial papillary muscle, while anterolateral infarction can cause rupture of the anterolateral papillary muscle.[252]

Right ventricular papillary muscle rupture is rare and can cause life-threatening tricuspid regurgitation.

Complete papillary muscle rupture causes wide-open mitral regurgitation and is usually fatal.

Risk factors for papillary muscle rupture include older age, female sex, history of heart failure, chronic kidney disease, and delayed presentation with a first acute MI.[252]

Patients typically present 3-5 days after a transmural infarct, with acute pulmonary oedema and cardiogenic shock.[100] [252] Diagnosis is made on echocardiogram, which shows severe mitral regurgitation, often with an eccentric jet and a mobile mass, sometimes prolapsing into the left atrium.[252]

Inotropic support, mechanical ventilation, and an intra-aortic balloon pump should be considered for transient stabilisation before emergency surgery.[252]

ventricular septal defects (VSD)	**short term**	**low**

Interventricular septal rupture causing a VSD can occur, though is rare, occurring in about 0.3% of acute MI.[252] Risk factors include older age, female sex, and delayed reperfusion following acute MI.[252] Patients typically present 3-5 days after an acute MI with symptoms such as dyspnoea and orthopnoea, hypotension, peripheral vasoconstriction, and oliguria.[252] Examination reveals a new pansystolic murmur with signs of pulmonary venous congestion.[252] Diagnosis is made on echocardiogram and cardiac catheterisation.[252]

Treatment depends on the severity at presentation, though is primarily emergency cardiac surgery.[252] Stabilisation with extracorporeal membrane oxygenation (ECMO) or Impella devices may be indicated.[252]

Mortality of an uncorrected VSD is around 80% at 30 days.[252]

Survival depends on early recognition, defect size, and degree of impairment of ventricular function.

acute pericardial tamponade	**short term**	**low**

The exact incidence of ventricular free wall rupture causing acute pericardial tamponade is unknown, because it most often presents with out-of-hospital sudden cardiac death.[252] Free wall rupture should be suspected in any patient who presents with cardiovascular collapse or haemodynamic instability after acute MI, particularly following delayed presentation/revascularisation.[252] Patients present with clinical signs of cardiac tamponade: muffled heart sounds, elevated jugular venous pressure, pulsus paradoxus, and/or frank electromechanical dissociation.[252]

There is an increased risk of ventricular wall rupture involving anterior and lateral walls after anterior MI. Incomplete rupture can result in the development of a pseudoaneurysm.

Free wall rupture is rapidly fatal; however, diagnosis can be confirmed on echocardiogram, and should precipitate immediate surgical correction of the necrotic myocardium and primary reconstruction.

Inotropic support and intra-aortic balloon counterpulsation (IABP) or extracorporeal membrane oxygenation (ECMO) for transient stabilisation should be considered before surgery. Even with surgical correction, the in-hospital mortality is approximately 35%.[252]

Complications	Timeframe	Likelihood
post-infarction pericarditis (Dressler syndrome)	**short term**	**low**
Dressler syndrome is mediated by inflammatory byproducts and the formation of ischaemic myocardium. Occurs in about 5% to of patients with MI.[253] Inflammation involves the pericardium and is treated with non-steroidal anti-inflammatory drugs (NSAIDs) or colchicine (preferably colchicine as it may reduce the risk of recurrence). If haemodynamic compromise is present, pericardiocentesis or surgical intervention is required.[253]		
venous thromboembolism (VTE)	**short term**	**low**
Myocardial infarction has been associated with an increased risk of VTE, particularly for pulmonary embolism.[256]		
congestive heart failure	**long term**	**high**
Congestive heart failure caused by decreased left ventricular (LV) function occurs frequently after MI because of myocardial damage, infarct progression, and LV remodelling after the acute episode. Heart failure post MI may be more common in females than in males.[250] Appropriate use of medications, including beta-blockers, ACE inhibitors, angiotensin-II receptor antagonists, and diuretics, when appropriate, decreases the incidence and progression of congestive heart failure. Biventricular pacing with or without an implantable cardioverter defibrillator should be considered if appropriate criteria are met.		
ventricular arrhythmias	**variable**	**high**
Ventricular tachycardia (VT) and ventricular fibrillation (VF) can occur during ischaemia and reperfusion and can be lethal. Between 6% and 8% percent of STEMI patients develop haemodynamically significant VT or VF.[244] The majority of VT/VF occurs within the first 48 hours after admission.[245] Arrhythmias can also occur at any stage following a MI because of re-entry circuits at the border of myocardial scar and normal myocardium, and are commonly seen in patients with decreased ejection fraction. Electrolytes should be optimised, especially potassium and magnesium, as electrolyte imbalance increases the risk of ventricular arrhythmias. Potassium should be maintained at >4 millimol/L (4 mEq/L) and magnesium at >1 millimol/L (2 mEq/L). Appropriate therapy should be initiated with direct current cardioversion and anti-arrhythmic therapy. Optimal medical management (in particular, early administration of intravenous or oral beta-blockers) decreases incidence of ventricular arrhythmias. Implantable cardioverter defibrillator should be considered for all patients with persistently decreased left ventricular ejection fraction (<35%) if there has been no response to 3 months of intensive medical therapy after MI.[246]		
recurrent ischaemia and infarction	**variable**	**high**
Patients with acute MI can have recurrent ischaemia or infarction caused by further plaque rupture and progression of atherosclerosis.		

FOLLOW UP

Complications	Timeframe	Likelihood
Recurrence should be treated in the same manner as the initial presentation. Aggressive risk factor modification after the initial presentation decreases incidence of recurrences.		
depression	**variable**	**high**
Depression is a risk factor for cardiovascular disease and for adverse outcomes following acute coronary syndrome (ACS).[56] ACS can also precipitate depression in people without prior psychiatric conditions.[56] [247] [248] Patients should routinely be screened for depression following an MI.[247] Data suggest that a combined psychosocial approach to the treatment of depression improves outcomes in patients. Exercise combined with pharmacotherapy may be the most efficacious approach.[55] Pharmacotherapy may be associated with excess risk in patients with residual cardiac dysfunction; cognitive behavioral therapy or exercise therapy may be more appropriate in this patient group.[249]		
in-stent thrombosis	**variable**	**low**
Dual antiplatelet therapy is recommended for at least 12 months in all patients whether they have been stented or not. In-stent thrombosis is often precipitated by premature cessation of dual antiplatelet therapy, but can also be caused by technical factors and other comorbidities such as diabetes mellitus. Patients and their family should be strongly cautioned in hospital and in follow-up appointments about the importance of dual antiplatelet therapy for 12 months.[254]		
left ventricular (LV) thrombus	**variable**	**low**
LV thrombus can be seen in the early days of acute MI, especially large anterior MI with dyskinesia of the apex.[255] The reported incidence of LV thrombus after anterior STEMI varies widely in the literature, ranging from 4% to 39%.[255] Echocardiography studies conducted before revascularization became routine practice suggested LV thrombus was present in about one third of patients with large anterior MI; more recent estimates are lower, likely relating to improved reperfusion interventions.[255] Formation of LV thrombus after acute MI is associated with a 5.5-fold increased risk of embolic events compared with no thrombus. Anticoagulation with a vitamin K antagonist (e.g., warfarin) or a direct oral anticoagulant is recommended for patients with LV thrombus.[255]		
LV aneurysm	**variable**	**low**
The incidence of LV aneurysm formation after acute MI is low (<5%) in the era of reperfusion therapy, and it is seen more frequently in large anterior MI, with total occlusion of the left anterior descending artery.[252] The presence of LV aneurysm may increase the risk for angina pectoris, thromboembolic complications, and arrhythmia, although surgery is rarely needed to correct the aneurysm.[71] [252] In comparison to true aneurysms, pseudoaneurysms more often occur in the inferior or lateral wall.[252] LV pseudoaneurysms present in a very diverse way, weeks to years after an acute MI.[252] Patients may be asymptomatic, though the majority present with shortness of breath, chest pain, or signs and symptoms of congestive cardiac failure.[252] LV pseudoaneurysm requires urgent surgical repair because of the risk of progression to LV rupture.[252]		

Prognosis

Prognosis for patients with STEMI varies depending on time to presentation after onset of chest pain and time to treatment after presentation. In-hospital mortality from STEMI is around 9%.[234] [235] Survival rates have improved significantly over the last 20 years but mortality remains substantial, particularly when complicated by cardiogenic shock. Major bleeding as defined by the Bleeding Academic Research Consortium (BARC) or the Thrombolysis in Myocardial Infarction (TIMI) bleeding score is associated with worse 1-year mortality.[236]

Patients with elevated troponin levels have a worse prognosis than those with normal troponin levels.[237] [238] Prognosis is improved by early reperfusion, adherence to appropriate medical therapy, and risk factor modification. Participation in cardiac rehabilitation reduces all-cause mortality and readmissions for cardiac reasons.[239] Non-fatal health outcomes (including development of heart failure, atrial fibrillation, cerebrovascular disease, peripheral arterial disease, severe bleeding, renal failure, diabetes mellitus, dementia, depression and cancer) and all-cause mortality are higher in patients who have had an MI.[240]

Adherence to evidence-based medicine has been shown to have better patient outcomes.[241] [242] Specific risk models to predict mortality following MI in older adults have been developed, including variables such as hearing impairment, mobility impairment, weight loss, and patient-reported health status.[243]

Diagnostic guidelines

United Kingdom

Acute coronary syndromes

Published by: National Institute for Health and Care Excellence **Last published:** 2020

Europe

2023 ESC guidelines for the management of acute coronary syndromes

Published by: European Society of Cardiology **Last published:** 2023

Fourth universal definition of myocardial infarction

Published by: European Society of Cardiology **Last published:** 2018

2018 ESC/EACTS guidelines on myocardial revascularization

Published by: European Society of Cardiology; European Association for Cardio-Thoracic Surgery **Last published:** 2018

North America

2021 ACC/AHA/SCAI guideline for coronary artery revascularization

Published by: American College of Cardiology; American Heart Association; Society for Cardiovascular Angiography and Interventions **Last published:** 2021

2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction

Published by: American College of Cardiology Foundation; American Heart Association **Last published:** 2012

Treatment guidelines

United Kingdom

Acute coronary syndromes

Published by: National Institute for Health and Care Excellence **Last published:** 2020

Recent-onset chest pain of suspected cardiac origin: assessment and diagnosis

Published by: National Institute for Health and Care Excellence **Last published:** 2016

Europe

2023 ESC guidelines for the management of acute coronary syndromes

Published by: European Society of Cardiology **Last published:** 2023

Pre-hospital management of patients with chest pain and/or dyspnoea of cardiac origin

Published by: Acute Cardiac Care Association of the European Society of Cardiology **Last published:** 2020

European Resuscitation Council guidelines 2021

Published by: European Resuscitation Council **Last published:** 2021

2018 ESC/EACTS guidelines on myocardial revascularization

Published by: European Society of Cardiology; European Association for Cardio-Thoracic Surgery **Last published:** 2018

North America

2021 ACC/AHA/SCAI guideline for coronary artery revascularization

Published by: American College of Cardiology; American Heart Association; Society for Cardiovascular Angiography and Interventions **Last published:** 2021

2019 CCS/CAIC guidelines on the acute management of ST-elevation myocardial infarction: focused update on regionalization and reperfusion

Published by: Canadian Cardiovascular Society; Canadian Association of Interventional Cardiology **Last published:** 2019

Clinical policy: emergency department management of patients needing reperfusion therapy for acute ST-segment elevation myocardial infarction

Published by: American College of Emergency Physicians **Last published:** 2017

Acute coronary syndromes: 2015 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care science

Published by: American Heart Association **Last published:** 2015

ACCF/AHA guideline for the management of ST-elevation myocardial infarction

Published by: American College of Cardiology Foundation; American Heart Association **Last published:** 2013

2013 AHA/ACC guideline on lifestyle management to reduce cardiovascular risk

Published by: American Heart Association; American College of Cardiology **Last published:** 2013

Effectiveness-based guidelines for the prevention of cardiovascular disease in women - 2011 update

Published by: American Heart Association **Last published:** 2011

Asia

Consensus recommendations of the Asia Pacific Cardiometabolic Consortium on secondary prevention strategies in myocardial infarction: recommendations on pharmacotherapy, lifestyle modification and cardiac rehabilitation

Published by: Asia Pacific Cardiometabolic Consortium **Last published:** 2023

CVIT expert consensus document on primary percutaneous coronary intervention (PCI) for acute myocardial infarction (AMI) update 2022

Published by: Japanese Association of Cardiovascular Interventional and Therapeutics (CVIT) **Last published:** 2022

2021 Korean Society of Myocardial Infarction expert consensus document on revascularization for acute myocardial infarction

Published by: Korean Society of Myocardial Infarction **Last published:** 2021

Experts' consensus: pharmaco-invasive therapy for ST#elevation myocardial infarction along with focus on secondary prevention and cardiac rehabilitation in India

Published by: Indian Expert Consensus **Last published:** 2021

Management of acute ST-segment elevation myocardial infarction (STEMI)

Published by: Academy of Medicine Malaysia; Ministry of Health Malaysia; International Heart Association of Malaysia **Last published:** 2019

API expert consensus document on management of ST-elevation myocardial infarction: adaptation of 2012 ESC guidelines

Published by: Association of Physicians of India **Last published:** 2018

Oceania

Australian clinical guidelines for the management of acute coronary syndromes 2016

Published by: National Heart Foundation of Australia; Cardiac Society of Australia and New Zealand **Last published:** 2016

Pathway for acute coronary syndrome (PACSA)

Published by: Emergency Care Institute; Agency for Clinical Innovation **Last published:** 2021

Online resources

1. QRISK3 *(external link)*

2. QRISK3-lifetime *(external link)*

Key articles

- Thygesen K, Alpert JS, Jaffe AS, et al. Fourth universal definition of myocardial infarction (2018). Glob Heart. 2018 Dec;13(4):305-38. Full text Abstract
- Byrne RA, Rossello X, Coughlan JJ, et al; ESC Scientific Document Group. 2023 ESC guidelines for the management of acute coronary syndromes. Eur Heart J. 2023 Oct 12;44(38):3720-826. Full text Abstract
- National Institute for Health and Care Excellence. Acute coronary syndromes. November 2020 [internet publication]. Full text
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Images

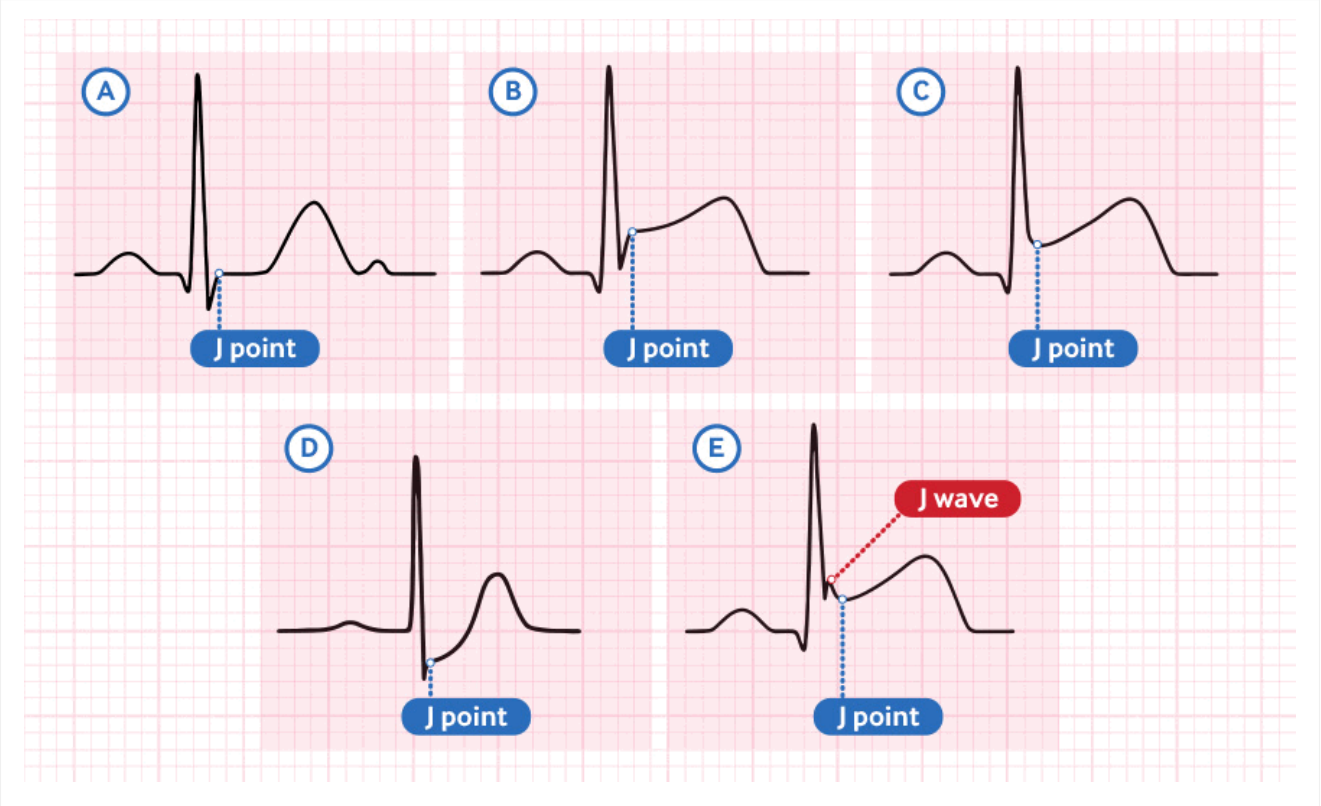

Figure 1: Identifying the J point on the ECG

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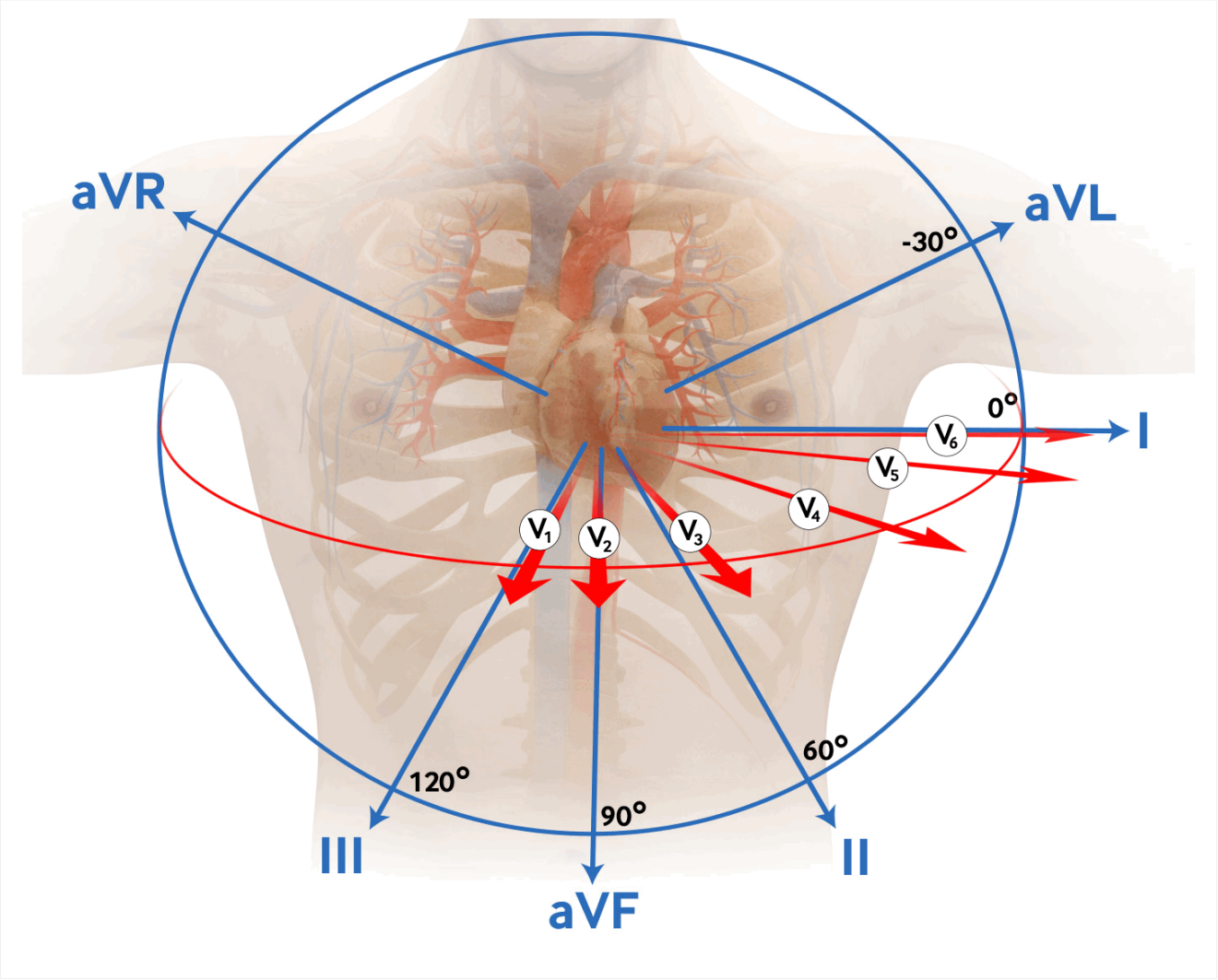

Figure 2: 12-lead ECG placement

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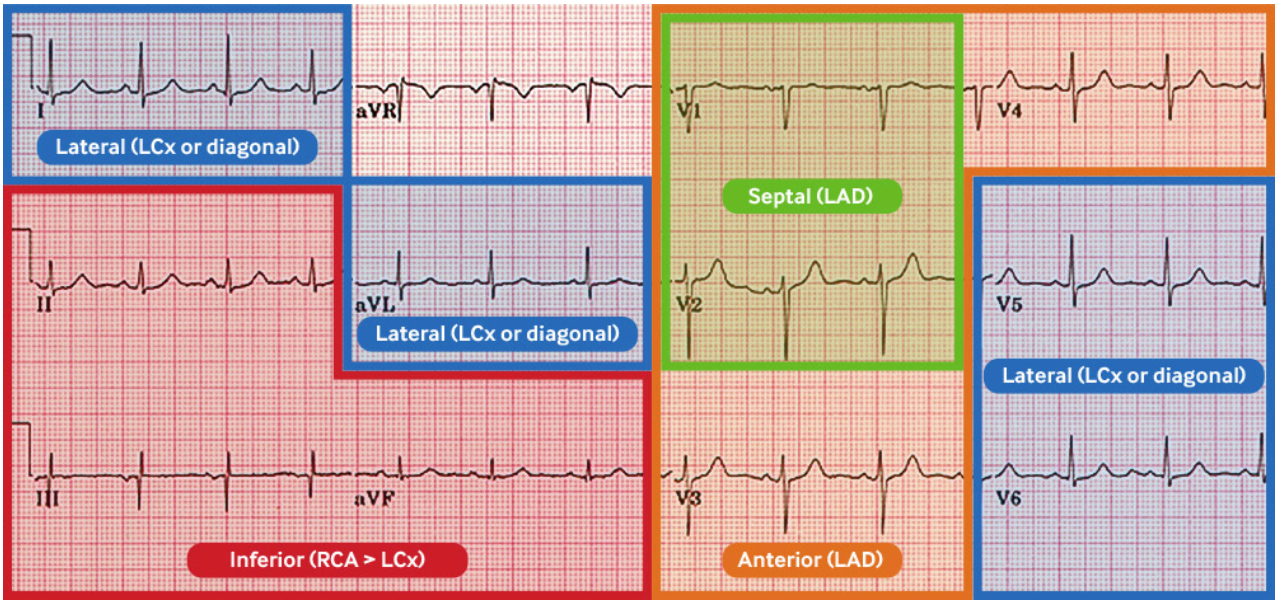

Figure 3: Coronary anatomy and ECG leads

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Leads	Anatomic location	Coronary artery
II, III, aVF	Inferior	RCA >> LCx
V2 - V4	Anterior	LAD
V1 - V4	Anteroseptal	LAD
I, aVL, V5, V6	Lateral	LCx > LAD
LBBB	Anterior	LAD
V4R	Right ventricle	RCA
V1, V2 depressions	Posterior	RCA >> LCx
Electrically silent		LCx

Figure 4: Coronary anatomy and ECG leads table

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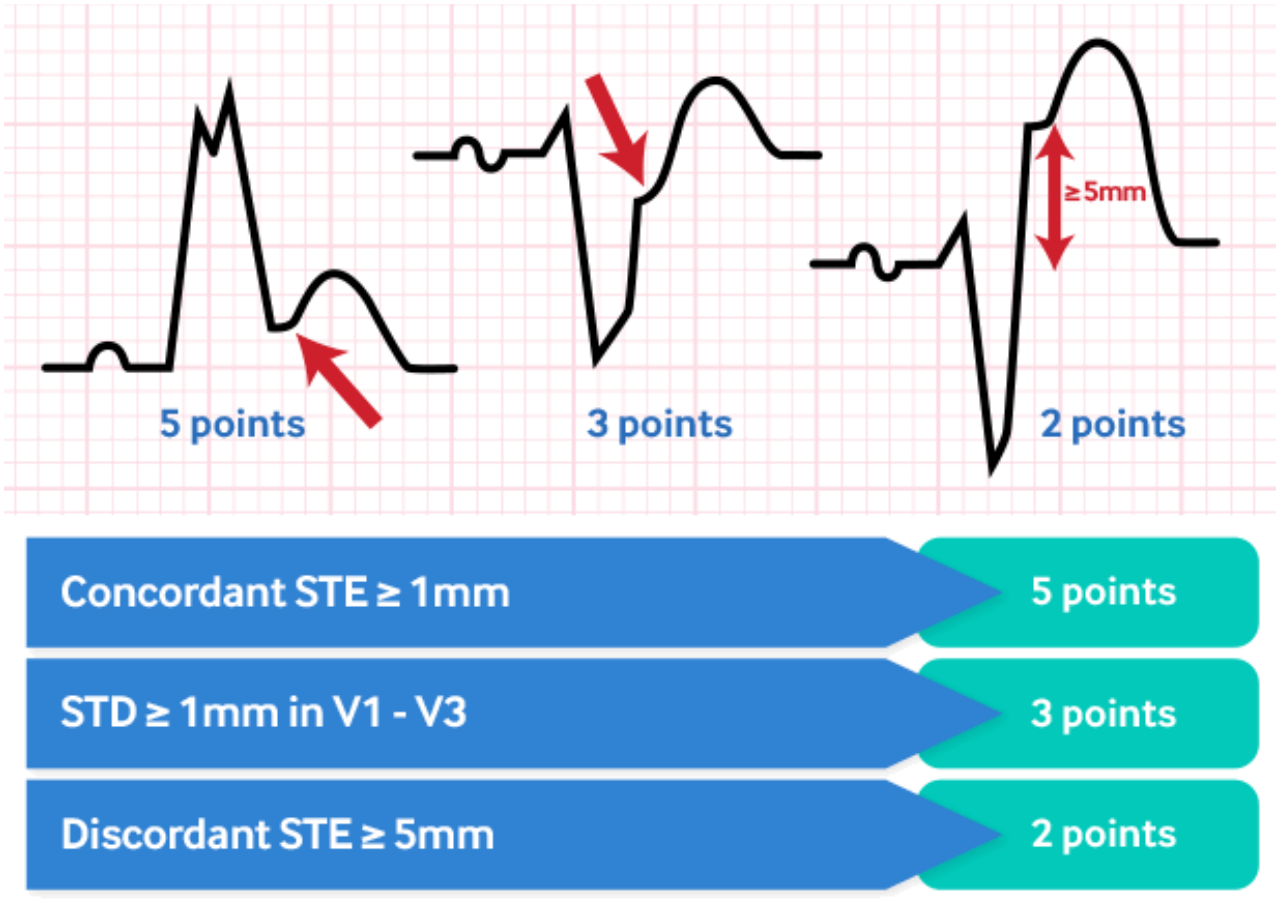

Figure 5: Sgarbossa criteria for MI in the presence of LBBB

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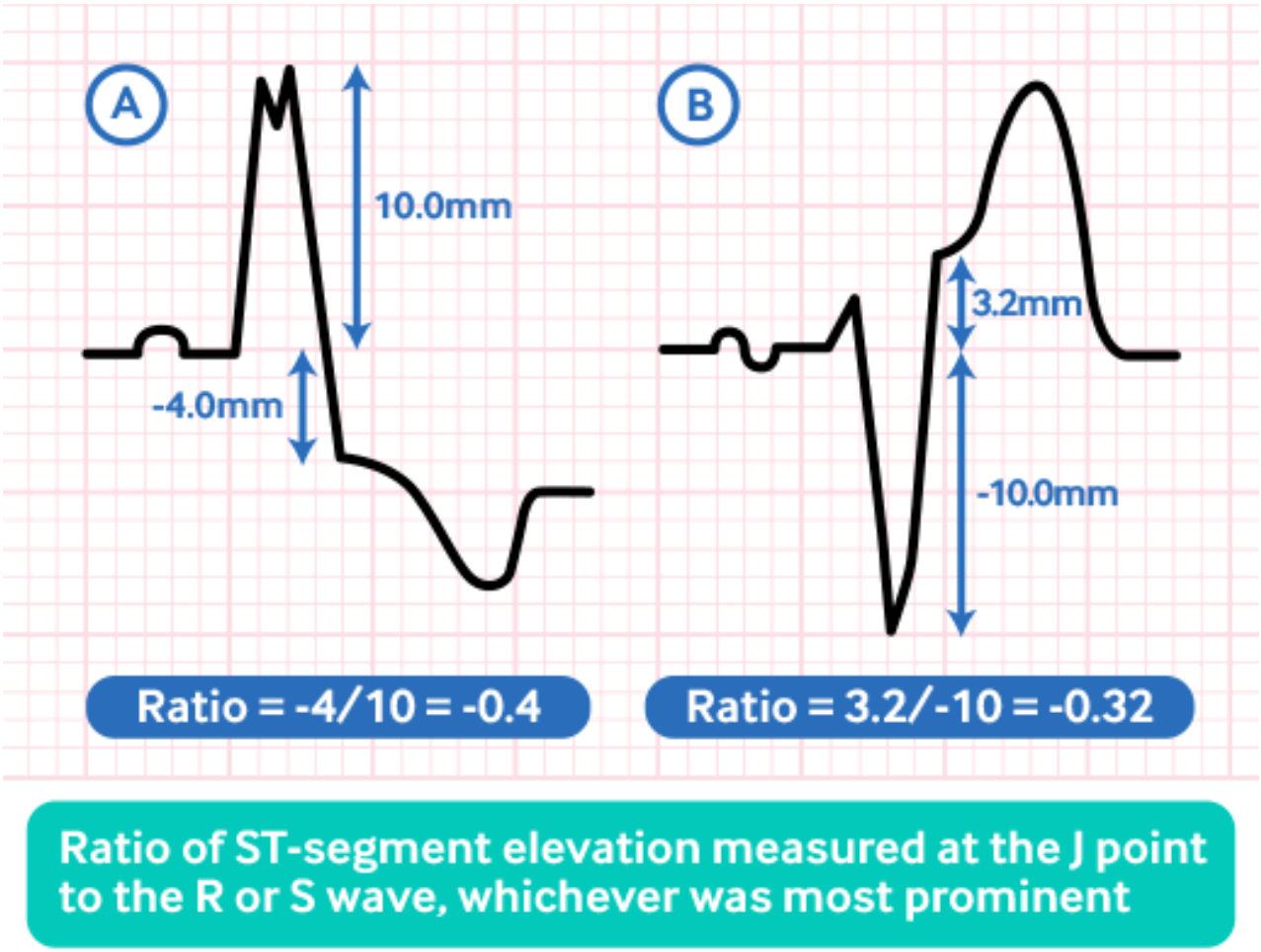

Figure 6: ST/S ratio under the modified Sgarbossa criteria

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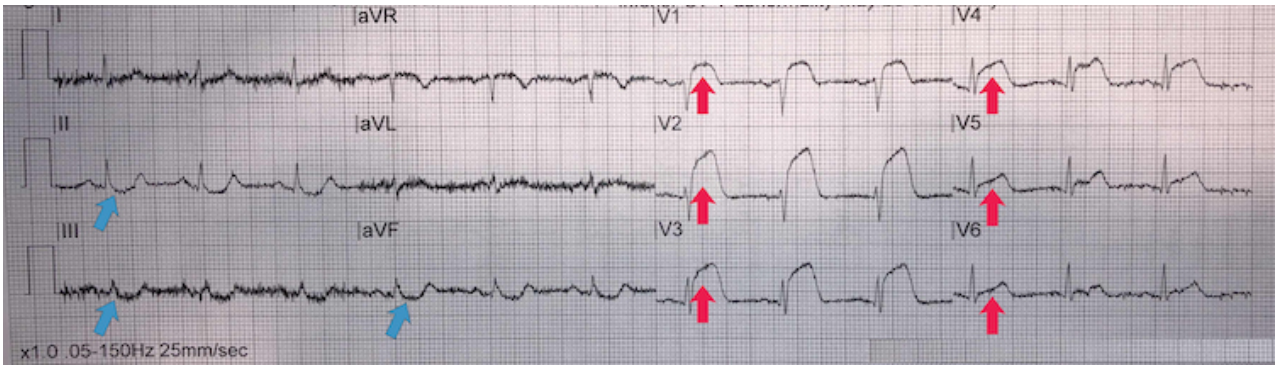

Figure 7: Anterior STEMI

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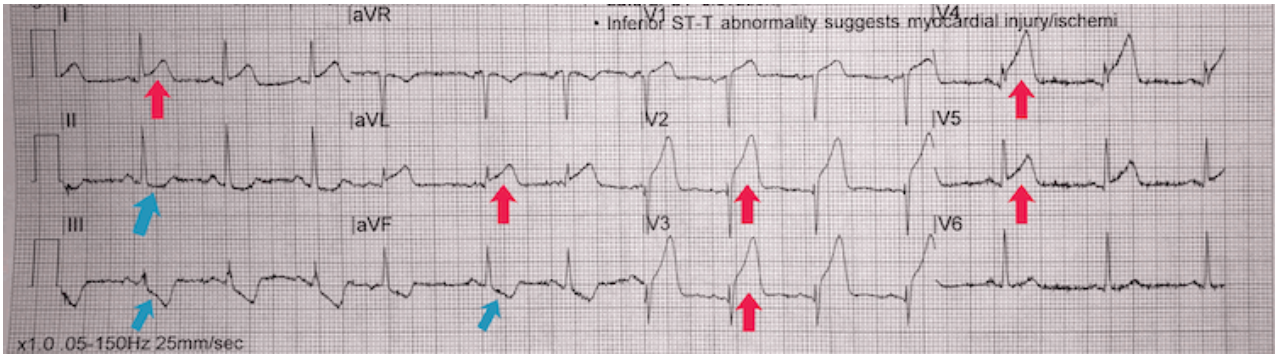

Figure 8: Anterolateral STEMI example I

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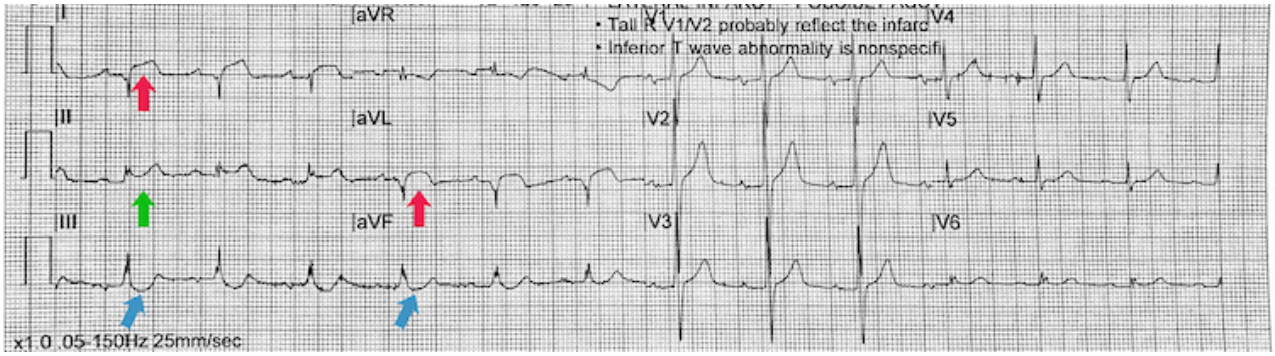

Figure 9: High lateral STEMI

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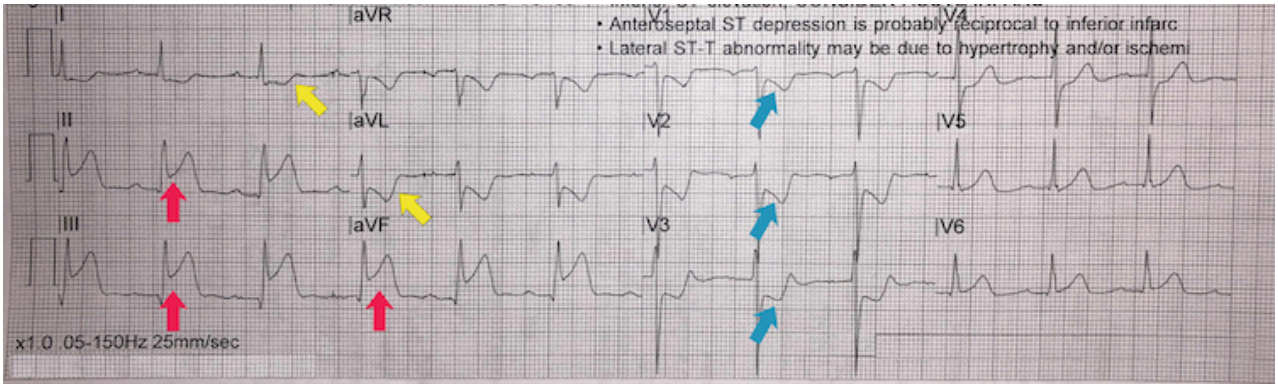

Figure 10: Inferoposterior STEMI#example II

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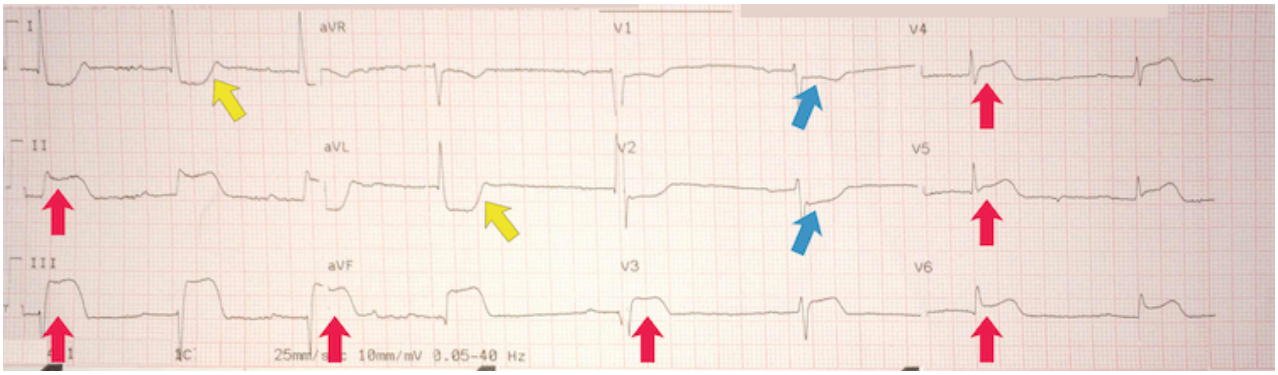

Figure 11: Inferoposterolateral STEMI example III

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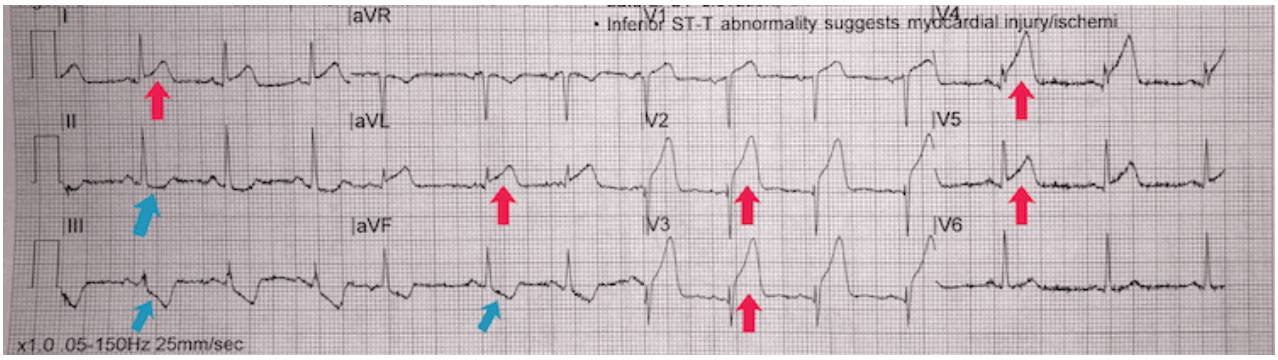

Figure 12: Anterolateral STEMI example II

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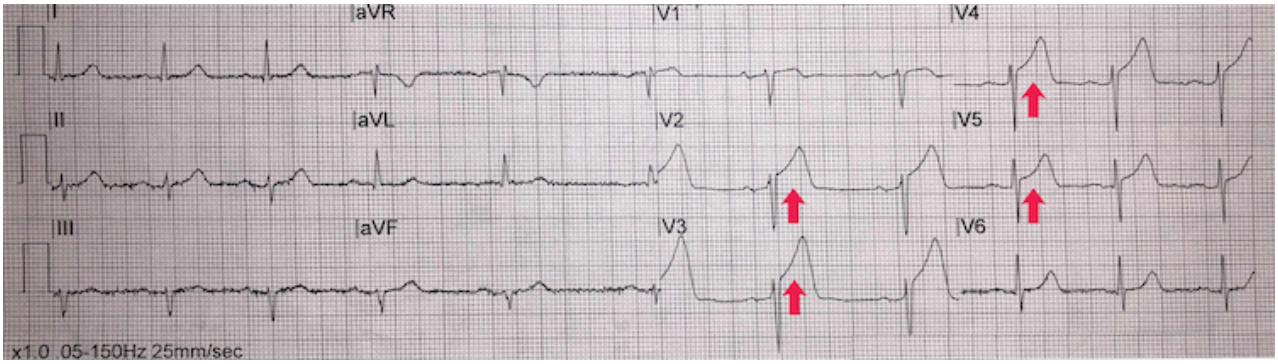

Figure 13: Anteroseptal STEMI example I

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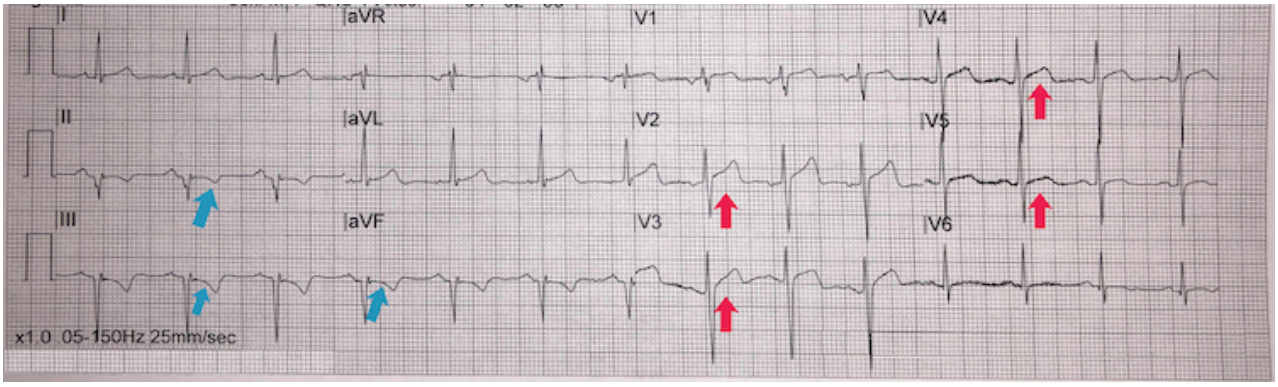

Figure 14: Anteroseptal STEMI example II

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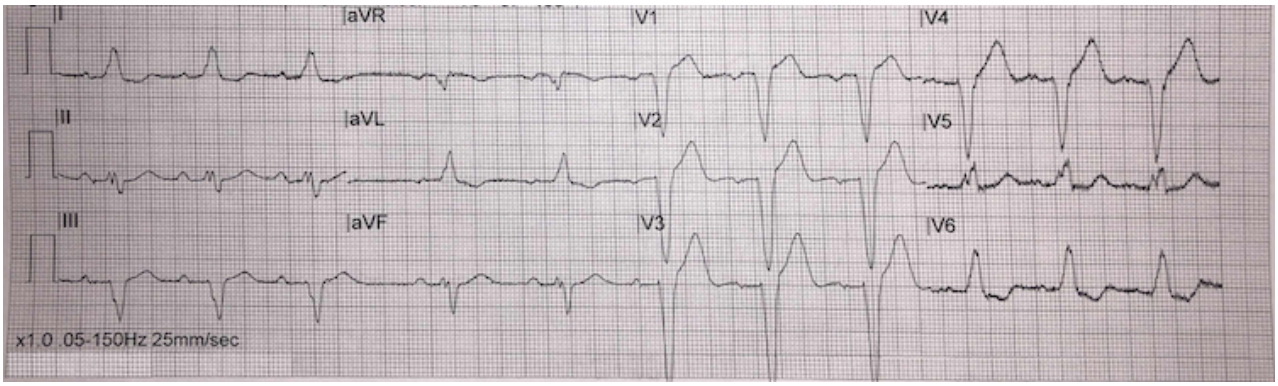

Figure 15: Left bundle branch block example I

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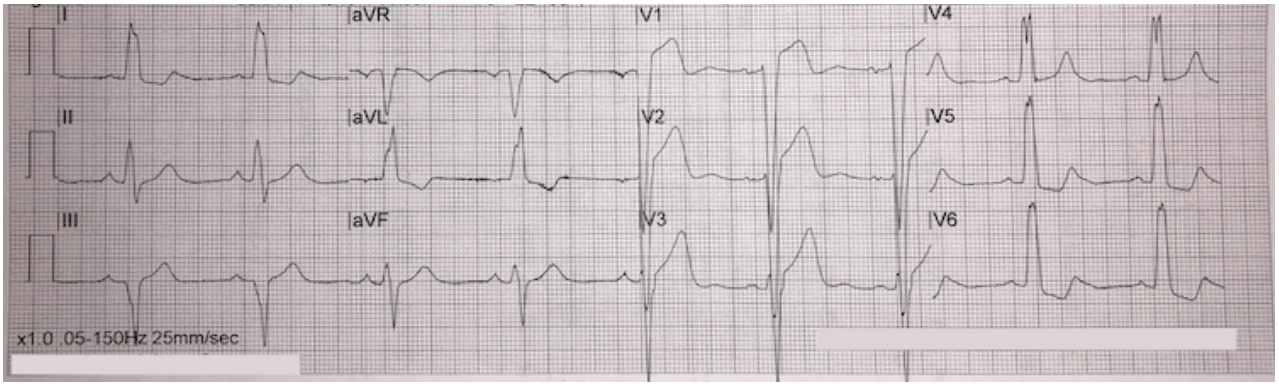

Figure 16: Left bundle branch block example II

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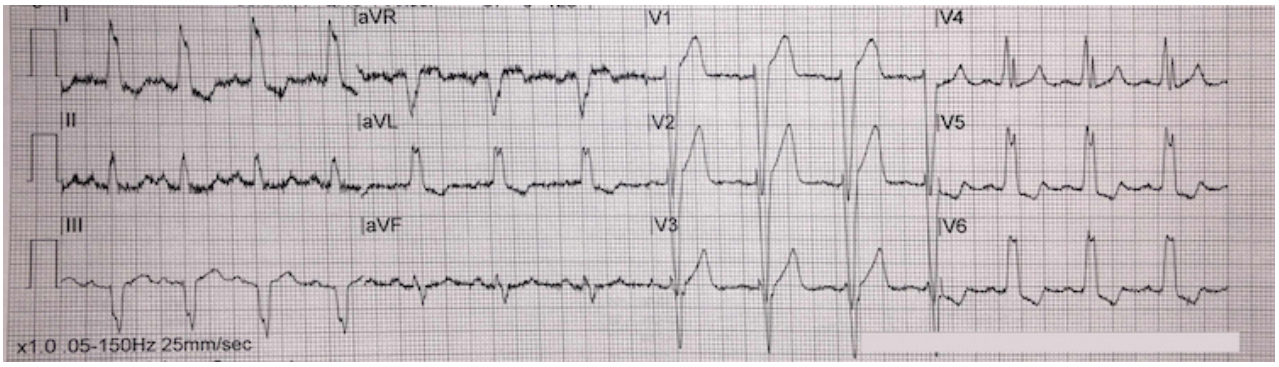

Figure 17: Left bundle branch block example III

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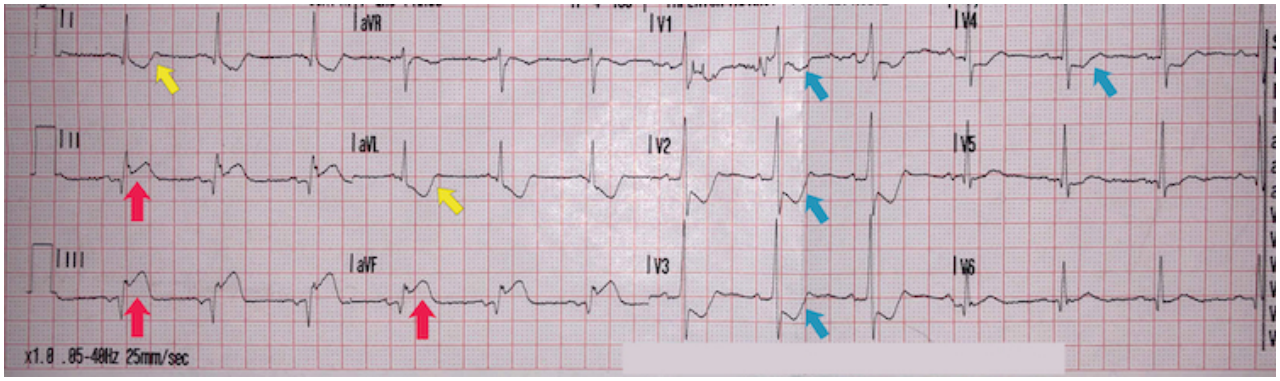

Figure 18: Inferoposterior STEMI example I

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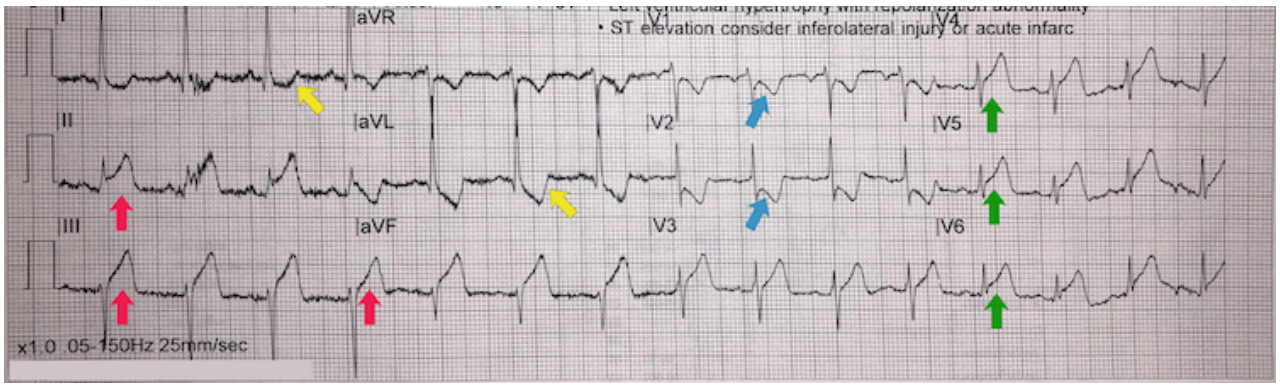

Figure 19: Inferoposterolateral STEMI example I

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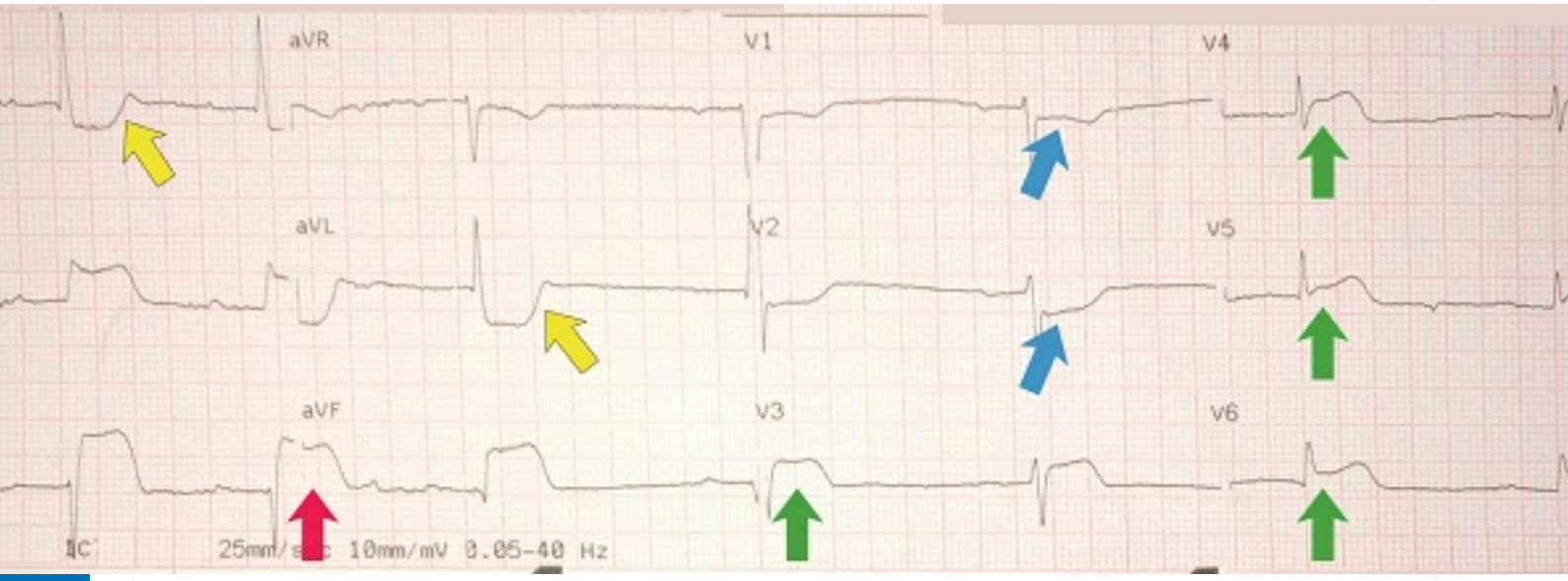

Figure 20: Inferoposterolateral STEMI example II

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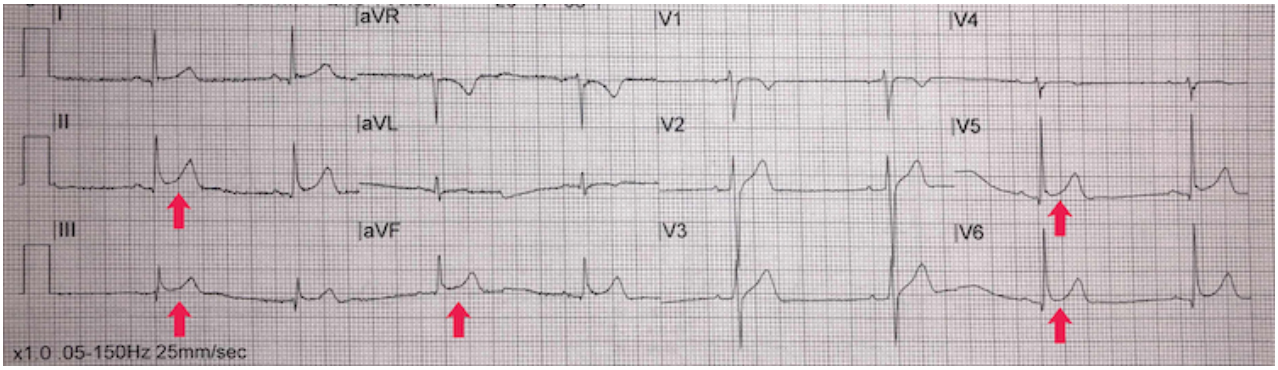

Figure 21: Possible' inferolateral STEMI

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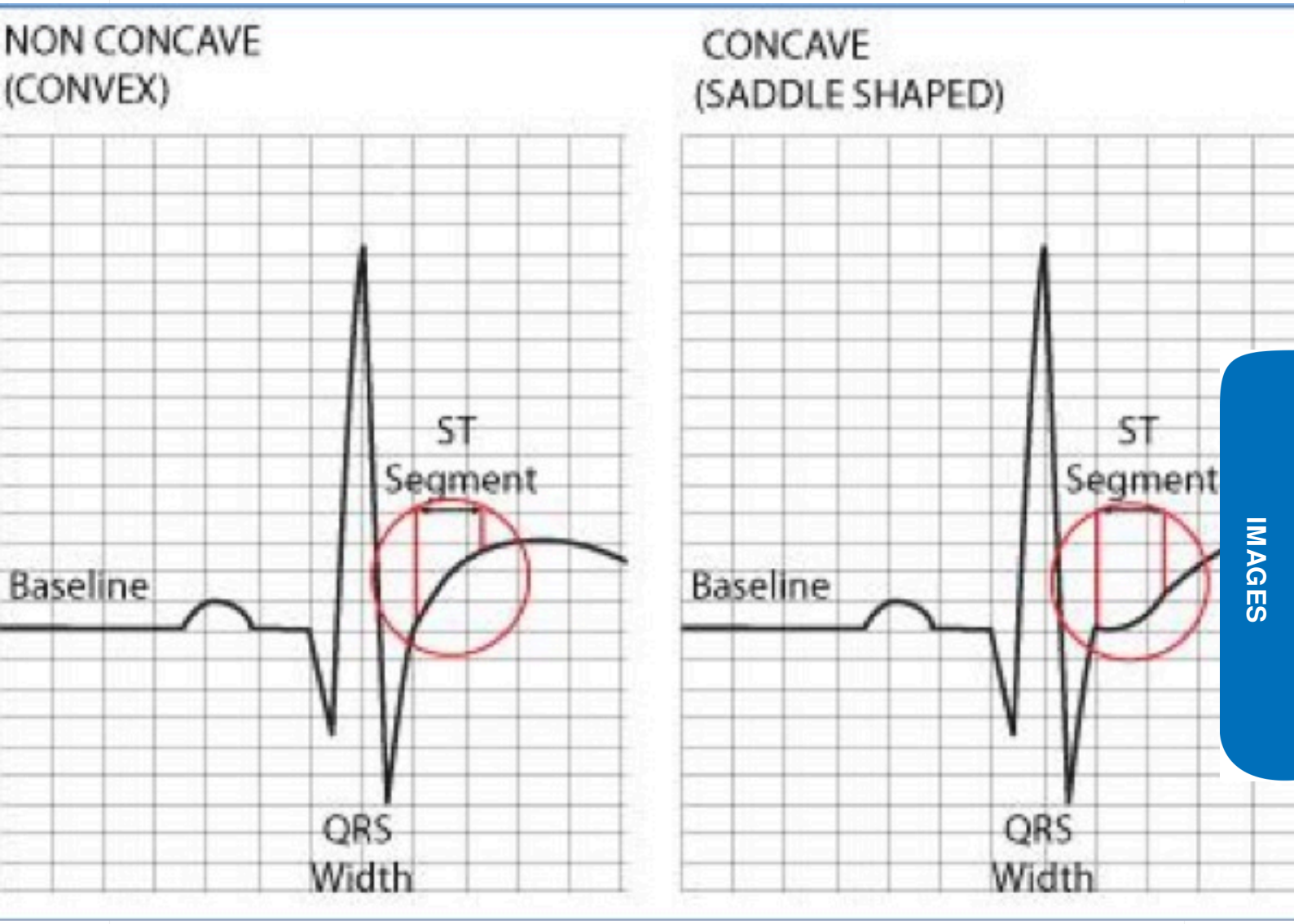

Figure 22: Possible' inferolateral STEMI: ST-segment shift

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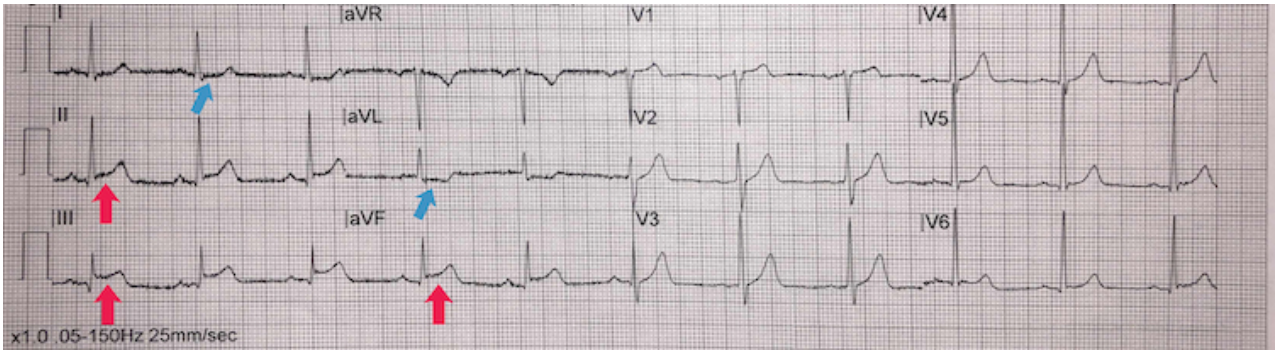

Figure 23: Inferior STEMI example I

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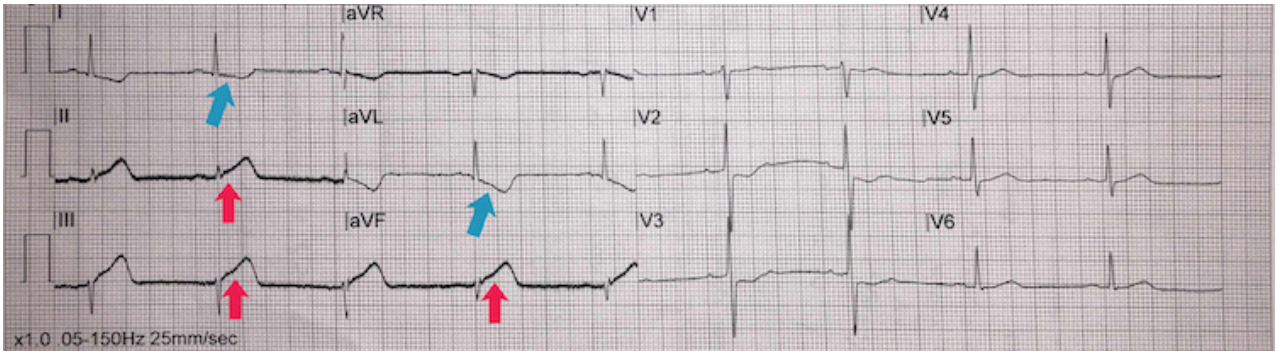

Figure 24: Inferior STEMI example II

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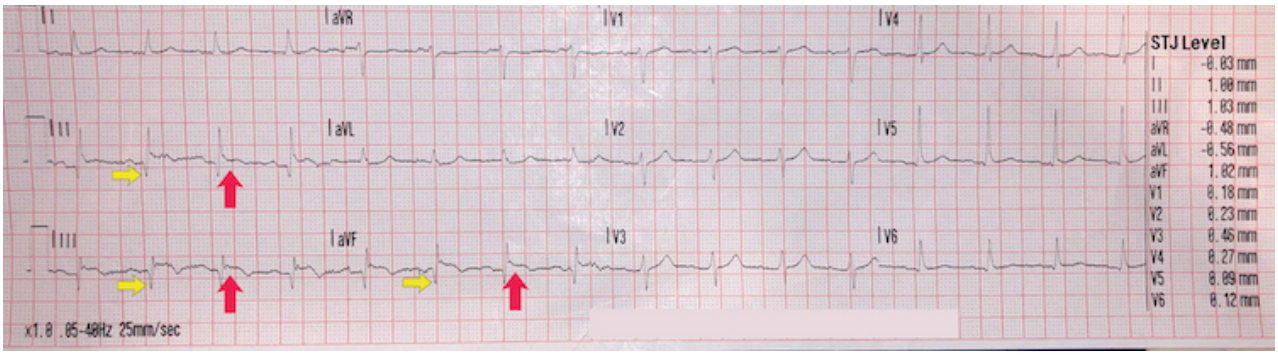

Figure 25: Inferior STEMI example III

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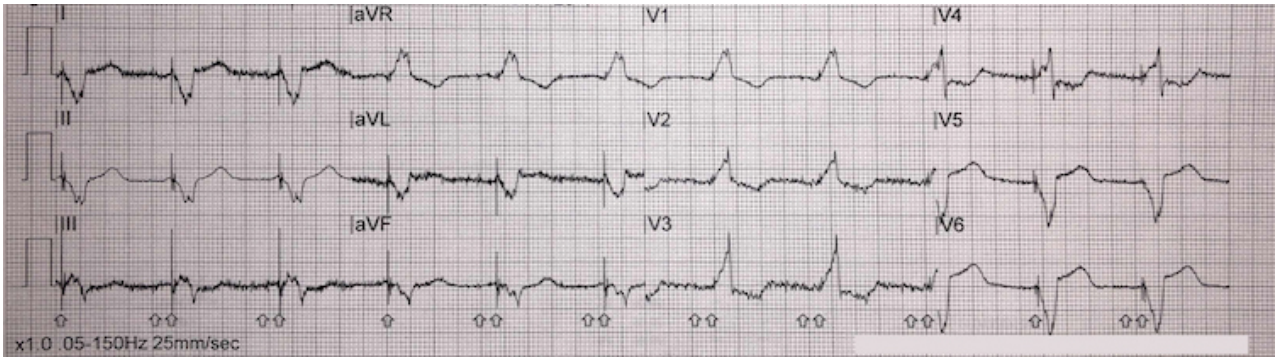

Figure 26: Paced rhythm example I

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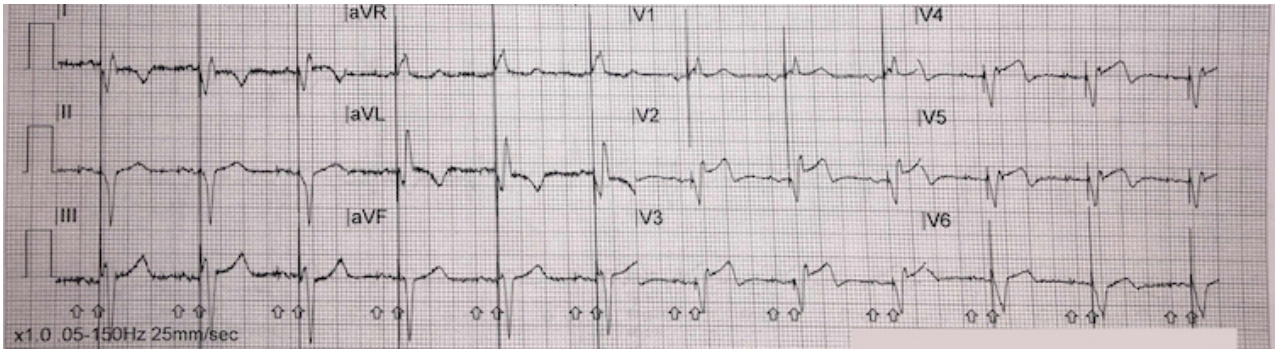

Figure 27: Paced rhythm example II

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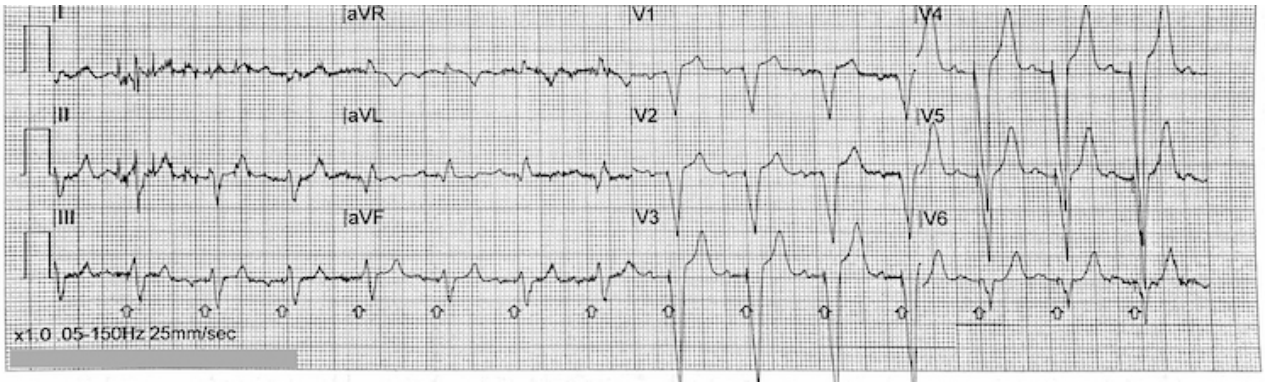

Figure 28: Paced rhythm example III

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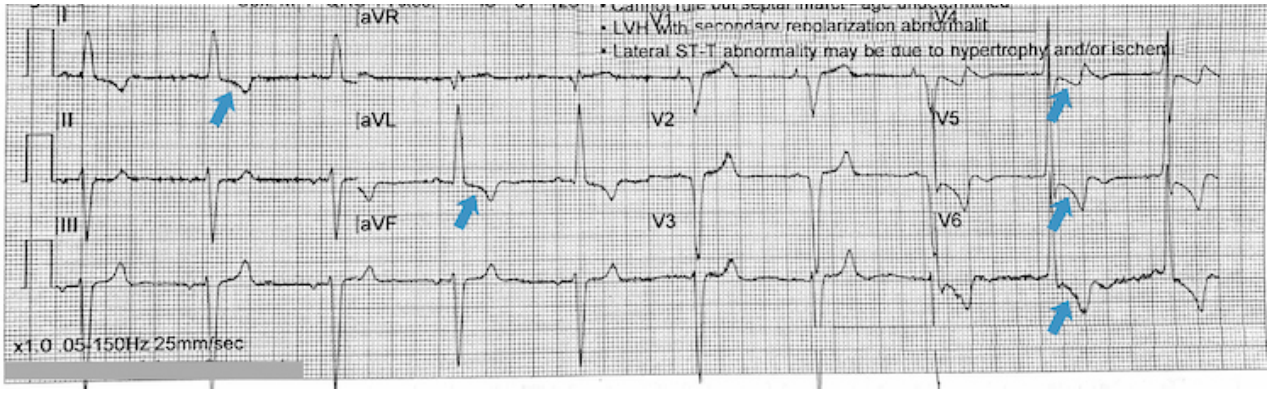

Figure 29: Left ventricular hypertrophy example I

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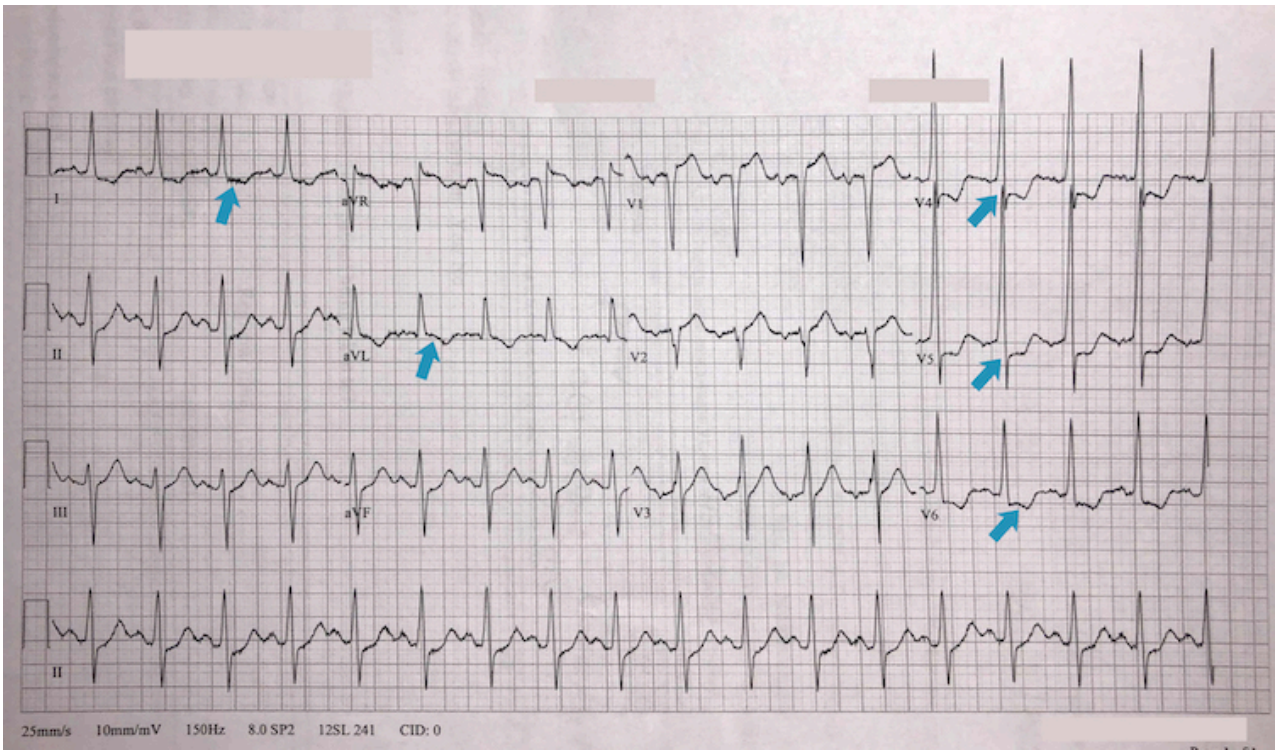

Figure 30: Left ventricular hypertrophy example II

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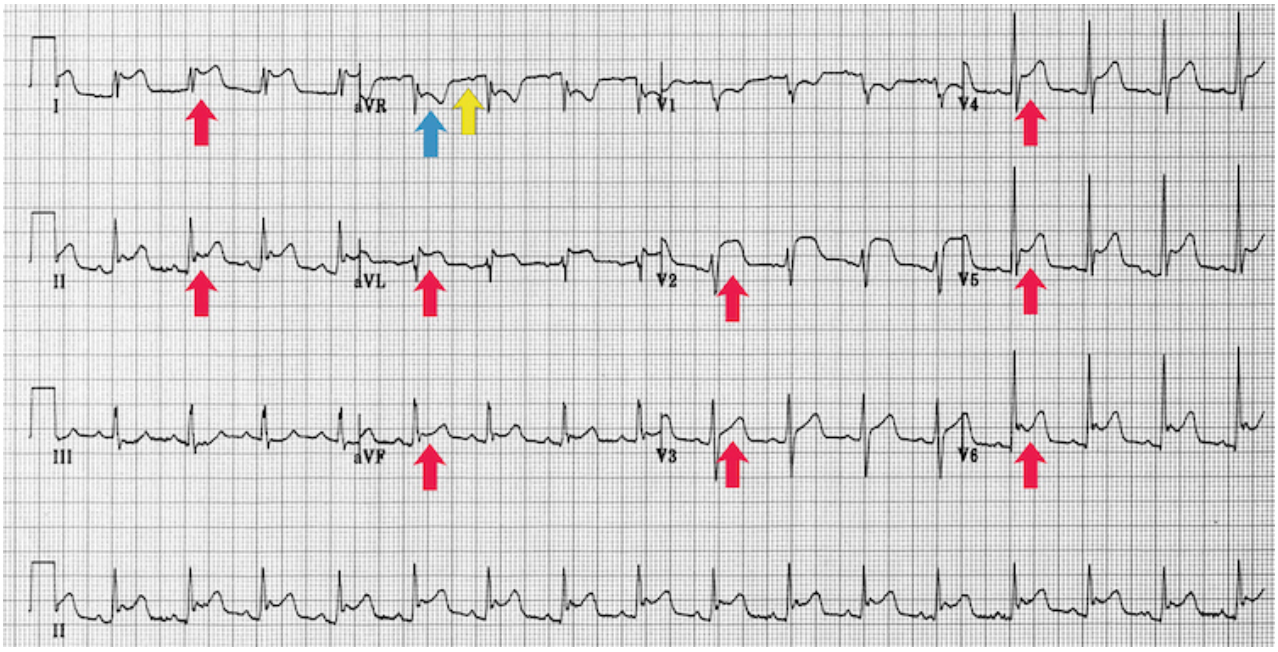

Figure 31: Acute pericarditis

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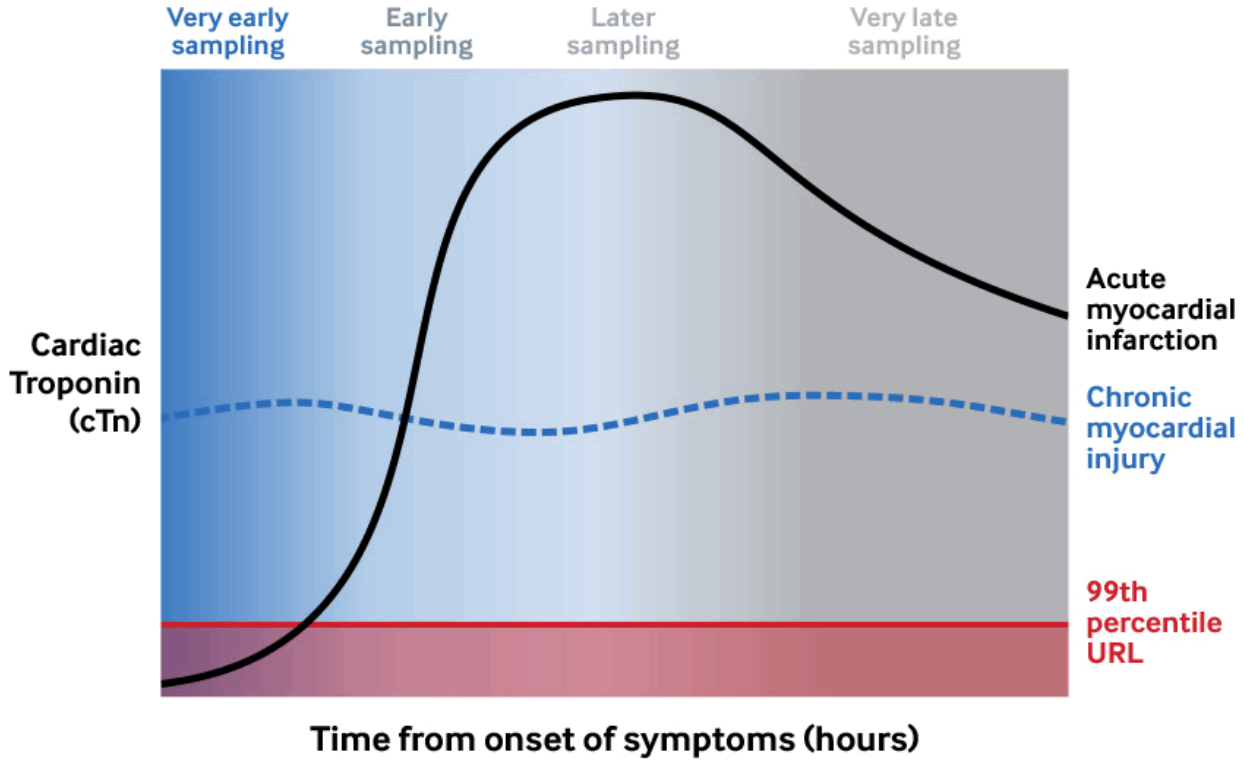

Figure 32: Cardiac troponin kinetics after acute myocardial injury including acute MI

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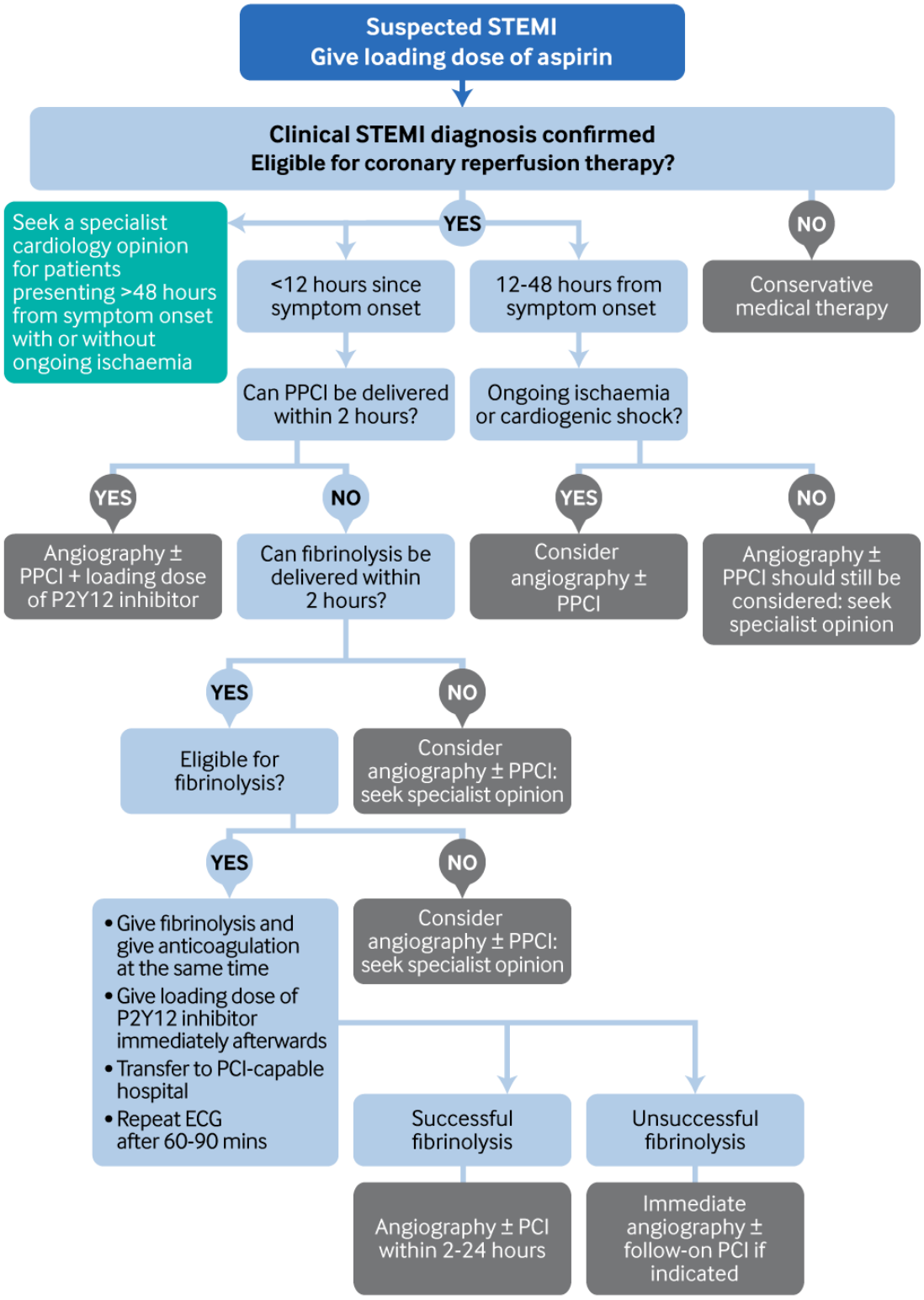

Figure 33: Selection of the most appropriate reperfusion strategy. PPCI, primary percutaneous coronary intervention

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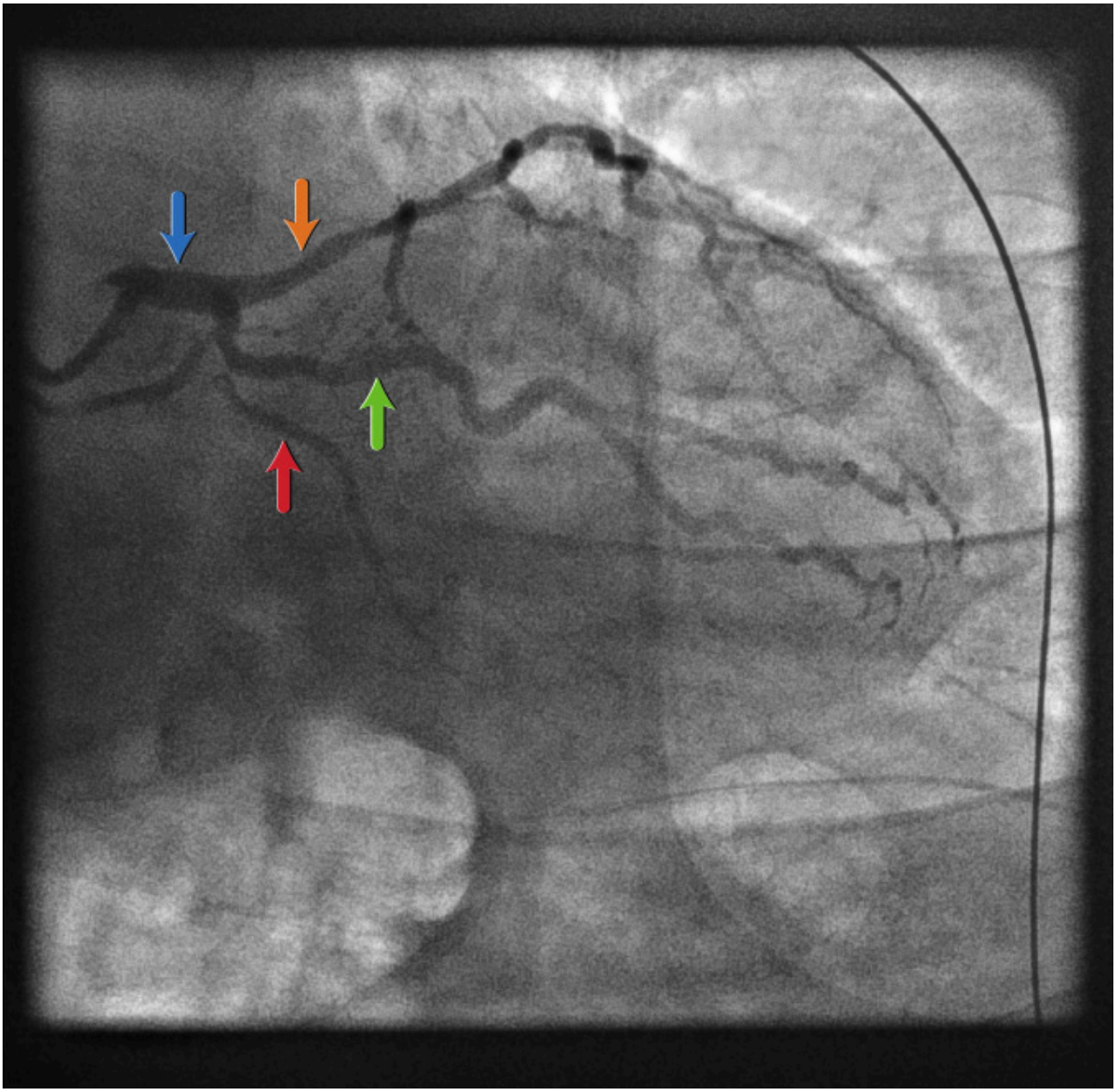

Figure 34: Left main coronary artery: right anterior oblique view

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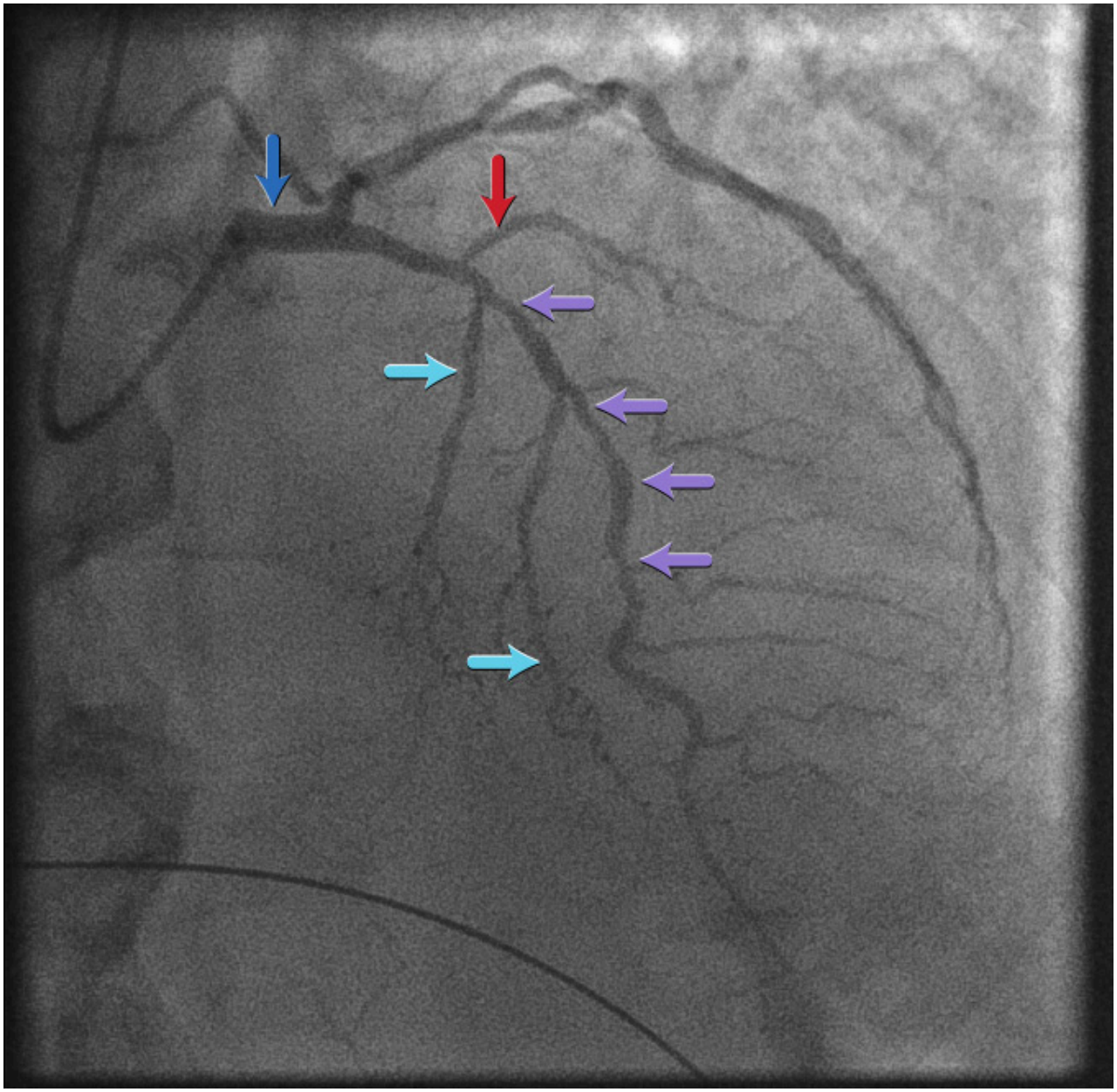

Figure 35: Left main coronary artery: posterior anterior cranial view

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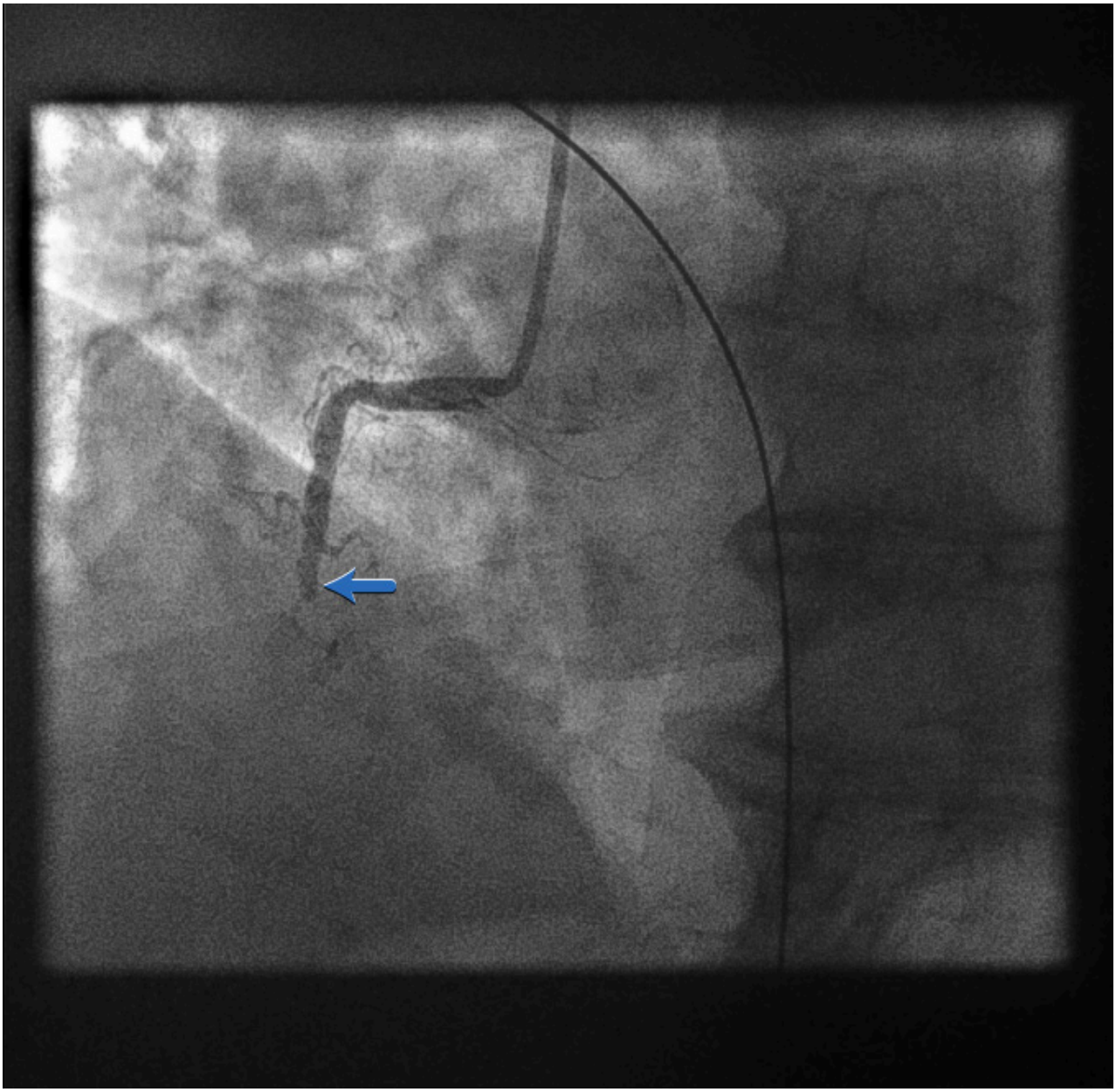

Figure 36: Right coronary artery: left anterior oblique view

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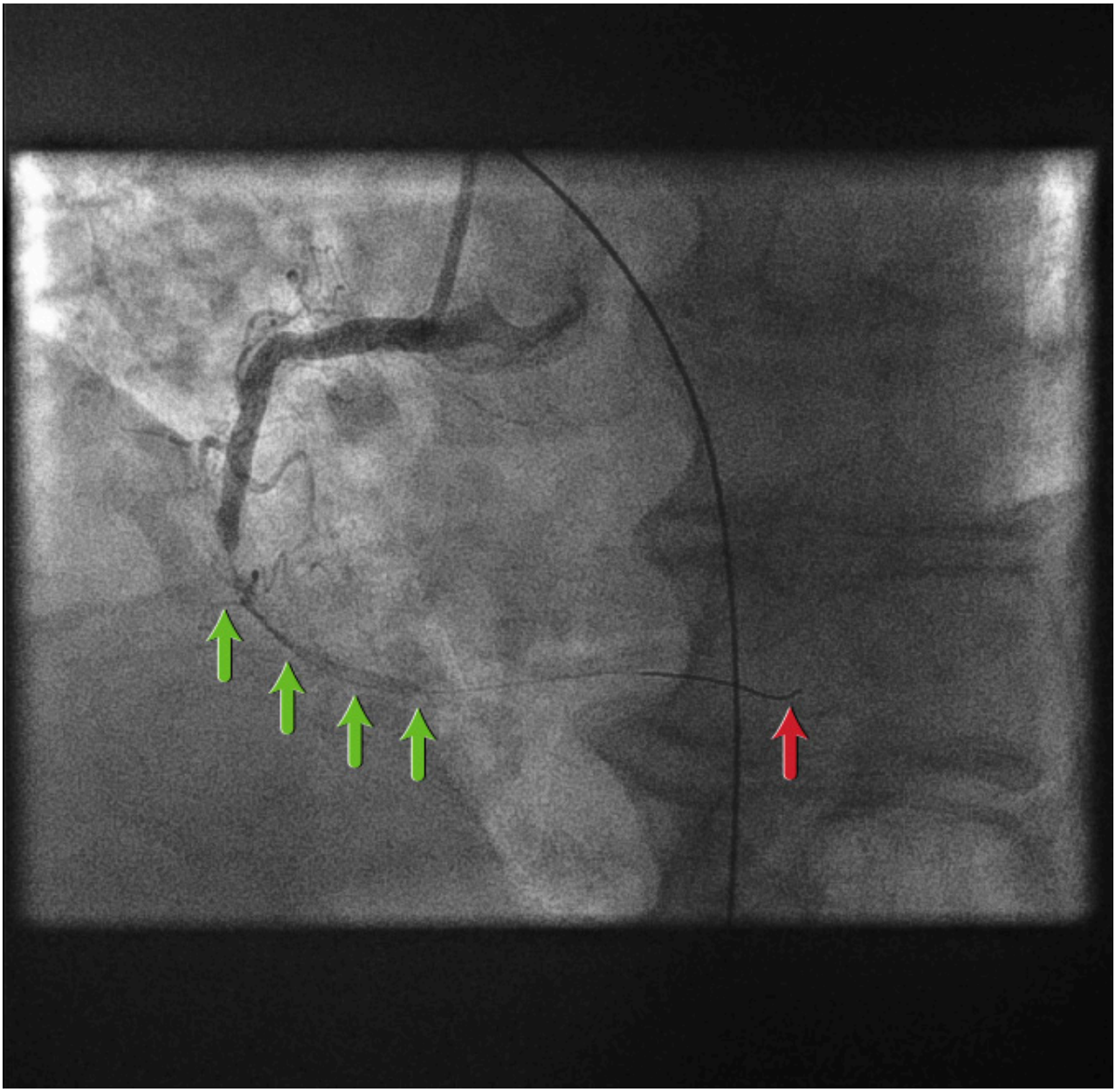

Figure 37: Coronary guidewire deployment

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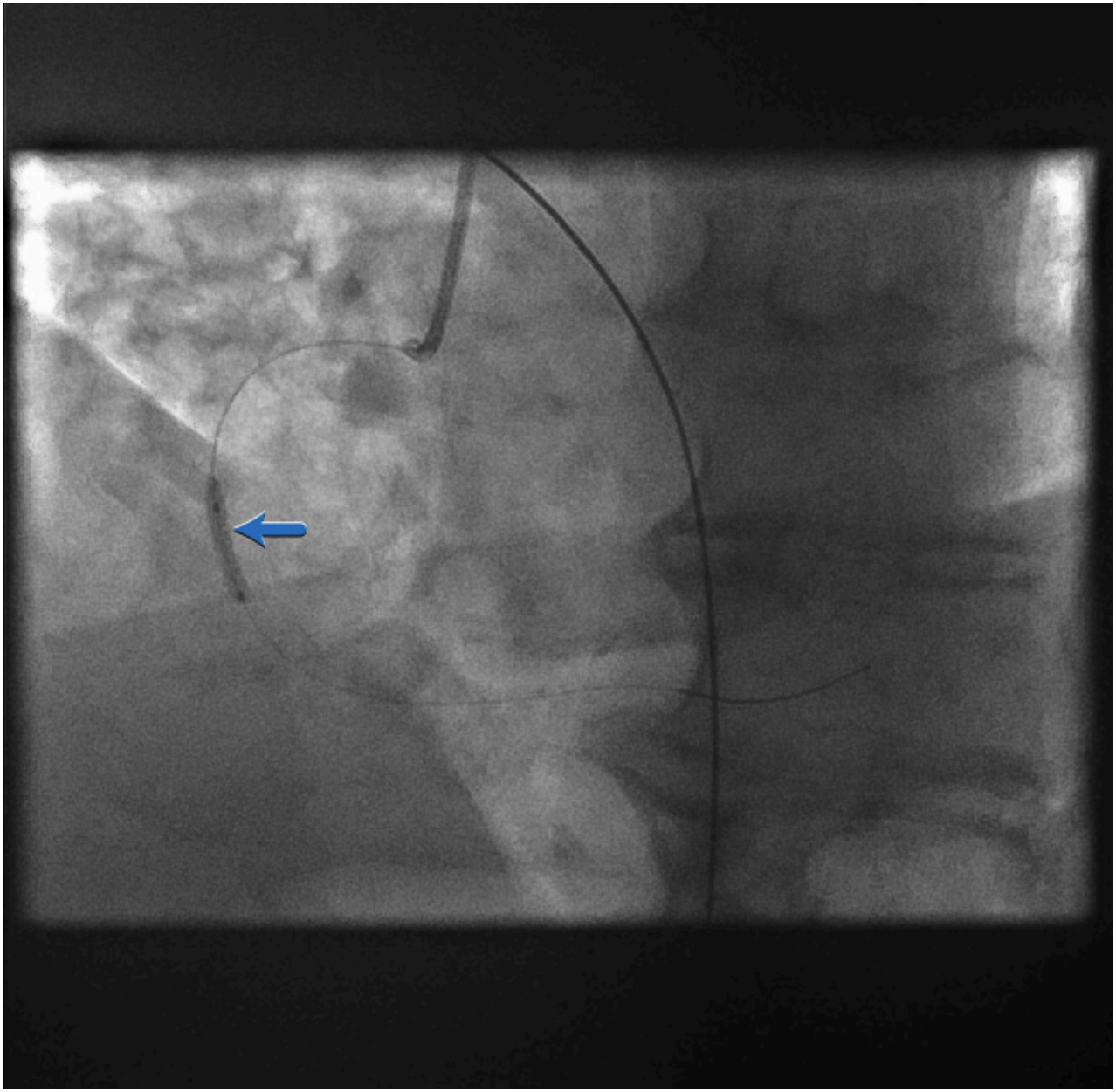

Figure 38: First balloon dilatation

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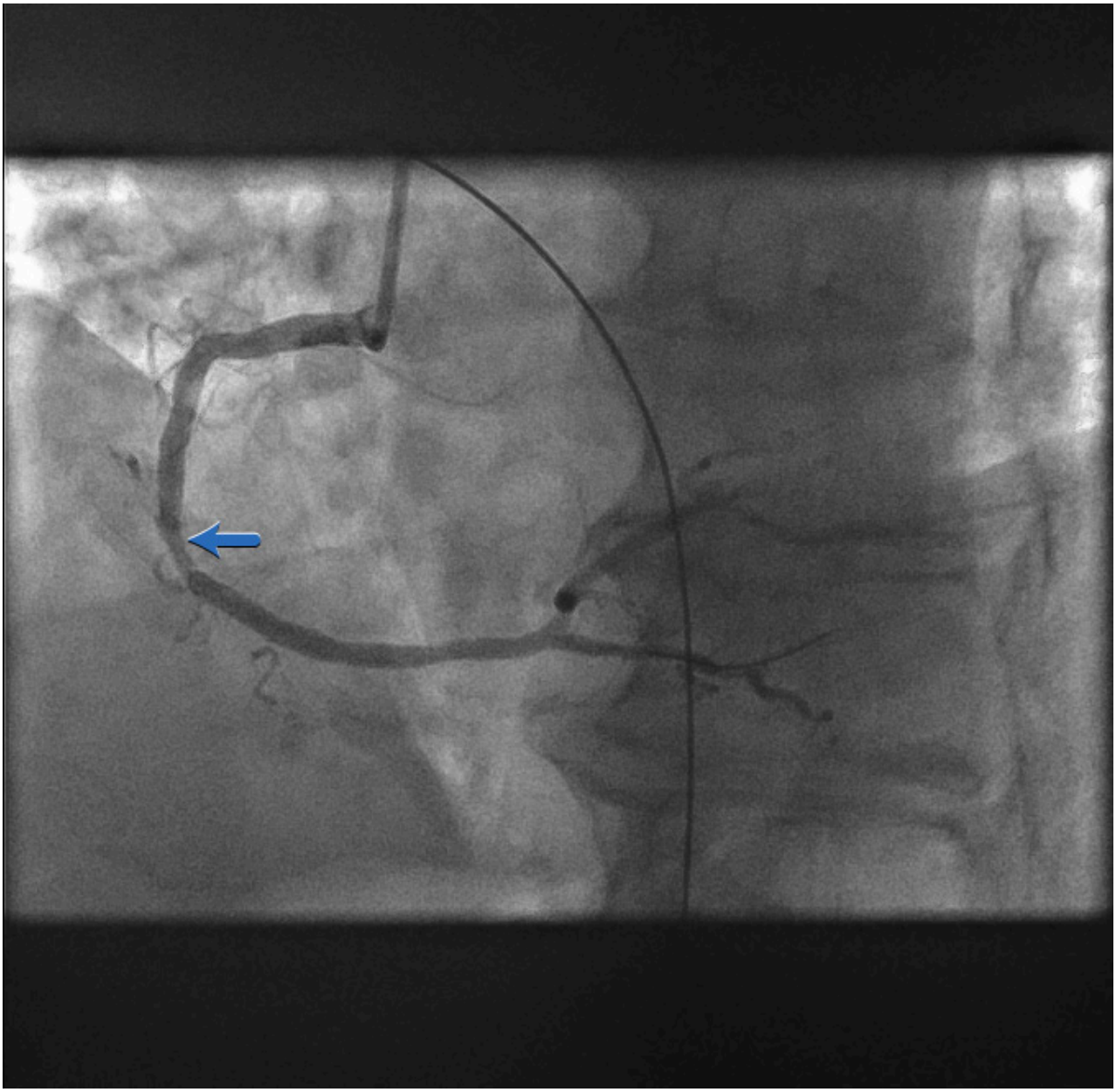

Figure 39: Restoration of coronary flow

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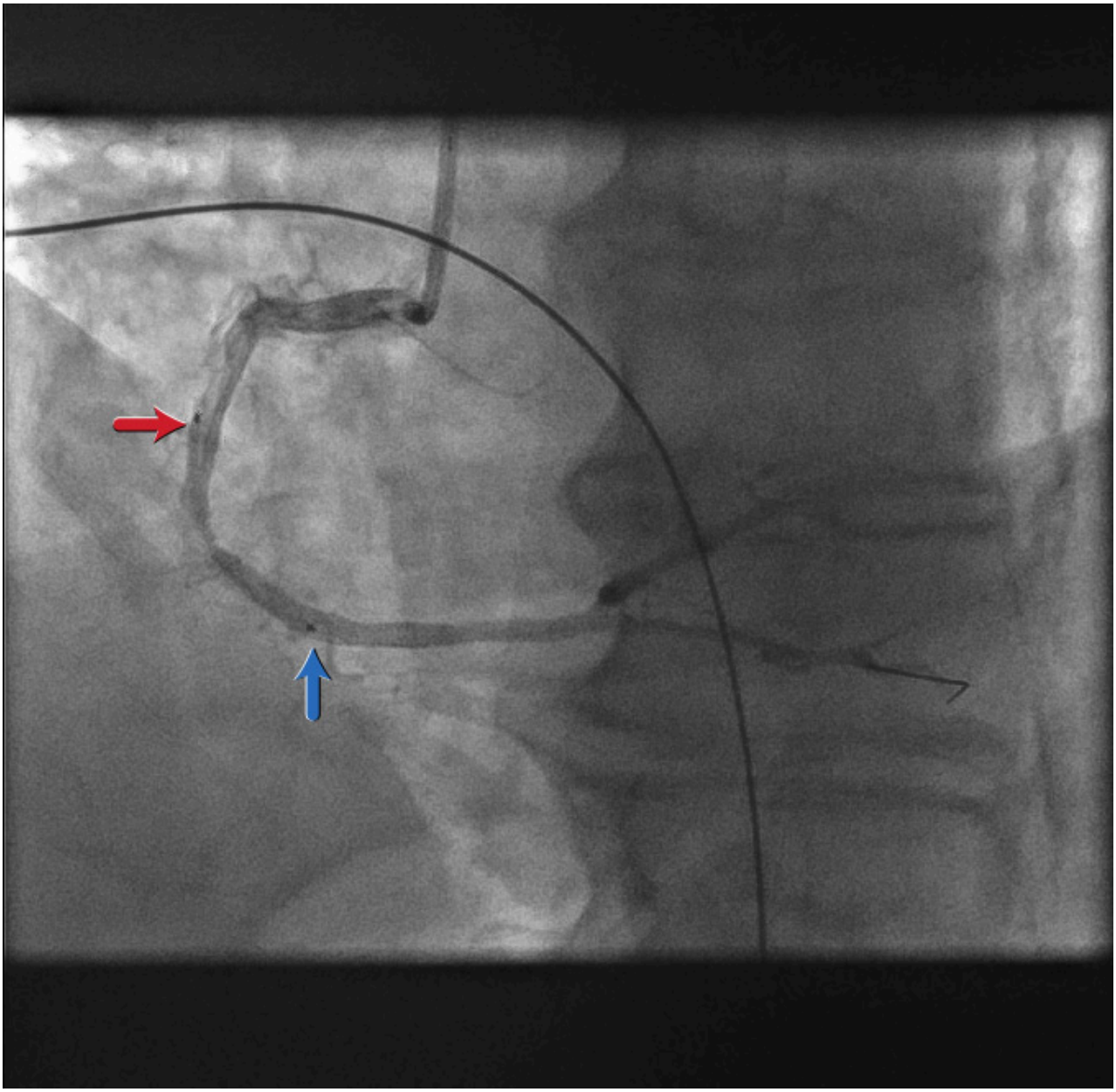

Figure 40: Stent positioning

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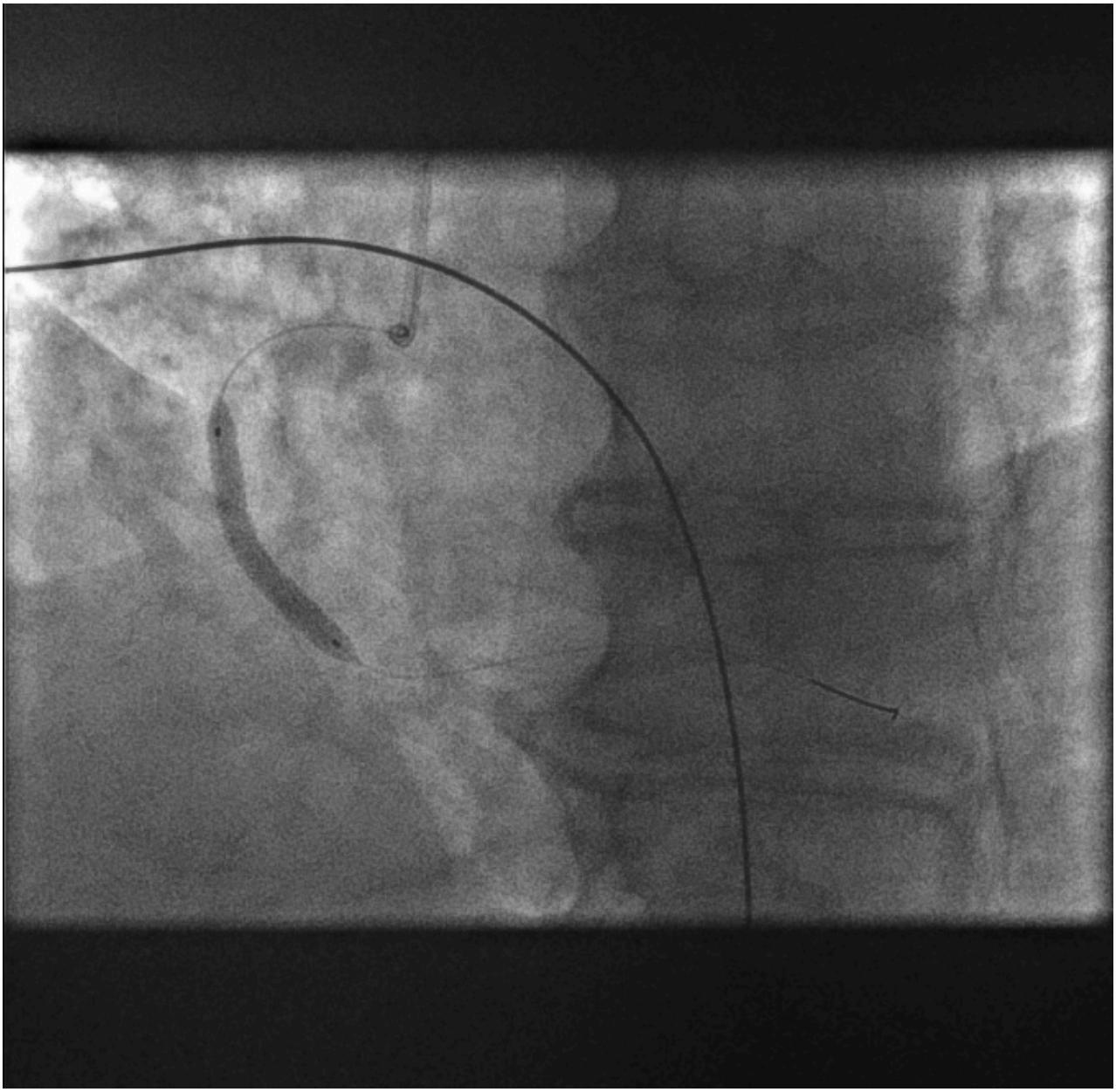

Figure 41: Stent deployed

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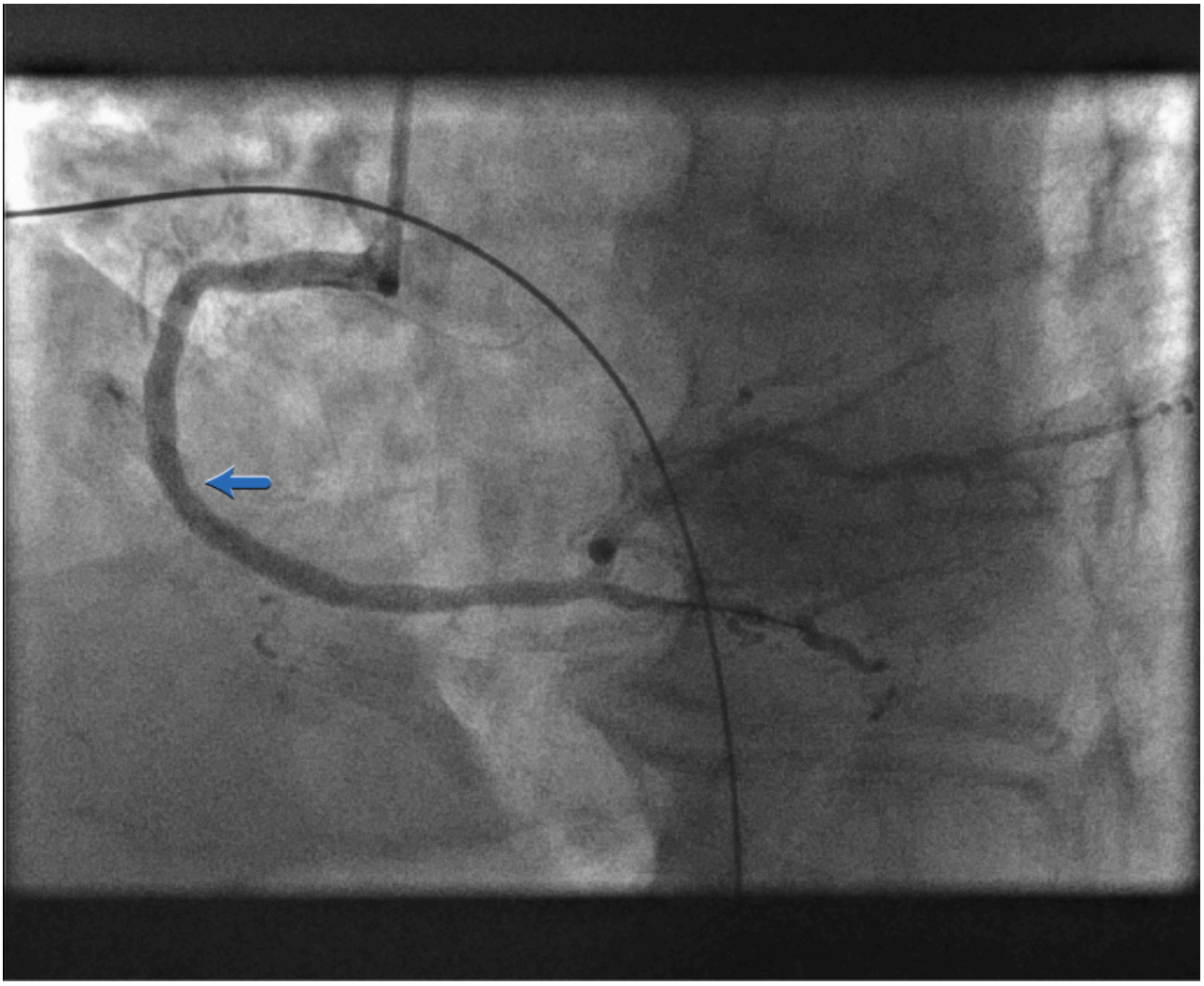

Figure 42: After stent deployment

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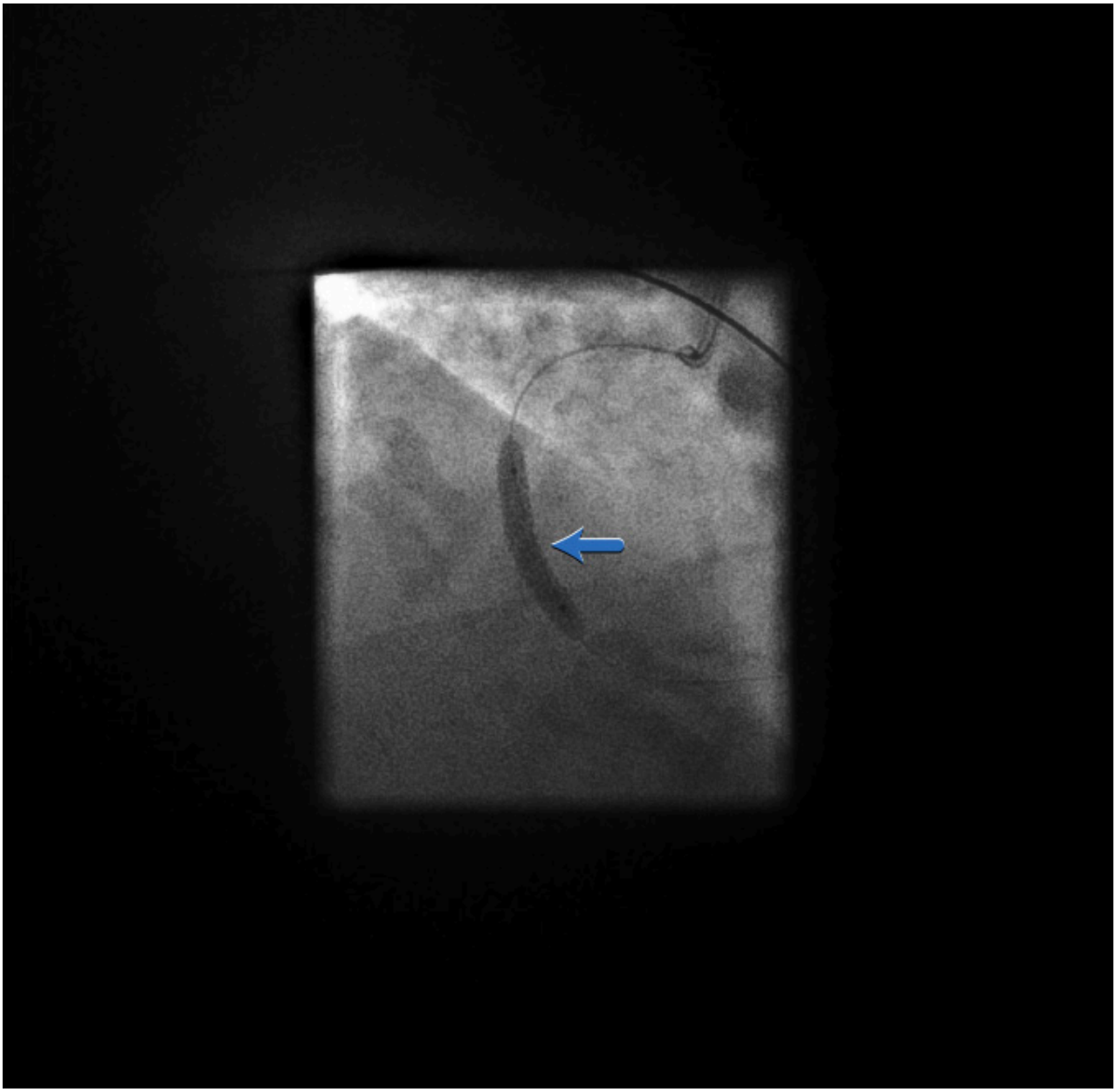

Figure 43: Stent post-dilatation

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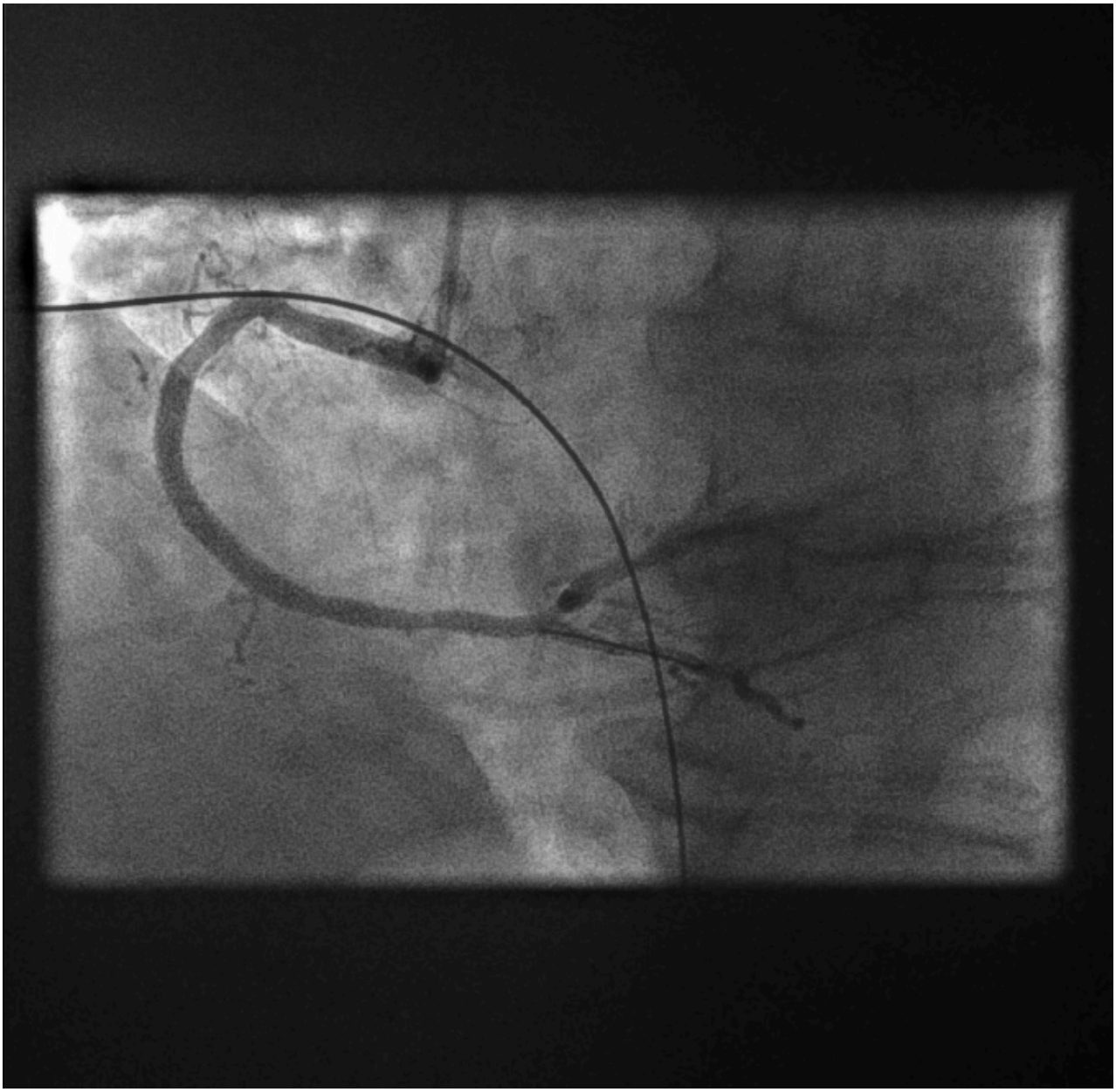

Figure 44: RCA primary angioplasty: final result

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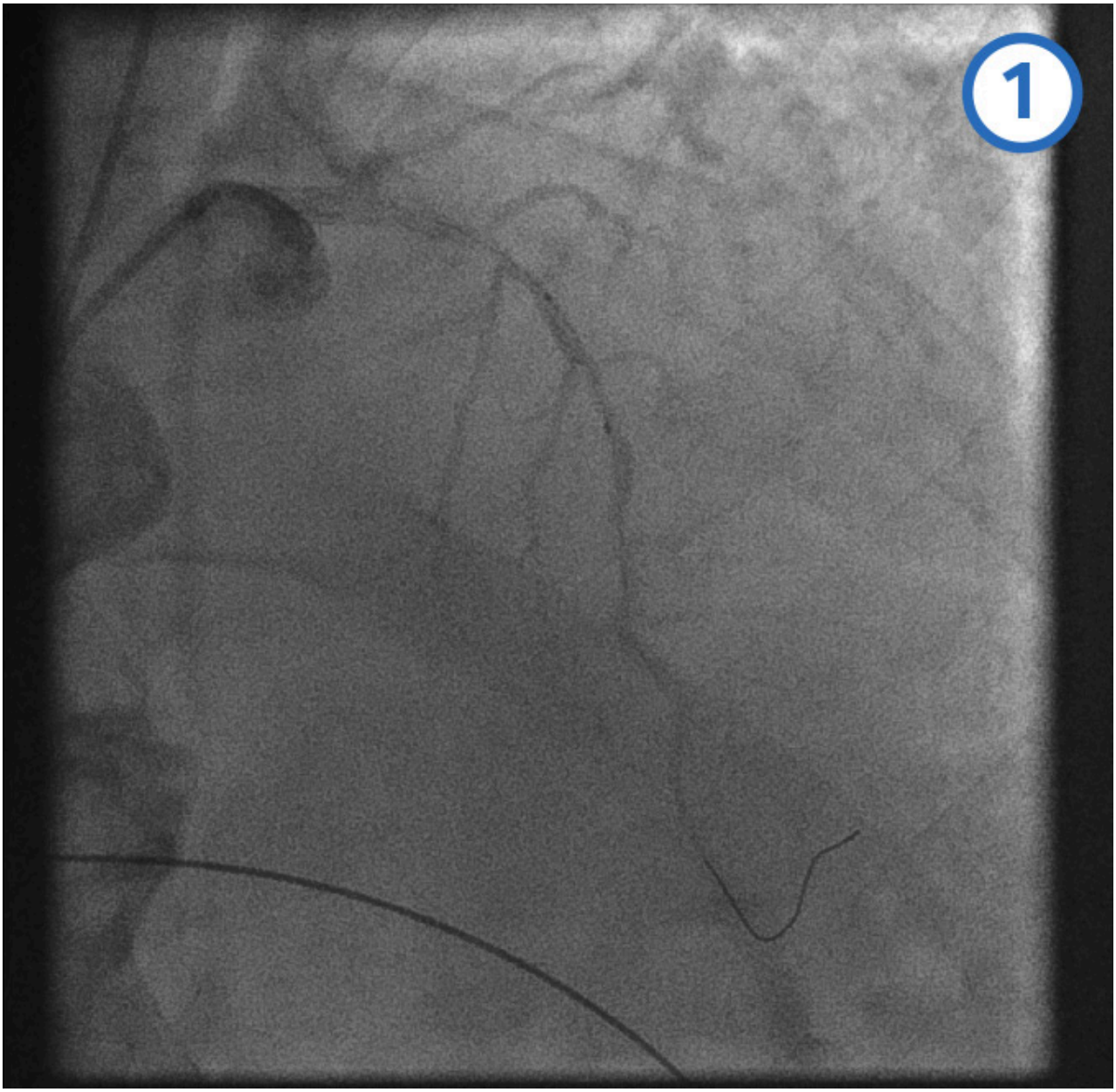

Figure 45: Left anterior descending artery pre-dilatation image 1

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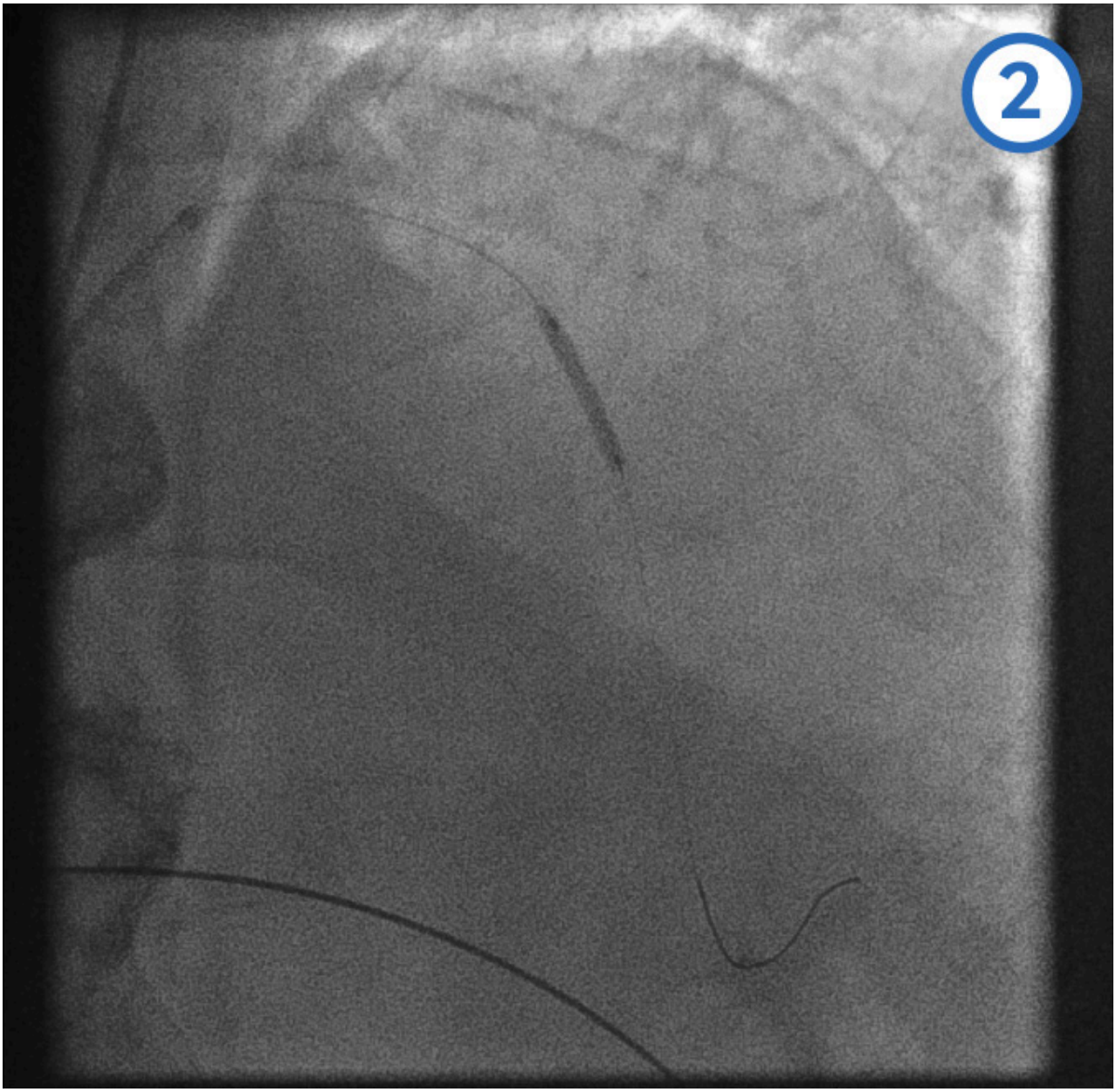

Figure 46: Left anterior descending artery pre-dilatation image#2

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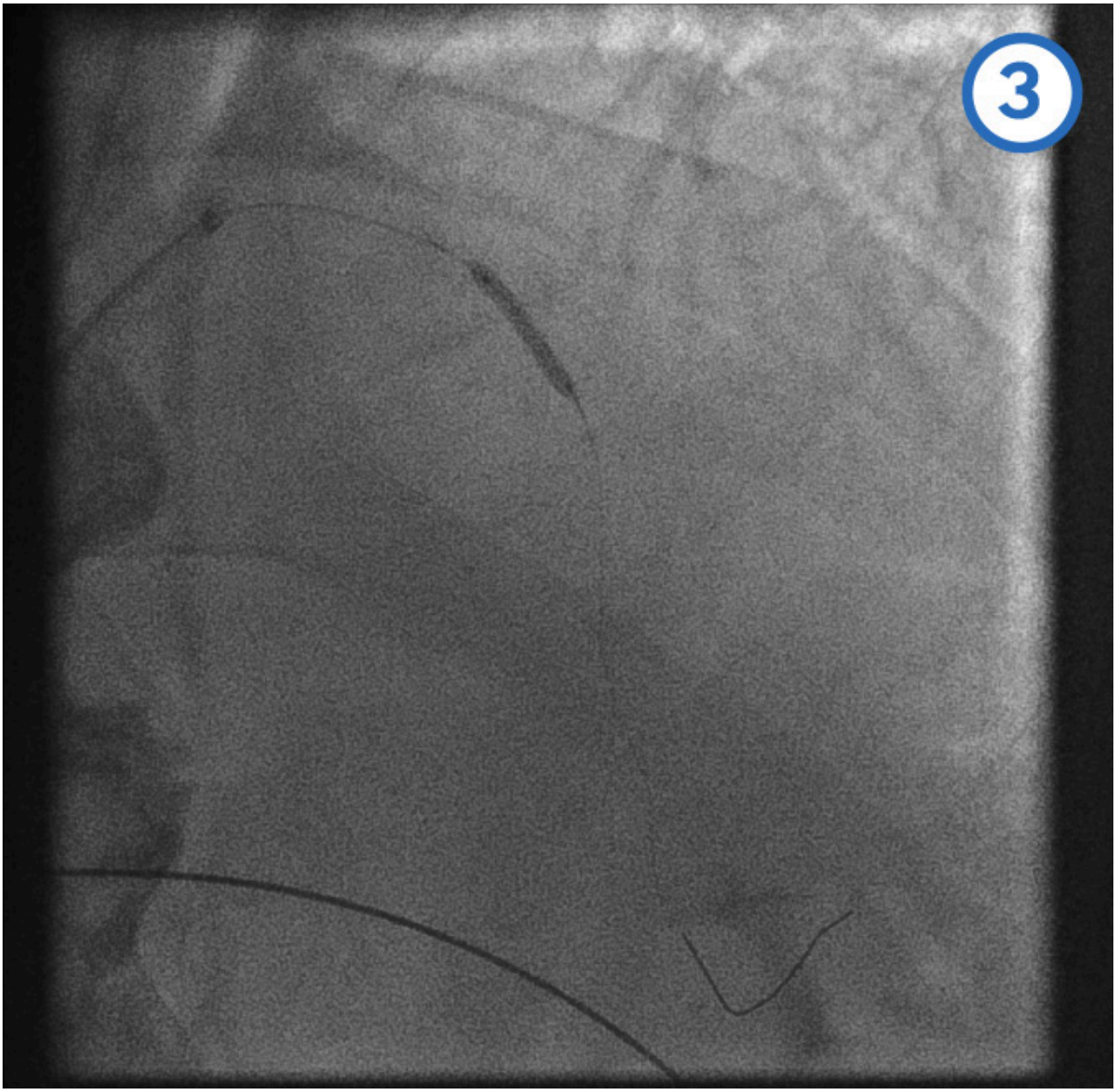

Figure 47: Left anterior descending artery pre-dilatation image 3

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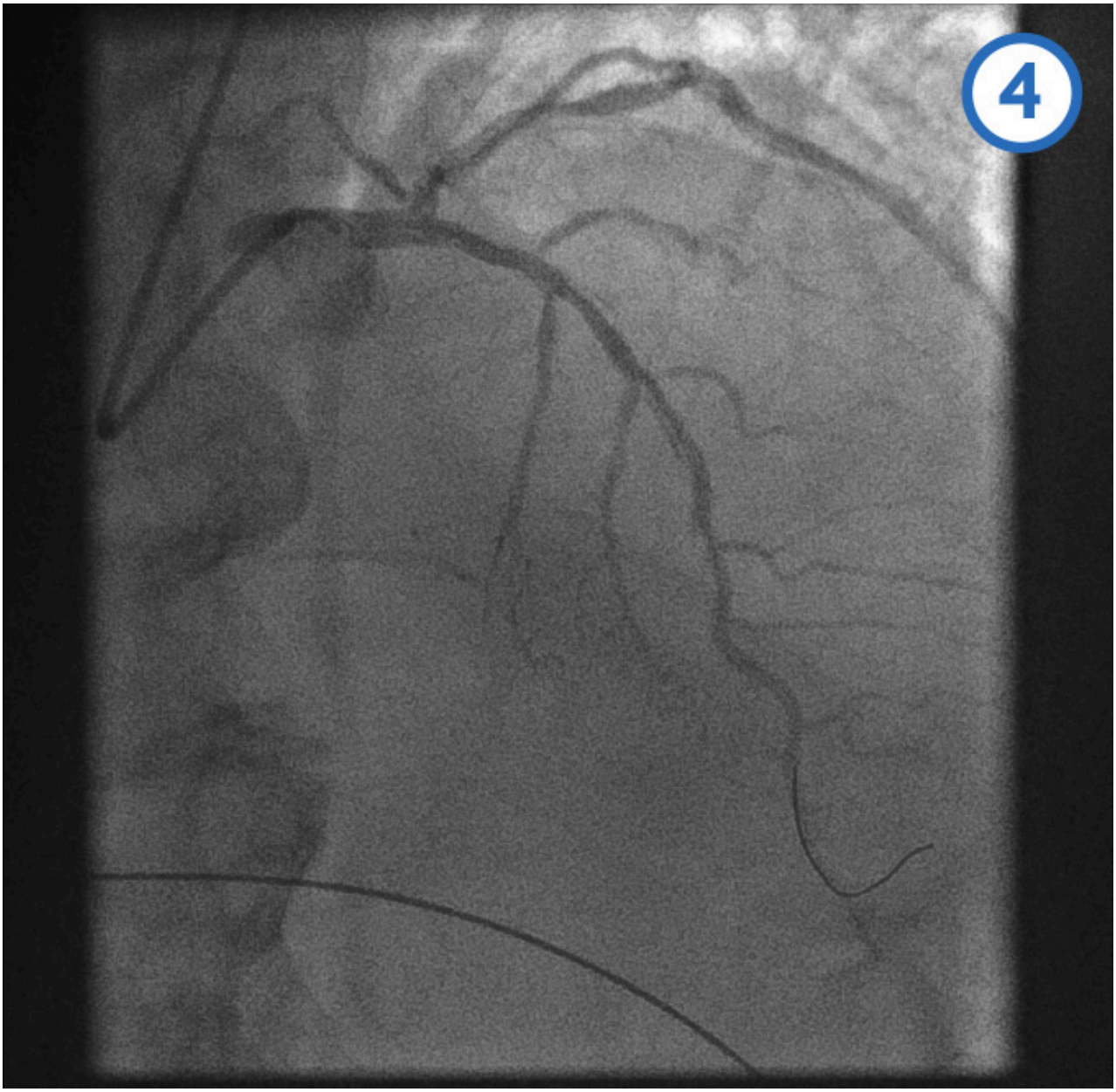

Figure 48: Left anterior descending artery pre-dilatation image 4

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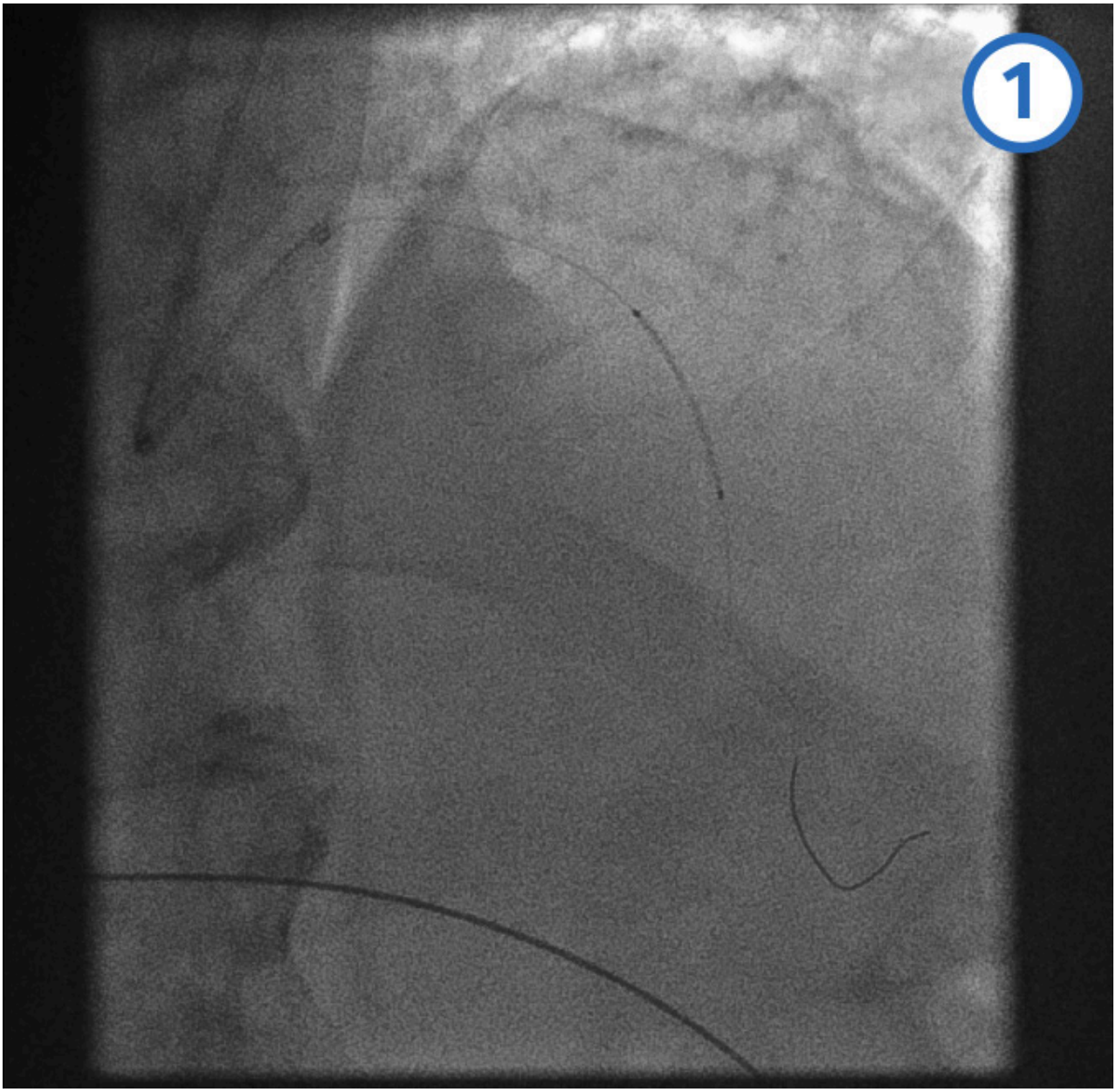

Figure 49: Mid-LAD artery stent deployment#mage 1 - stent positioned in the mid-LAD artery

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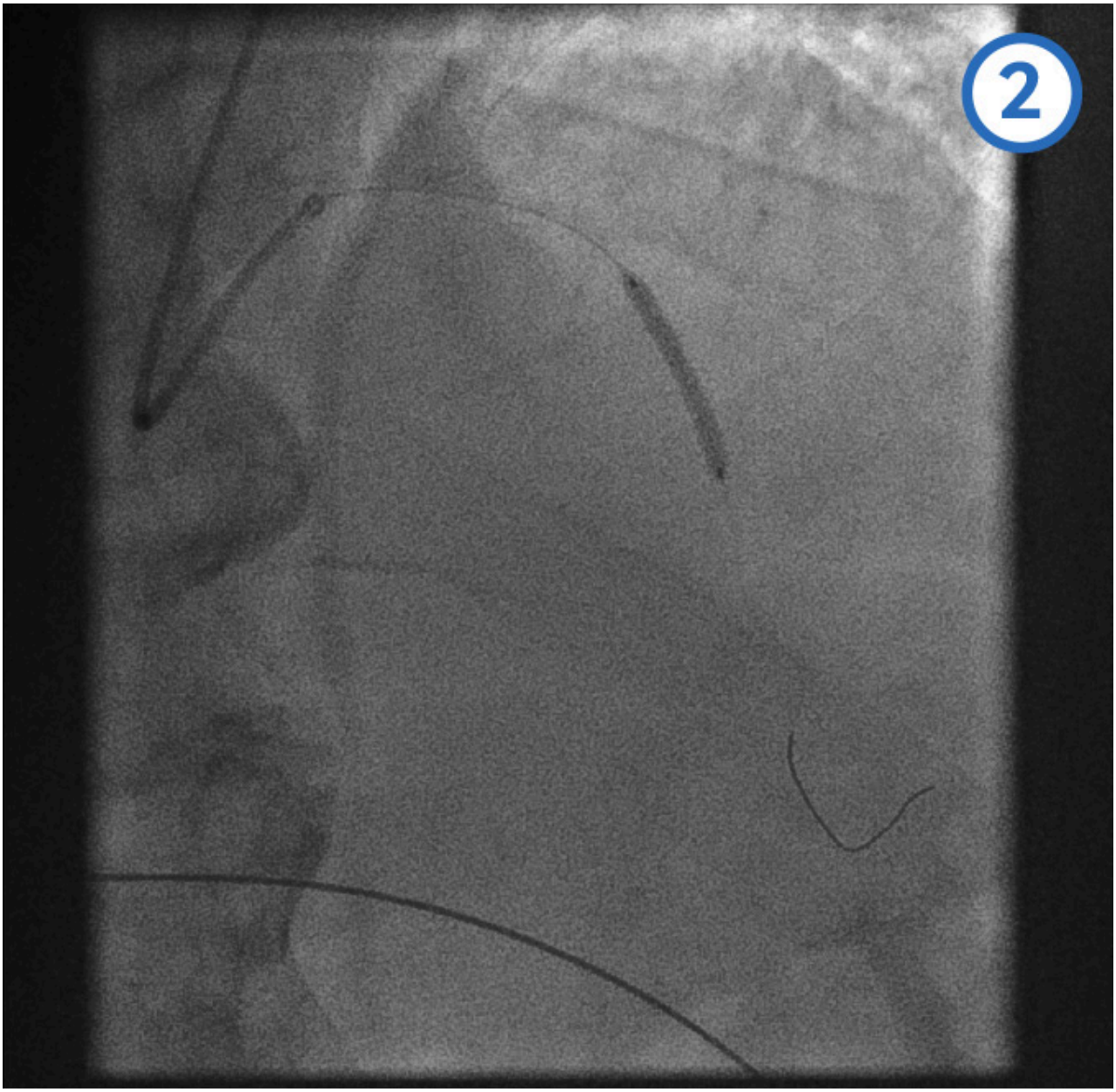

Figure 50: Mid-LAD artery stent deployment image 2 - stent balloon inflated

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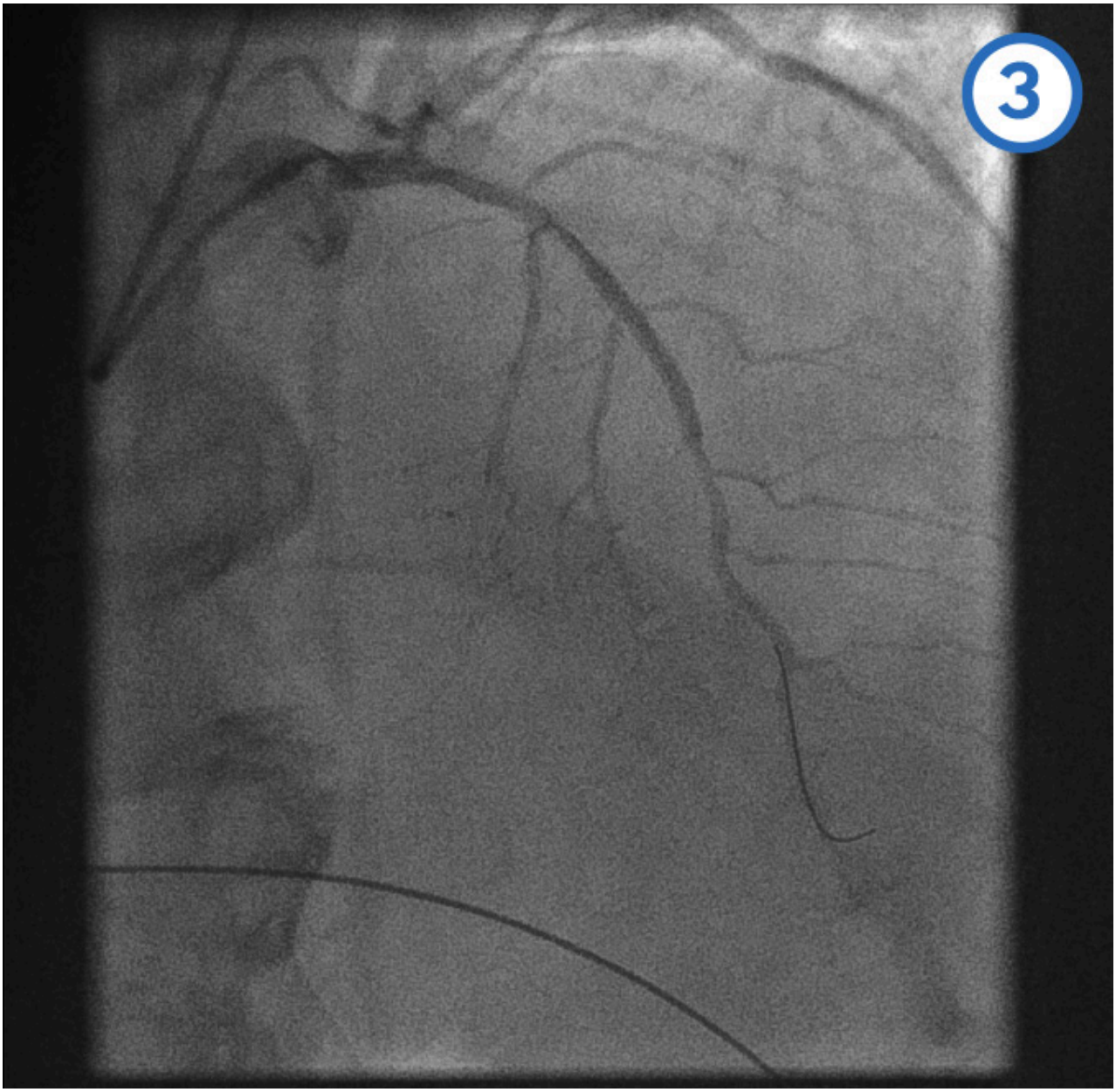

Figure 51: Mid-LAD artery stent deployment image 3 - post mid-LAD stent deployed

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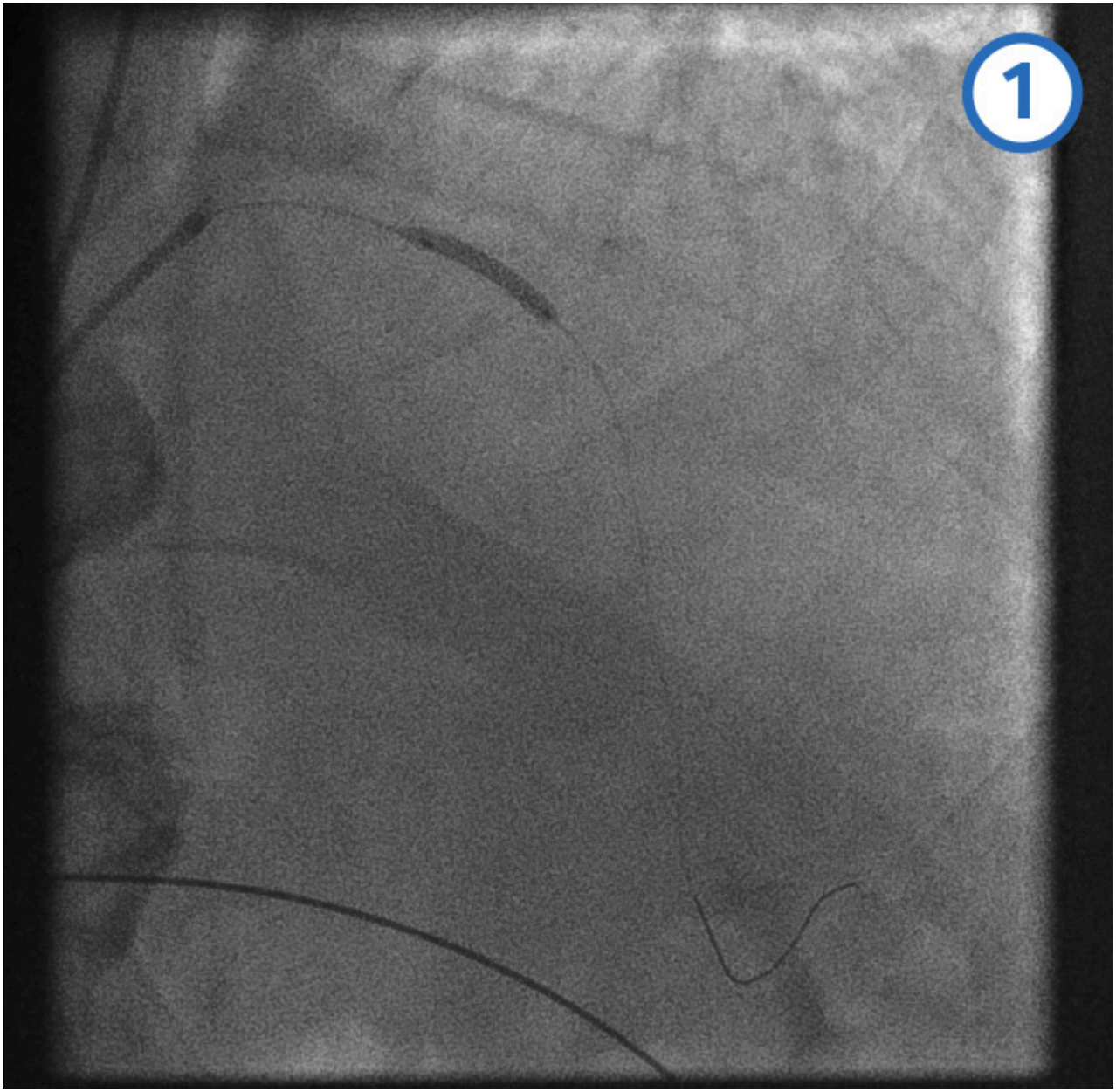

Figure 52: Proximal LAD stent deployment image 1 - following the mid-LAD stent deployment the proximal vessel is pre-dilated

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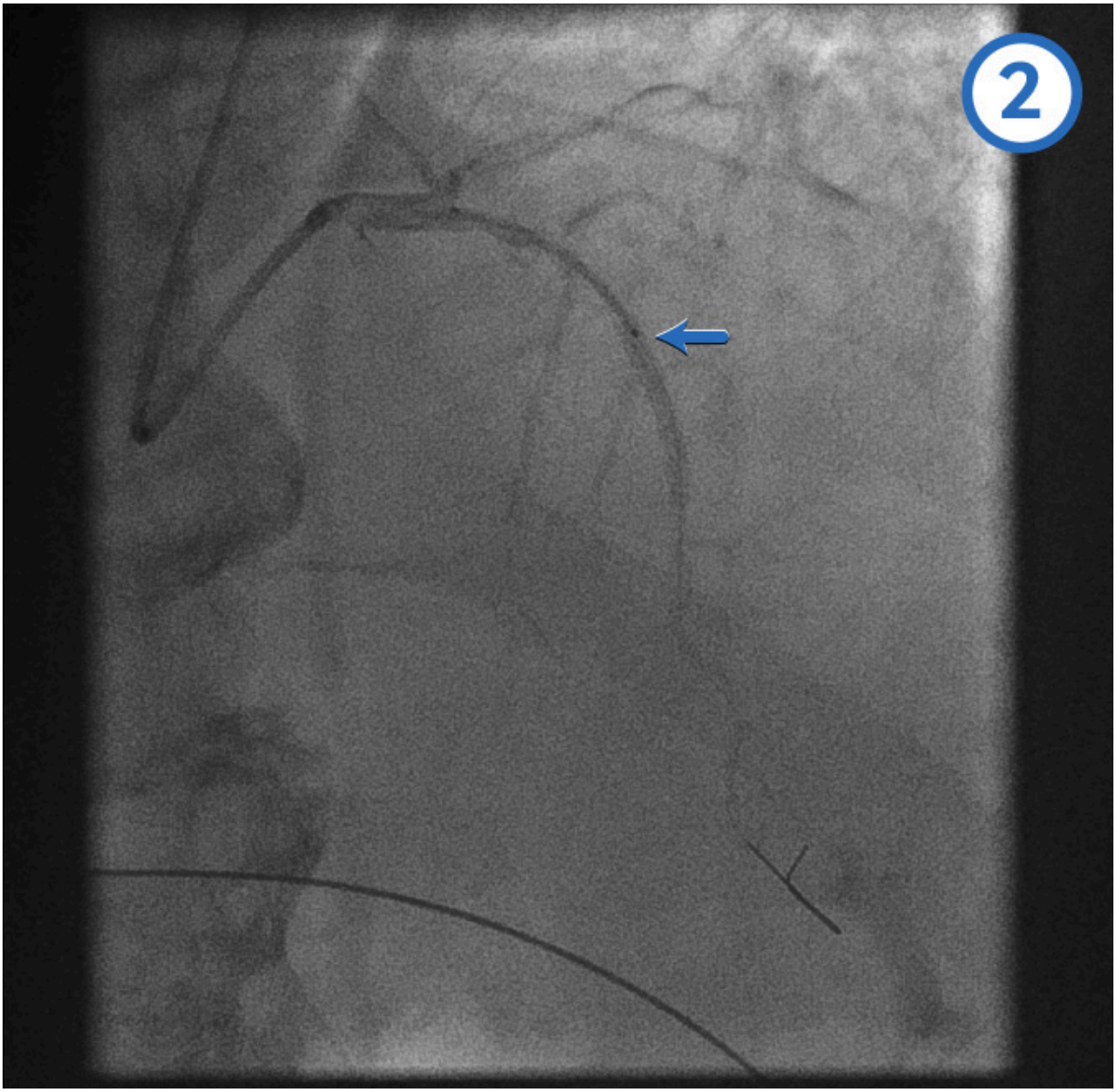

Figure 53: Proximal LAD stent deployment image 2 - proximal stent is positioned with the distal marker (blue arrow) overlapping/within the proximal end of the previously deployed mid-LAD stent

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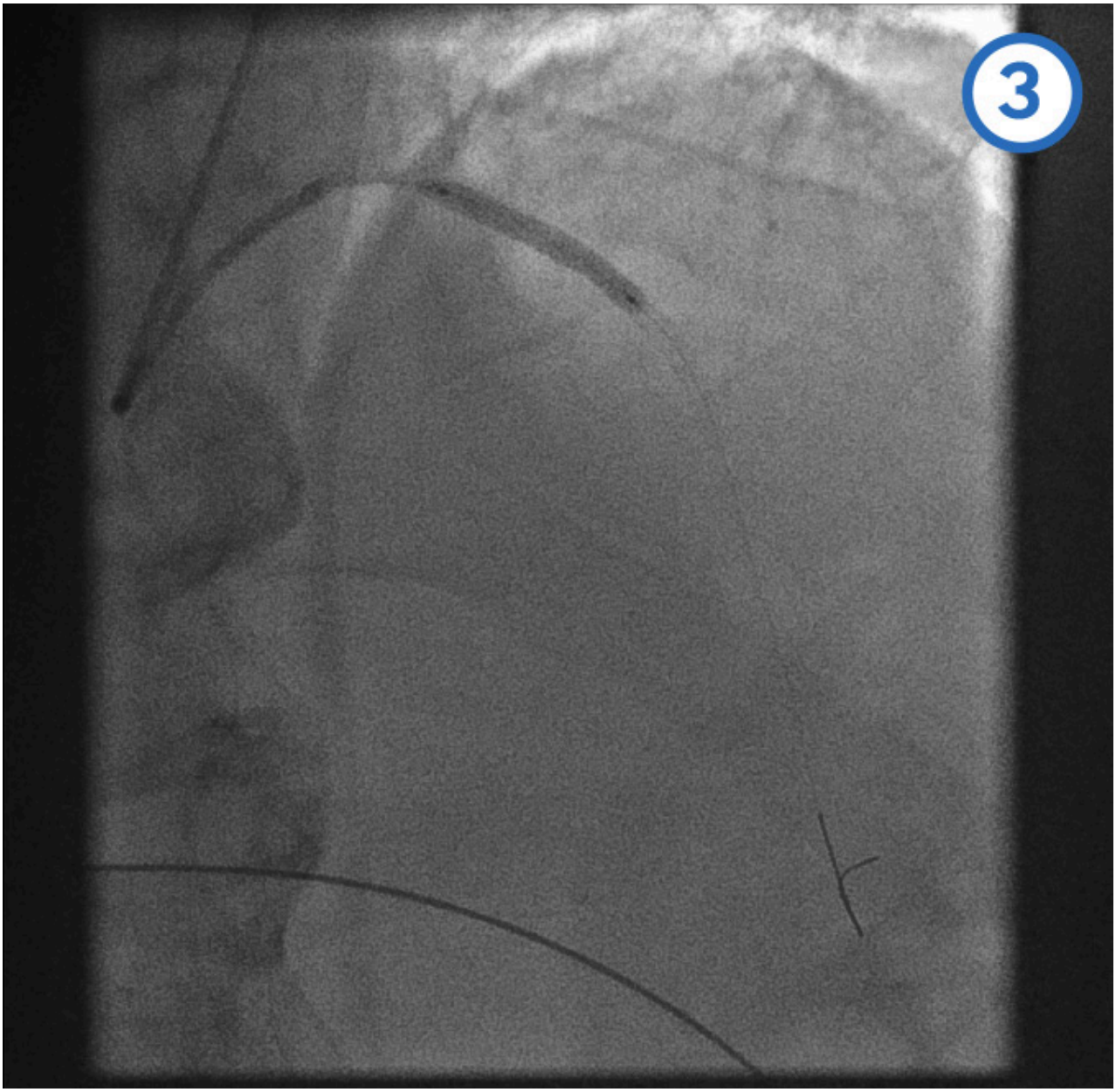

Figure 54: Proximal LAD stent deployment image 3 - proximal stent deployed

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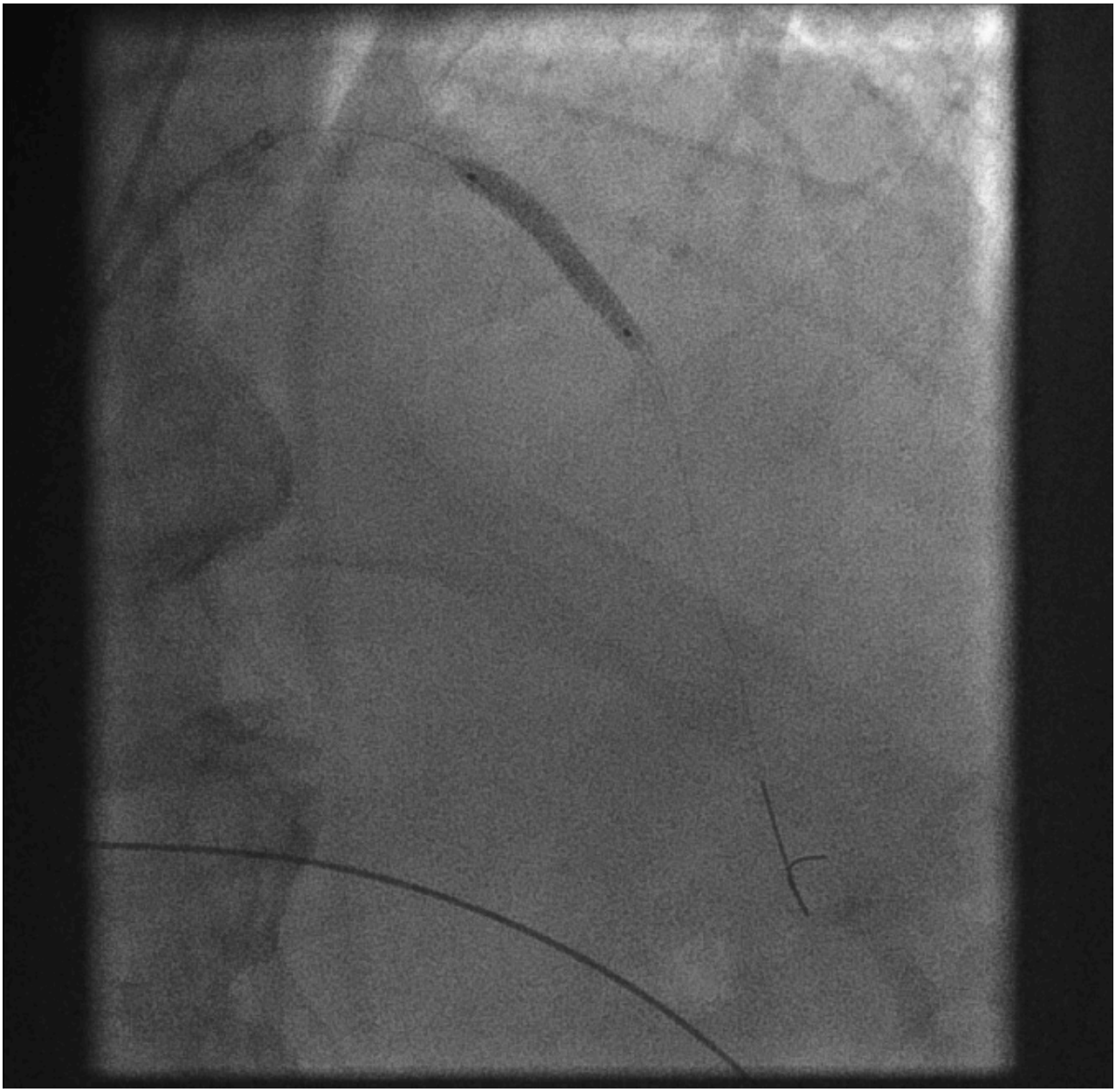

Figure 55: Post-dilatation of overlapping stents

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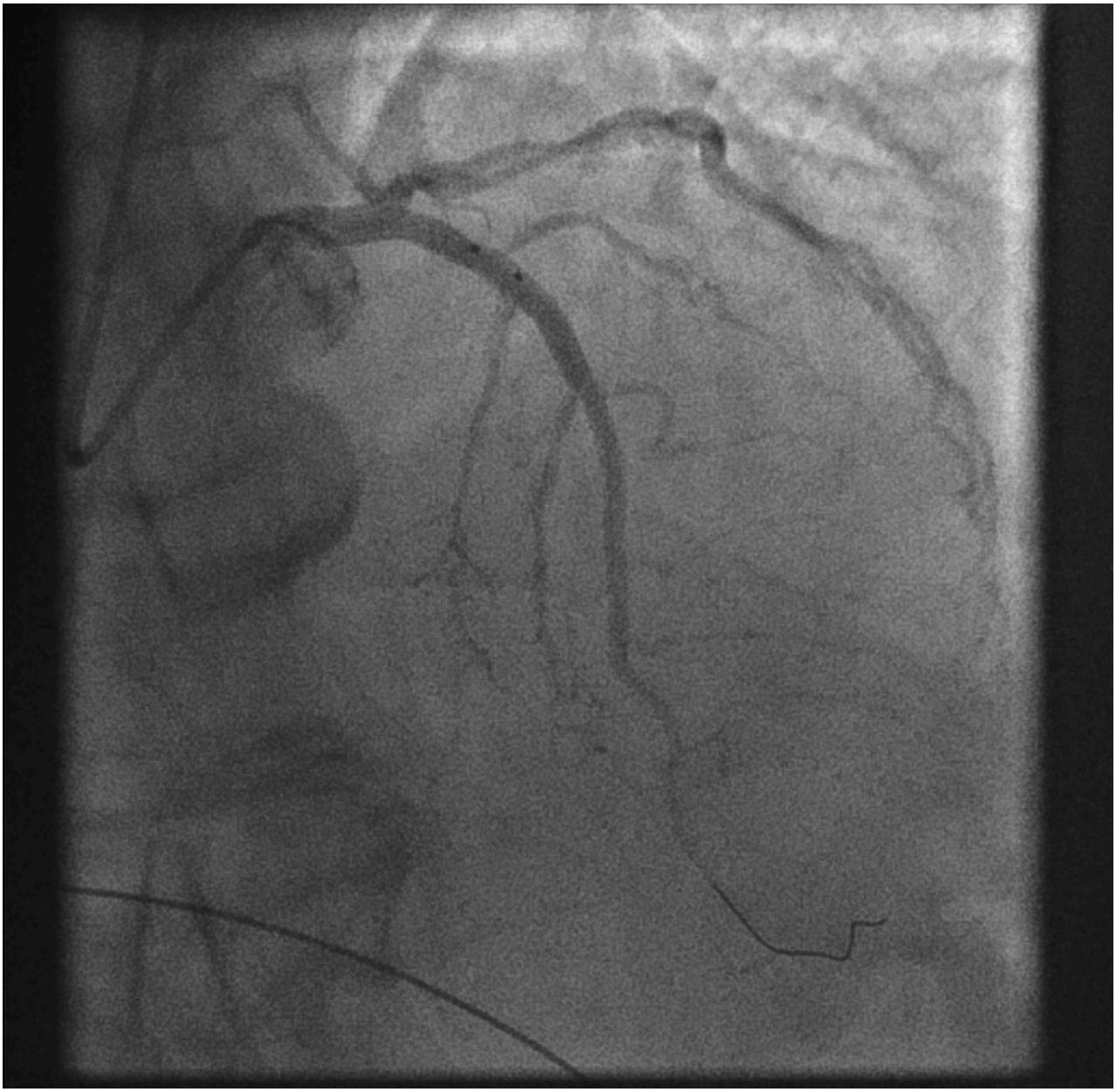

Figure 56: Final LAD stenting result: posterior anterior cranial view

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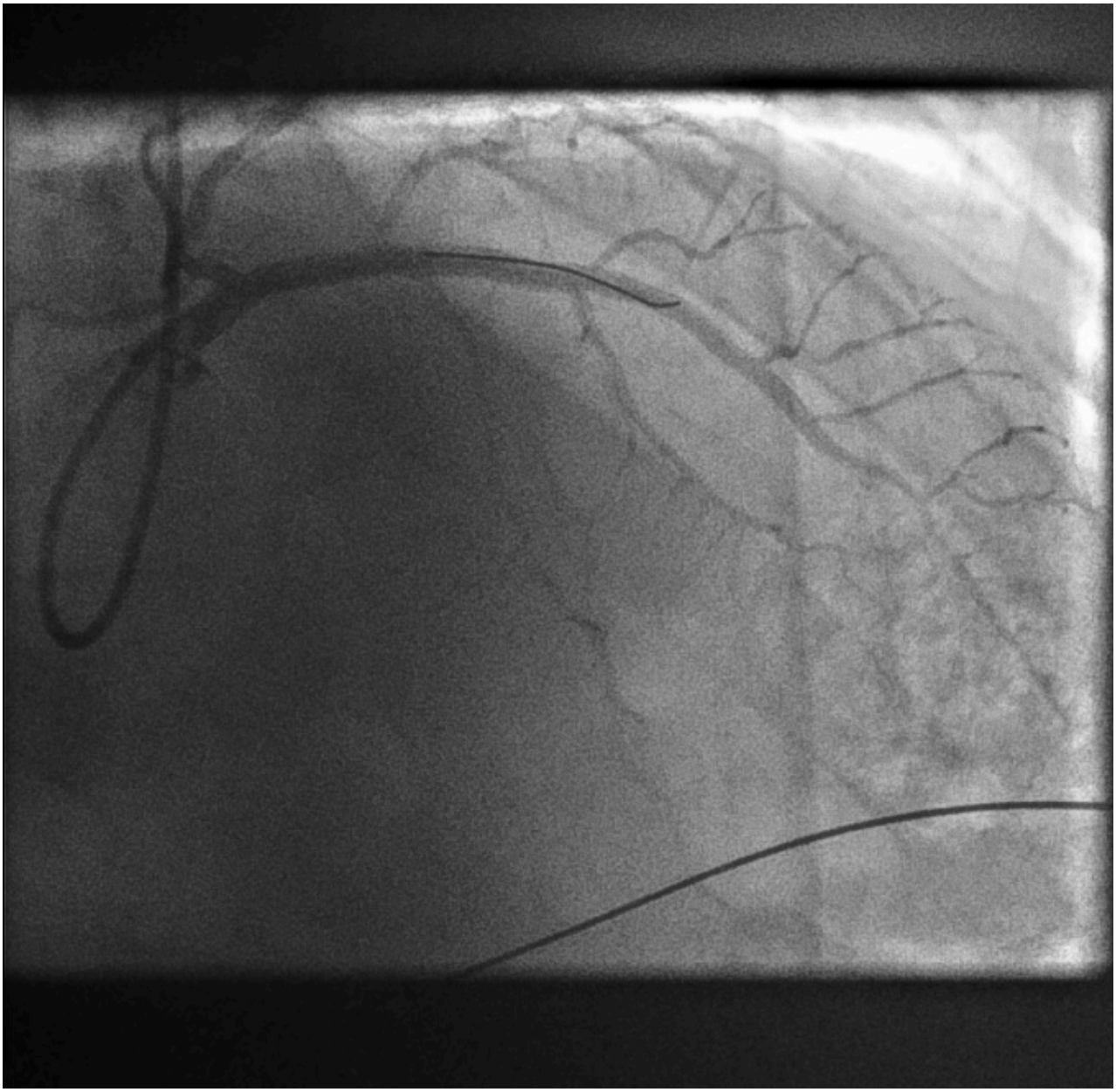

Figure 57: Final LAD stenting result: right anterior oblique cranial view

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4-digit numerals: 1000

numerals < 1: 0.25

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