

Acute coronary syndrome and myocardial infarction

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Essentials

- An acute coronary event results from an abrupt reduction or cessation of blood flow locally within a coronary artery.
 - The cause is often a sudden rupture of an atherosclerotic plaque and the subsequent thrombus formation, which results in an abrupt reduction, or complete cessation, of blood flow within the coronary artery.
 - Also tachycardia and bradycardia, coronary spasm, hypotension, anaemia, respiratory insufficiency or other severe disease may cause a momentary imbalance between oxygen need and availability and lead to acute coronary syndrome (ACS).
- Classification of ACS
 - ACS without ST elevation
 - Non ST elevation myocardial infarction (NSTEMI)
 - Unstable angina (UA)
 - ST elevation myocardial infarction (STEMI)

Symptoms and clinical diagnosis [evd](#)

- The diagnosis of ACS is based on the ischaemic symptoms, clinical findings and ECG changes. The diagnosis is confirmed if the concentration of cardiac biomarkers is increased. For non-ischaemic causes of chest pain, see table [1](#).
- Acute myocardial ischaemia causes chest pain.
 - Starts abruptly, often severe, crushing, heavy or "band-like" in nature and not greatly affected by breathing or changing position
 - Pain suggestive of myocardial infarction is prolonged, persisting for over 20 minutes, and usually has constant intensity.
 - Widespread in the retrosternal area, possibly radiating to the arms (usually to the left arm), back, neck or the jaw
 - In some patients the pain may resemble the symptoms of acute abdomen (pain begins in the upper abdomen accompanied by nausea).
- In particular, elderly patients (> 75 years of age) and patients with diabetes, chronic heart failure, renal failure or dementia may present with a feeling of nausea, weakness or heaviness as well as sweating without chest pain.
- UA is suggested by angina pain that occurs for the first time and is then experienced with increasing frequency or that is a known symptom of previously diagnosed stable angina which is rapidly worsening (over days – in less than 2 weeks).
- Particularly in inferior wall damage, severe vagotonia may induce bradycardia and hypotension which will manifest as dizziness or fainting.
- Other clinical manifestations of myocardial ischaemia include acute pulmonary oedema, loss of consciousness and sudden death.

Table 1. Non-ischaemic causes of chest pain

Disease	Differentiating signs and symptoms
Aortic dissection	1. Sudden intense chest pain 2. Blood pressure may be low and pulses asymmetrical 3. New-onset aortic valve regurgitation 4. Dissection may obstruct the origins of coronary arteries with signs of impending infarction 5. Broad mediastinum on chest x-ray

Disease	Differentiating signs and symptoms
Acute pulmonary embolism	1. Dyspnoea and tachypnoea as the principal symptoms 2. Chest pain in about half of patients 3. Tachycardia, RBBB, low blood pressure in extensive pulmonary embolism; echocardiography shows right-sided dilatation and increased pulmonary pressure 4. Chest x-ray is often normal 5. PaO₂ decreased or normal, PaCO₂ decreased or normal 6. D-dimer assay positive; negative result excludes pulmonary embolism with high probability
1. Spontaneous pneumothorax, 2. tension pneumothorax	1. Dyspnoea, chest pain 2. Quiet breath signs on auscultation 3. Chest x-ray will confirm diagnosis
Oesophageal tear, perforated ulcer	Chest pain, upper abdominal pain
Pericarditis , myocarditis	1. Pain is usually retrosternal and is sharp or tearing in nature 2. The pain is aggravated by inspiration, coughing and changing of position 3. A friction rub may be heard 4. ST-T changes with almost daily alternations
Pleuritis	1. Signs and symptoms of respiratory tract infection 2. Stabbing chest pain, aggravated by inspiration and coughing
Costochondral pain	1. Pain on palpation 2. Chest wall movements and breathing may aggravate the pain
Oesophageal inflammation or spasm, dyspepsia	1. Heartburn, chest pain, upper abdominal pain 2. May be worse in recumbent position and on exertion (reflux) 3. No ECG changes 4. Relief from PPIs
Early herpes zoster	1. No ECG changes 2. Rash appears within a few days 3. Localised paraesthesia before the appearance of the rash
Hyperventilation syndrome	1. Strong feeling of lack of air 2. Fast and deep breathing 3. Cold limbs with tingling and numbness 4. Dizziness, headache, dry mouth 5. PaCO₂ decreased, PaO₂ increased or normal
Depression	1. Continuous feeling of heaviness in the chest, no correlation to exercise 2. ECG normal

Diagnostic investigations [evd](#)

Principles of ECG diagnosis

- A 12 lead ECG is the most important diagnostic procedure, and it should be recorded immediately at the first point of care. The following additional leads should also be recorded: V3R, V4R (right ventricle); V7–V9 (posterior wall).
- Serial ECG recordings (every 15–30 minutes) are indicated if the pain continues, particularly when ECG changes are not noted in the initial recordings or changes are evident in the repeated recordings.
- In the initial phase, it is advisable to leave the chest leads in place or mark their position on the skin to ensure the comparability of the subsequent recordings.
- A Q wave in an ECG will increase the likelihood of coronary heart disease (CHD).
- Differential diagnosis of ECG changes, see table [2](#).

Table 2. Conditions to be considered in the differential diagnosis of an MI when interpreting ECG changes

ECG change	To be considered in the differential diagnosis
ST elevation	Early repolarisation Perimyocarditis Hypertrophic cardiomyopathy Brugada syndrome Pulmonary embolism Hyperkalaemia Left ventricular hypertrophy
ST depression	Sympathicotonia Hyperventilation Microvascular angina Left ventricular hypertrophy Digoxin Post tachyarrhythmia Mitral prolapsed
T wave changes	Normal variant Hyperventilation Increased intracranial pressure An electrolyte disturbance Acute cor pulmonale (pulmonary embolism) Takotsubo syndrome
Q wave	Left ventricular hypertrophy (Lead V1) Hypertrophic cardiomyopathy Right ventricular pressure and volume overload Pneumothorax Duchenne muscular dystrophy Abnormal position of the heart (Leads II, III and aVF) Myocarditis Left anterior fascicular block (right sided chest leads)

Source: Nikus et al. Akuuttien sepelvaltimo-oireyhtymien diagnoosi, luokittelu ja epidemiologia (Diagnosis, classification and epidemiology of acute coronary syndromes (In Finnish). Publication: Heikkilä et al. (ed.) Kardiologia, Kustannus Oy Duodecim, 2008, p. 447.

ECG diagnosis: UA and non ST elevation myocardial infarction (NSTEMI)

- New ST depression of > 0.5 mm in two contiguous leads, even transient, is suggestive of myocardial ischaemia in a patient with chest pain.
- The deeper and more widespread the ST depressions are, the more extensive ischaemia and worse prognosis they suggest.
- T wave inversion (> 1 mm) in two contiguous leads with R/S ratio > 1 .
- Deeply inverted T waves (> 2 mm)
- A normal ECG recording does not exclude acute myocardial ischaemia.

ECG diagnosis: ST elevation myocardial infarction

- A new ST elevation of ≥ 2.5 mm in men below 40 years of age, of ≥ 2 mm in men at least 40 years of age, and of ≥ 1.5 mm in women in at least two anatomically contiguous leads V1–V3, and/or ST elevation of ≥ 1 mm in at least two other anatomically contiguous leads, provided that there are no LBBB or LVH changes or other abnormalities of the ST segment in the ECG
 - Contiguous leads: lateral leads I, aVL, V6; inferior leads II, III, and aVF; anterior leads V4–V6
- New bundle branch block (LBBB) with chest pain
- ST elevation of > 0.5 mm in leads V7–V9 (posterior wall) and reciprocal ST depression of > 0.5 mm in leads V1, V2, V3, R/S ratio > 1 in leads V1–V2

- Comparison with an earlier ECG is important.

Special remarks

- If the patient is known to have LBBB, new ST elevation of > 1 mm parallel to the QRS complex or new ST depression of > 1 mm parallel to the QRS complex in leads V1, V2 or V3 may be in association with a new ischaemic event.
- ST elevation in leads aVR and V1 together with ST depression in leads V4–V6 is suggestive of obstruction of the left main coronary artery branch or of severe CHD.
- If the patient has a ventricular-paced rhythm, comparison with earlier ECG recordings may reveal a new ST elevation; see LBBB .

Cardiac biomarkers evd

- Myocardial ischaemia causes tissue damage and myocardial cell death with the consequent release of measurable cardiac biomarkers into the circulation. Troponins (TnT and TnI) are the primary markers in use; creatine kinase isoenzyme MB mass (CK-MBm) is used in special cases.
- When the high-sensitivity troponin assay is in use, concentrations exceeding the upper reference range threshold (> 14 ng/l) can be measured in almost all patients with myocardial infarction already 3 hours after the onset of symptoms, and 76% of the patients exceed the diagnostic decision threshold > 50 ng/l. If the troponin concentration on arrival at hospital is 14–50 ng/l, a concentration increase of more than 50% in a control sample taken 3–6 hours later is suggestive of myocardial infarction. If the concentration on arrival is < 14 ng/l and the increase in the control sample is > 100% and exceeds the value 14 ng/l, the change may be suggestive of myocardial ischaemia. The reference range applied in the local organization must be taken into account.
- The improved sensitivity of cardiac biomarkers has increased the number of myocardial infarction diagnoses and, respectively, reduced the number of UA diagnoses. It also makes diagnostics more rapid in emergency services. The negative predictive value of sensitive TnT is excellent, 98–100%.
- Increased troponin concentrations may be present in other conditions that are associated with myocardial damage without myocardial infarction. These include myocarditis, heart failure, cardiomyopathy, sequela of a tachyarrhythmic episode, severe anaemia and shock. The concentration may also increase in pulmonary embolism, sepsis, renal failure, cerebral infarction and subarachnoid haemorrhage. A slight increase in the concentration of sensitive TnT is less specific than a large increase.
- A new increase in the troponin concentration when it already was increased (renal failure, fresh myocardial infarction) may also be diagnostic in recurrent myocardial infarction (an increase of more than 20%). In such a case, CK-MBm measurement may also be used.
- If the high-sensitivity troponin assay is not available, the troponin concentration is measured immediately on arrival at the emergency unit and again when 6–8 hours have elapsed from the onset of chest pain. The measurement is then repeated once or twice with an interval of 6–8 hours if the chest pain continues. If the troponin concentration is normal 9–12 hours after the onset of chest pain, ACS is unlikely.
- The plasma concentration of troponin T increases on average about 6 hours after the onset of chest pain (interpatient variation 3–8 hours), and the concentration may remain elevated for 2 or even 3 weeks.

Other investigations

- A chest x-ray should be taken in the hospital to aid the haemodynamic assessment.
- Echocardiography should be performed as soon as possible, in order to evaluate the myocardial function.

Initial management evd

- The patient is placed at bed rest. Aspirin 250–500 mg to chew and swallow or intravenously, unless the patient is allergic to aspirin. The initial dose should be followed by long-term treatment of aspirin 100 mg/day, unless contraindicated ^A.
- Short-acting nitrate spray to relieve chest pain, may be repeated twice with an interval of 5 minutes if necessary, provided that systolic blood pressure is over 100–110 mmHg.
 - Supplemental oxygen if the patient has dyspnoea or symptoms of cardiac failure, or if hypoxia is detected ($\text{SpO}_2 < 90\%$) in continuous oxygen saturation monitoring. Oxygen saturation target is > 90%, in severe COPD 88–90%.

- Continuous monitoring is started (ECG, blood pressure, SpO₂) and an intravenous line is placed.
- For persistent chest pain, an initial dose of morphine 4–8 mg followed by repeated intravenous bolus doses of 2–4 mg.
- If reassurance is not sufficient to calm the patient, intravenous diazepam 2.5–5 mg may be given.
- A nitrate infusion if blood pressure is elevated or there are signs of heart failure. The patient must not be hypotensive (systolic BP should be > 100–110 mmHg) or hypovolaemic nor exhibit signs of right ventricular infarction.
- Ondansetron 4 mg or droperidol (DHBP) 1.25 mg i.v. for nausea
- If tachycardia, arrhythmias or hypertension occur, a beta blocker (metoprolol 2–5 mg i.v.) is commenced during the initial management period (be careful if the patient has hypotension or heart failure).

Treatment of UA and NSTEMI

- The risk of death and further cardiac events in patients with NSTEMI is greatest during the first few days after the acute event, and the risk will remain increased during the first month. In order to identify the optimal treatment approach, the risk of short-term adverse outcomes must therefore be assessed (history, physical examination and ECG) without delay in all patients who are suspected to have cardiac chest pain. The more the high-risk criteria are fulfilled (table 3) the greater the likelihood of a cardiac event.
- The treatment is carried out in a coronary care unit or another ward with facilities to continuously monitor the patient for the haemodynamic state and symptoms of ischaemia.

Table 3. Short-term risk stratification in UA and NSTEMI

Risk category	Risk assessment criteria and urgency of angiography*
High risk	<i>Very high risk: angiography < 2 h</i> ▪ Haemodynamic instability or cardiogenic shock ▪ Repeated or prolonged chest pain despite medication ▪ Significant arrhythmia (cardiac arrest, recurring ventricular tachycardia (VT), ventricular fibrillation (VF)) ▪ Mechanical complication of myocardial infarction ▪ Acute heart failure ▪ Recurring ST depression or transient ST elevation or global ischaemia on ECG <i>High risk: angiography < 24 h</i> ▪ Elevated troponin concentration ▪ ST depression on ECG ▪ GRACE risk score > 140 <i>Medium risk: angiography within 3 days (72 hours)</i> ▪ Diabetes ▪ Renal failure (eGFR < 60) ▪ Decreased function of left ventricle (LVEF < 40%) ▪ Recurring pain or ischaemia soon after myocardial infarction ▪ Earlier coronary balloon angioplasty ▪ Earlier coronary bypass surgery ▪ GRACE risk score 110–140
Low risk	None of the above mentioned signs of danger No repeating chest pain during observation No changes suggesting ischaemia on ECG
1. Source: Eskola et al. Epävakaa angina pectoris ja sydäninfarkti ilman ST-nousua (NSTEMI): vaaran arviointi ja ennuste. [Unstable angina pectoris and non-ST elevation myocardial infarction (NSTEMI): assessment of danger and prognosis] (In Finnish). Publication: Airaksinen et al. (ed.) Kardiologia. Duodecim Medical Publications Ltd., 2016, p. 404. 2. *The timing of angiography indicated in the table is in accordance with the most recent European guideline. It is important to follow the guidelines locally agreed on.	

Antithrombotic therapy evd

- An ADP receptor blocker (clopidogrel, ticagrelor, prasugrel). The drug is chosen according to the local guidelines and to the clinical assessment of the patient.
 - The initial loading dose:
 - ticagrelor 180 mg (continued with 90 mg × 2)
 - clopidogrel 600 mg (continued with 75 mg/day) ^A
 - prasugrel 60 mg (continued with 10 mg × 1)
 - The effect of clopidogrel has a slower onset, and in some patients (about 20 %) its anti-platelet effect remains inadequate.
- Low molecular weight heparin ^A: enoxaparin 1 mg/kg twice daily s.c.; the dose should be reduced in patients over 75 years (–25%) and in renal failure (GFR < 30 ml/min, 50%). Fondaparinux 2.5 mg once daily s.c. is an alternative if emergency invasive care is not considered.
- Thrombolysis is of no benefit.
- If the patient is being treated with warfarin and INR is in the therapeutic range, the treatment is continued without interruption instead of heparin treatment. If INR is below the therapeutic range, anticoagulation can be continued at discretion with warfarin together with a reduced dose of heparin until the target INR level has been reached, or with heparin only. If INR is too high, the action of warfarin can be reversed by administering Vitamin K1 (phytomenadione) 1–3 mg intravenously, even though there is only limited evidence of its benefit. Treatment with warfarin may then be continued. INR is determined daily, if warfarin is continued.
- If the patient uses a direct oral anticoagulant (dabigatran, rivaroxaban, apixaban, edoxaban), local guidelines should be followed regarding suspension or continuation of the drug, as well as regarding other antithrombotic medication. There is variation between guidelines, because no unambiguous directive can be given on the basis of current knowledge.
- *The risk of bleeding must always be assessed.* The following increase the risk: anaemia, thrombocytopenia, age > 75 years, renal failure, liver failure, female gender, weight < 65 kg, systolic BP > 160 mmHg, a previous history of a bleeding tendency and medications affecting the platelet function.

Anti-ischaemic and other treatment evd

- A nitrate infusion if the chest pain persists, blood pressure is elevated or there are signs of heart failure. The patient must not be hypotensive (systolic BP should be > 100–110 mmHg) or hypovolaemic nor exhibit signs of right ventricular infarction.
 - The initial dose is 20 µg/min = 12 ml/hour when the concentration is 100 µg/ml. If necessary, the dose may be increased every few minutes up to 120 ml/h whilst closely monitoring blood pressure.
- A beta blocker (metoprolol) intravenously as 2.5–5 mg bolus doses if blood pressure is elevated or tachycardia or arrhythmias occur (be careful if the patient has hypotension < 120 mmHg or heart failure).
- An oral beta blocker if the patient has heart failure or left ventricular dysfunction; initiation of a beta blocker is considered in all patients after haemodynamic stabilization if there are no contraindications.
- An ACE inhibitor is started within the first 24 hours of treatment in patients with heart failure, systolic left ventricular dysfunction, anterior wall infarction or diabetes. Be careful if the patient has low blood pressure (systolic BP < 100–110 mmHg) or renal failure, but consider the initiation in all patients if there are no contraindications.
- Early introduction of a statin, i.e. during the first day of treatment with a large therapeutic dose is recommended. The dosage can later be decreased but so that the treatment goal is reached.
- NSAIDs should be stopped and should not be used for pain relief.
- Stomach-protective medication is initiated, if the risk of GI bleeding is increased. This risk is increased by earlier GI bleeding, anticoagulant therapy, continuous glucocorticoid or NSAID medication, age > 65 years, dyspepsia, reflux disease and excessive use of alcohol.

Invasive treatment, revascularization (PTCA and CABG) evd

- Coronary angiography is performed if the criteria for invasive treatment are fulfilled, the risk involved with the intervention is not evaluated to be too high and the patient is willing to have the intervention done.
- *Urgent invasive assessment and treatment < 2 h*

- If the patient has chest pains despite treatment and the haemodynamic state is unstable (arrhythmias, hypotension, heart failure; see table 3), an urgent revascularization reduces the incidence of cardiac events, as compared with pharmacotherapy alone, and is thus warranted.
- *Early invasive assessment and treatment 24–72 h*
 - If the ischaemic symptoms can be controlled by pharmacotherapy, an angiography is performed within 24–72 hours from the onset of chest pain.
 - Recurrent ST–T fluctuations, high release of cardiac biomarkers, heart failure, earlier PCI/CABG, diabetes and renal failure all have an influence on the more specific timing of the investigation.
 - In most cases only the occluded infarct-related artery is treated, and other possible stenoses are treated at a later date.
- If the chest pain does not recur, the ECG changes during chest pain are minor and no release of cardiac biomarkers is detected, but the pretest probability of coronary heart disease is nonetheless increased ($\geq 15\%$), an exercise stress test, a myocardial perfusion scintigraphy or a coronary angiography is carried out.
 - Counselling and advice concerning risk factors are provided.

Treatment of STEMI

- STEMI is, more often in men than in women, the first presentation of CHD without preceding angina pain. The patient may therefore not necessarily be able to interpret the symptoms as originating from the heart, which results in an unnecessary delay before the first medical contact is made.
- It is of the utmost importance that primary care providers are able to recognise the symptoms since STEMI is associated with a high risk of life threatening arrhythmias, conduction defects and sudden death. Integrated care pathways incorporating local guidelines implemented in all health care facilities providing on-call services facilitate the easy identification and management of STEMI patients.
- A diagnosis should be made as quickly as possible, and an ECG must be recorded as soon as the first medical contact is made. Serial ECGs should be recorded if no changes are noted but the symptoms are suggestive of an MI.

Initial management

- See above 1.
- If the ST elevation is corrected with the initial medication, the patient is treated on the same principles as a high-risk patient with NSTEMI.

Reperfusion: thrombolytic therapy or emergency percutaneous coronary intervention (PCI)? [evd](#)

- Choosing the treatment approach requires a consultation with a cardiologist on call or with an emergency physician at a hospital, or some other procedure that has been locally agreed upon (care pathway).
 - The most appropriate option for reperfusion treatment and for antithrombotic medication is selected, taking into account patient-specific premises, the patient's consent and the local circumstances.
- Emergency primary PCI (PPCI) is the first-line choice, often combined with the placement of a stent [A](#).
 - The following conditions speak in favour of choosing PPCI: a high-risk MI (signs of heart failure, haemodynamic instability), increased risk of bleeding, a long symptom duration (> 3 hours from onset of symptoms) and uncertainty of the STEMI diagnosis (LBBB, difficulty in interpreting the ECG).
 - Also elderly patients (> 75 years of age) and those under anticoagulant treatment benefit from PPCI; in them, thrombolytic therapy is in particular associated with a greater bleeding risk.
- The results achieved with thrombolytic therapy [A](#) are best if it is administered within 1 to 2 hours of symptom onset. The benefit of the treatment is markedly reduced when more than 6 hours have elapsed from the symptom onset and no benefit is to be expected when more than 12 hours have elapsed from the symptom onset.
 - Thrombolytic therapy is best suited when the patient is below 75 years of age, less than 2–3 hours have passed since the onset of symptoms, the haemodynamic state is stable (infarction area is not very large), ECG finding is reliable and there are no contraindications for thrombolytic therapy (see table 4).
- Agents used in thrombolytic therapy and their doses are presented in table 5.

Table 4. Contraindications to thrombolytic therapy in STEMI

Absolute contraindications	Relative contraindications
Ischaemic stroke or SAH treated in preceding 6 months	TIA in preceding 6 months
Cerebral haemorrhage at any time previously	Anticoagulant therapy
Central nervous system neoplasms or blood vessel anomalies, untreated aneurysm in a cerebral blood vessel	Pregnancy or within 1 week post partum
Major trauma, head injury or major surgery within preceding 3 weeks or neurosurgical operation within preceding 1 month	Systolic BP >180 mmHg or diastolic BP > 110 mmHg, refractory to treatment
Gastrointestinal bleeding within one month	Infective endocarditis
Known bleeding disorder (coagulation disorder, anaemia, thrombocytopenia)	Active peptic ulcer
Confirmed or suspected aortic dissection	Prasugrel or ticagrelor therapy, especially in patients who have just received a loading dose
Recent intervention (liver biopsy, lumbar puncture)	Advanced liver disease (cirrhosis)

Table 5. Agents used in thrombolytic therapy

Drug	Initial dose
Tenecteplase	Single i.v. bolus dose according to weight
	weight < 60 kg 30 mg
	60–69 kg 35 mg
	70–79 kg 40 mg
	80–89 kg 45 mg
	≥ 90 kg 50 mg
Reteplase	10 units × 2 i.v. bolus doses given 30 minutes apart

- Patients who arrive to treatment with delay (12–24 h from the onset of symptoms) and have undergone an STEMI with stable symptoms may benefit from an invasive assessment and treatment. Haemodynamically stable patients with no pain who have had an infarction more than 24 hours ago do not benefit from a routine revascularization of the infarct vessel.
 - Assessment of the severity of the CHD will be based on echocardiography and on evaluation of ischaemia (exercise stress test, myocardial perfusion scintigraphy or coronary angiography).

Other medication in association with reperfusion [evd](#)

- Local guidelines are to be followed.
- Consult first* with the cardiologist on call at the cardiology unit regarding the feasibility of primary PCI and the antithrombotic medication.
- Oral or intravenous ASA 250–500 mg before both thrombolytic therapy and PPCI (see initial management [1](#))
- An initial loading dose of an ADP receptor blocker before PPCI: clopidogrel 600 mg (continued with 75 mg/day), ticagrelor 180 mg (continued with 90 mg × 2), prasugrel 60 mg (continued with 10 mg × 1). The effect of clopidogrel has a slower onset, and in some patients (about 20 %) its anti-platelet effect remains inadequate.
- Before thrombolytic therapy, 300 mg of oral clopidogrel is given to patients under 75 years but not to those over 75 years. If the patient has received a loading dose of prasugrel or ticagrelor, thrombolytic therapy must not be given.
- Enoxaparin before PPCI: starting dose 30 mg intravenously
- Enoxaparin before thrombolytic therapy: 30 mg intravenously for patients under 75 years; not for patients over 75 years. 15 minutes after thrombolytic therapy: for patients under 75 years, a 1 mg/kg dose of enoxaparin s.c. (maximum single dose 100 mg), for patients over 75 years 0.75 mg/kg s.c. (maximum single dose 75 mg).
- If renal impairment is present (eGFR < 30 ml/min [1](#)), the enoxaparin dose should be halved and the anti-FXa concentration monitored.

- Patients with STEMI who decline reperfusion therapy, or in whom it is not possible due to comorbidities, are treated as considered suitable at the given situation.

Complications of thrombolytic therapy

- Intracranial bleed is a rare (1–2%) but serious complication of thrombolytic therapy. The signs of intracranial haemorrhage include a reduced level of consciousness and neurological deficits.
 - If haemorrhage is suspected or the patient exhibits cerebral symptoms, a CT scan of the head must be carried out.
- Intestinal and other non-cerebral bleeds are more frequent (5–10%) than intracranial haemorrhage.
- Bleeding complications associated with thrombolytic therapy usually appear within 24 hours.
- Age > 75 years, female gender, systolic BP > 160 mmHg, lighter body weight and concurrent anticoagulant therapy are significant predictors of haemorrhage.

Assessment of revascularization

- The most useful sign of successful thrombolysis is ST segment resolution > 50% in the lead with the highest elevation within 90 minutes of the start of thrombolytic therapy.
- Disappearance of chest pain and reperfusion arrhythmias are suggestive findings but when appearing alone are not sufficient to confirm revascularization.
- Thrombolysis fails in about one third of patients, and reocclusion develops during hospitalisation in about 20% of patients after initial successful thrombolysis.
- After thrombolysis, a coronary angiography is carried out within 24–48 hours, or earlier if needed on the basis of the clinical situation.

Arrhythmias in the acute phase

Causes

- The mechanisms of arrhythmias include myocardial ischaemia and damage, reperfusion changes, autonomic imbalances as well as electrolyte and acid-base disturbances.
- Rapid reperfusion therapy should be pursued if any of the following is detected: ventricular fibrillation (VF), ventricular tachycardia (VT), abundant ventricular ectopic beats or disturbances in the AV conduction.

Ventricular fibrillation (VF)

- Primary VF occurring within 24–48 hours of the onset of STEMI does not increase long-term mortality.
- VF is treated with immediate defibrillation. Recurrent VF is treated with defibrillation and with a beta blocker and/or amiodarone. Ischaemia starts recurrent VF; therefore, it should be rapidly evaluated and revascularisation should be aimed at.
- The risk of recurrence can be reduced by optimising the drug therapy for myocardial ischaemia and heart failure (adequate beta blockade, ACE inhibitors, nitrates). Similarly, electrolyte disturbances must be corrected as well as factors associated with fluid balance and oxygenation.

Ventricular tachycardia (VT) evd

- Repeated and prolonged episodes of VT are suggestive of a worsening heart disease and are an indication for more detailed ischaemia studies (coronary angiography) and an assessment of the extent of heart failure (echocardiography).
- Monomorphic VT that is either sustained or causes haemodynamic instability is treated with immediate DC cardioversion. If the arrhythmia recurs it should be treated with intravenous amiodarone, and sotalol or lidocaine may also be used.
- If monomorphic non-sustained VT recurs frequently, it is treated with intravenous amiodarone, sotalol (note QT interval) or metoprolol.
- The treatment of polymorphic VT consists of sotalol (note QT interval), other beta blocker or amiodarone, provided that the QRS complex is not widened in an ECG taken during sinus rhythm.
- If the QRS complex is widened, a magnesium infusion may be given, and the electrolytes should be checked. Temporary pacing with overdrive may be indicated as well as urgent coronary angiography.

Other ventricular arrhythmias

- Ventricular ectopic beats are common in patients with MI during the first few days of treatment and no specific treatment is required.

- Idioventricular rhythm is a consequence of reperfusion and requires no specific treatment. Similarly, salvos of non-sustained VT (< 30 seconds) without haemodynamic effects require no specific treatment. None of the above mentioned arrhythmias serve as reliable predictors of VF.

Atrial fibrillation (AF)

- AF is common in the acute phase of an MI (10–20%) and does not require treatment if it is haemodynamically well tolerated. AF often terminates spontaneously.
- It is more prevalent in older patients with MI and in patients with left ventricular dysfunction or failure.
- AF increases the risk of stroke and in-hospital mortality.
- Anticoagulant therapy is indicated unless the patient is already receiving an anticoagulant (heparin).
- An intravenous, later oral beta blocker may be used to provide ventricular rate control in AF. If beta blockers are not suitable or not effective enough, intravenous amiodarone or digoxin may be used.

Sinus bradycardia and AV conduction disturbances

- Sinus bradycardia is common in the first few hours of an MI, particularly in inferior infarction. Opioids may also predispose to sinus bradycardia. If sinus bradycardia causes haemodynamic compromise it should be treated with atropine 0.5 mg intravenously, followed by supplemental doses of 0.2–0.3 mg up to 2.0 mg.
- First-degree AV block is often associated with inferior wall damage and is usually transient requiring no treatment.
- More serious conduction disturbances, such as second- and third-degree AV block as well as LBBB, RBBB and left fascicular block indicate extensive myocardial damage and the placing of a temporary pacing electrode may be warranted. In some cases, permanent pacing is required. In second-degree AV block, atropine may improve the conduction. Isoprenaline may provide temporary help. Transient AV block is most commonly associated with an inferior MI.

Antiarrhythmic medication

- Antiarrhythmic and antibradycardia medications are presented in table [6](#).

Table 6. Intravenous doses of antiarrhythmic and antibradycardia medications

Drug	Intravenous dose
Amiodarone	1. 150 mg over 10 minutes. Supplemental bolus doses of 150 mg may be given over 10–30 minutes for recurrent arrhythmias, but limited to 6–8 supplemental boluses in any 24 hour period. 2. A maintenance infusion of 1 mg/min for 6 hours followed by 0.5 mg/minute may be necessary after the initial dose.
Metoprolol	2.5–5 mg over 2 minutes, up to 3 doses
Digoxin	0.25 mg every other hour, up to 1.0 mg
Lidocaine	0.5–0.75 mg/kg
Verapamil	0.075–0.15 mg/kg over 2 minutes
Atropine	Rapid bolus dose of at least 0.5 mg, repeated up to a total dose of 1.5–2.0 mg (0.04 mg/kg)
Isoprenaline	0.05–0.1 µg/kg/min, up to 2 µg/kg/min. The dose should be adjusted to heart rate and rhythm.

Adapted from the source: Eur Heart J 2008;29:2909-2945

Right ventricular infarction

- Right ventricular infarction is caused by the occlusion of the proximal right coronary artery.

Symptoms and diagnosis

- Clinical symptoms are hypotension and high jugular venous pressure. However, a chest x-ray will fail to demonstrate venous congestion or pulmonary oedema.
- ST elevation ≥ 1 mm in lead V4R is strongly suggestive of right ventricular infarction. Q-waves and/or ST elevation in V1–V3 are also suggestive of right ventricular damage.
- AF may lead to haemodynamic collapse when the synchrony between the atria and ventricles is lost. AF must be promptly treated aiming to restore sinus rhythm. AV conduction disturbances are treated with atropine or dual chamber pacing.

Treatment

- Early reperfusion; see the treatment of STEMI [2](#)
- Hypotension is treated with adequate intravenous fluid loading, initially 500–1,000 ml, in order to maintain adequate preload. Dobutamine may be indicated.
- It is desirable to avoid drugs that lower blood pressure, such as ACE inhibitors, angiotensin receptor blockers, nitrates, diuretics and opioids.

Inpatient treatment

Monitoring and care

- Continuous cardiac monitoring is indicated during the first few treatment days, preferably in a coronary care unit.
- In an uncomplicated MI, the patient is allowed to sit up straightaway, eat unassisted and use the commode at the bedside.
- In a complicated MI (heart failure, arrhythmias) bed rest should continue longer (2–4 days), mobilisation when considered appropriate.
- An ACE inhibitor [A](#) should be introduced during the first treatment day. If an ACE inhibitor is not suitable, an angiotensin receptor blocker should be prescribed.
- A beta blocker is started during the hospitalisation period when the patient is haemodynamically stable.
- Nicotine replacement therapy [A](#) is introduced for smokers.
- Patient education and motivation for treatment is important. The patient should be given information about the disease, the associated drug therapy as well as the modification of risk factors.

Level of care

- If the patient's prognosis is otherwise poor, treatment of an MI may justifiably be carried out in a care facility other than a large hospital. This involves patients in institutional care or those so severely disabled that invasive treatment is not considered appropriate.

Follow-up management [evd](#)

- The follow-up management of ACS consists of antithrombotic and anti-ischaemic drug therapy as well as of modification of risk factors with lifestyle changes and pharmacotherapy.
- *Aspirin* [evd](#) long-term 100 mg daily. Clopidogrel 75 mg daily for patients with aspirin allergy.
- *An ADP receptor blocker* [evd](#) (clopidogrel, ticagrelor, prasugrel) is used in the treatment of ACS; both during the acute hospital phase and in the continued treatment (12 months) after the MI. In special situations, when the risk of ischaemia is high without increased risk of bleeding, the aspirin + ADP receptor blocker medication may be continued for more than 12 months.
- If anticoagulation therapy (warfarin, dabigatran, rivaroxaban or apixaban) is necessary due to some other indication, after insertion of a coronary stent the patient is given *triple therapy* (ASA + ADP receptor blocker [clopidogrel] + anticoagulant) for 1–6 months depending on the risk of bleeding, then ADP receptor blocker (or aspirin) + anticoagulant for a total of one year after the cardiac event. After one year, only anticoagulant is used. Treatment durations are affected by the patient's bleeding risk. The insertion of drug-eluting stents is avoided in patients on permanent anticoagulation therapy.
- *Anticoagulant therapy* is indicated for patients with a left ventricular thrombus, for 3–6 months. In association with AF, the long-term need of anticoagulant therapy is assessed with CHA₂DS₂VASc criteria .
- *An ACE inhibitor* [evd](#) should be prescribed for MI patients with heart failure (in the acute phase), EF < 40%, anterior infarction, hypertension or diabetes. The recommended maintenance doses should be used, or the highest tolerated dose. If an ACE inhibitor is not suitable, an angiotensin receptor blocker should be prescribed.
 - The treatment is recommended to all patients who have had an MI (atherosclerotic disease); research knowledge concerning patients who have undergone revascularization (primary PCI) and who do not have the aforementioned risk factors.
- *A statin* [evd](#) should be prescribed for all patients with MI. Ezetimibe or fibrates may be used when statins are not suitable.

- A *beta blocker* should be prescribed for all patients with MI unless contraindicated. The aim is a heart rate of 50–60/minute at rest. If the function of the left ventricle is not damaged, long-term beta blocker therapy does not reduce the risk of cardiac events after one year of treatment. The length of pharmacotherapy is considered on an individual basis.
- *Calcium-channel blockers* [evd](#) are mainly appropriate for the treatment of hypertension and/or chest pain if beta blockers are not suitable. There is no clear evidence of the benefit of calcium-channel blockers in terms of improved prognosis in CHD. See stable coronary heart disease: .
- A prescription for *short-acting glyceryl trinitrate* (GTN) should always be given to the patient on discharge after an MI.
- *Long-acting nitrates* are reserved for patients with continuing chest pain.
- *Influenza immunization* [C](#) is indicated for all patients who have had an MI.
- After the hospitalisation phase, the long-term management of patients who have sustained an MI will almost without exception be the responsibility of the general practitioner. Primary care health providers are in a key position to motivate the patient to adopt permanent lifestyle changes and to ensure that drug therapies to manage risk factors and improve prognosis are adhered to and that regular primary care follow-up visits are arranged.
- Emphasis should be put on patient guidance [evd](#) (lifestyle changes, diet, exercise) both during the hospital stay and after that in the primary care. Progress of the changes should be actively monitored during follow-up visits. These factors are important in the rehabilitation of the patient back into normal life.
- Rehabilitation measures are needed if the return to normal life is not actualized as planned.

Related resources

- Cochrane reviews [1](#)
- Other evidence summaries [1](#) [evd](#)
- Clinical guidelines [1](#)
- Literature [1](#)

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