

Coronary heart disease

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Essentials

- If coronary heart disease (CHD) is suspected:
 - assess pretest probability and risk factors
 - examine the patient carefully
 - perform a 12-lead ECG and have the required laboratory tests done
 - assess the need for other investigations (see Workup [1](#))
 - consider the need for starting pharmacotherapy (see Pharmacotherapy [2](#)) and the urgency of investigations.
- The pretest probability of CHD can be assessed based on the patient's age and sex and the type of chest pain (see [\[2\]](#)). The probability of developing CHD/myocardial infarction or dying of arterial disease can be estimated using various risk calculators (e.g. SCORE).
- If the pretest probability of CHD is < 15%, other possible causes of chest pain should be individually assessed (see article Acute coronary syndrome and myocardial infarction, table).
 - Provide instruction and guidance concerning established risk factors.
 - If symptoms persist without any detectable cause, consultation of a specialist should be considered.
- If CHD is suspected based on the symptoms and the increased pretest probability of the disease (15–85%), a clinical exercise tolerance test is indicated .
 - The rather low sensitivity of clinical exercise testing adds to the number of false negative findings particularly in patients with a high pretest probability of CHD (> 65%). Specialist consultation for further investigations should be considered.
 - An exercise tolerance test may be necessary for assessing the severity of the disease and sometimes also for assessing various treatment alternatives and the patient's working ability.
- If the pretest probability of CHD is very high (> 85%), coronary angiography should be considered or action taken according to the local chain of care.
- The treatment of CHD consists of symptom management by drug therapy and, if necessary, by invasive interventions, as well as of careful risk factor reduction.
 - Optimal drug therapy combined with lifestyle changes is crucial for the prognosis.
 - If symptoms of CHD persist despite optimal drug therapy, the patient should be referred for assessment by a specialist to assess the need for invasive interventions, if such assessment has not yet been performed and the symptoms have increased during drug therapy.

Causes and clinical manifestation

- For the main risk factors, see table [1](#).
- CHD may present as stable CHD or as acute coronary syndrome .

Table 1. Risk factors of CHD

Classic risk factors	Others
High blood LDL cholesterol concentration	Low blood HDL cholesterol concentration
Smoking	High blood triglyceride concentration
Hypertension	Insulin resistance
Diabetes	Obesity
Age	Physical inactivity
	Genetic factors (family history)
	Psychological factors
	Factors associated with blood clotting and flow
	Nutritional factors

- Obstructive atherosclerotic plaques, endothelial dysfunction, microcirculatory dysfunction and coronary artery spasm reduce blood flow in the coronary arteries thus reducing the oxygen supply to the myocardium, which, in turn, produces the typical symptom of CHD, chest pain (angina pectoris).
- Other symptoms include exertional dyspnoea, fatigue and nausea on exertion, arrhythmias and heart failure.
- For differential diagnosis of chest pain, see article Acute coronary syndrome and myocardial infarction, table).

Symptoms and clinical diagnosis

- Stable angina is a clinical diagnosis which indicates the repeated occurrence of chest pain, induced by an exercise level typical for the patient.
- The pain is relieved by rest and shows no great daily variation, occurring at predictable levels of exercise, i.e. when the rate-pressure product exceeds the patient's personal threshold.
- Typical angina pain
 1. starts gradually during exertion, more quickly if the exertion is sudden, and becomes worse as the exertion continues. Sometimes continuing the exertion does not increase the pain but instead brings about relief (the walk-through angina phenomenon).
 - Emotional stress may also provoke pain as the rate-pressure product increases.
 2. is felt across the chest as widespread constrictive, heavy or tight pain that may make the person slow down or stop
 3. is relieved in a few minutes by rest or glyceryl trinitrate.
- The pain may also radiate to the neck, jaw, arms, epigastric region or between the shoulder blades. The sites of radiation usually stay constant. The pain is often aggravated by cold, eating or smoking.
- If the pain meets all three characteristics, it is highly probable that the patient has typical angina.
- If the pain only meets two of the above characteristics, the patient has atypical angina.
- If the pain only meets one or none of the above characteristics, the patient is likely to have non-cardiac chest pain.
 - The characteristics of non-cardiac chest pain include:
 - the patient's exercise tolerance is usually normal
 - pain occurs at rest and is sharp or stabbing in nature and may last for several hours or even days
 - the location of pain may vary, and the pain sites are sometimes palpable
 - pain is associated with chest wall movements
 - glyceryl trinitrate does not help or takes more than 10 minutes to bring pain relief.
- About one in two patients with CHD presents with typical symptoms.
- Grading of angina pectoris (CCS, Canadian Cardiac Society): see table 2.

Table 2. Grading of angina pectoris (Canadian Cardiac Society)

Grade	Activity provoking chest pain	Level of exercise
CCS grade 1	Strenuous exertion only	120 W or more
CCS grade 2	Walking rapidly or uphill	80–120 W
CCS grade 3	Walking at normal pace on the level	20–80 W
CCS grade 4	Rest, talking or dressing	Less than 20 W

Workup

- Examinations in primary health care:
 - clinical examination (status)
 - 12-lead ECG
 - laboratory tests

- chest X-ray
- clinical exercise test
 - depending on regional procedures (chains of care), additionally echocardiography.
- Investigations performed in hospital include coronary angiography, myocardial perfusion scan, exercise echocardiography or coronary CT angiography.

Clinical findings

- Height and weight
- Auscultation of the heart, lungs and arterial trunks, palpation of peripheral pulses
 - A patient with CHD may have concomitant carotid artery disease and/or arteriosclerosis.
- Blood pressure measurement

12-lead resting ECG

- The ECG is usually normal at rest.
- Reversible ST segment depression that appears during pain and disappears as the pain subsides is highly suggestive of CHD.
- A Q wave, signifying a prior myocardial infarction (MI), is suggestive of CHD. For Q wave criteria, see Acute coronary syndrome and myocardial infarction, table).
- ST-T changes are a sensitive but non-specific finding.
- Fascicular block and bundle branch block (LAHB, RBBB and LBBB) are more common in patients with multivessel CHD, but the finding is not specific.

Laboratory tests

- Basic blood count with platelet count, sodium, potassium, creatinine
- Cholesterol, HDL cholesterol, LDL cholesterol, triglycerides
- Fasting blood glucose and HbA_{1c}
 - A 2-hour oral glucose tolerance test should be carried out if any of these are abnormal.
- Liver function tests no later than when statins are started
- ProBNP if heart failure is suspected

Exercise test [evd](#)

- An exercise test is indicated for making the diagnosis and for defining the severity of the disease.
- For details, see .

Imaging studies

- Chest x-ray
 - Heart size, pulmonary congestion, signs of heart failure, valvular calcification and other causes of chest pain
- Echocardiography
 - Done to examine systolic (ejection fraction) and diastolic left ventricular function and the presence of any disturbance of septal motion.
 - The findings are often normal but the examination gives even more information on the function of the heart, including the valves (murmurs).

Indications for additional investigations

- In addition to pharmacotherapy, invasive investigations (coronary angiography) should be considered if
 - the patient's exercise tolerance and quality of life are clearly reduced due to chest pain despite optimal drug therapy
 - signs of myocardial ischaemia are evident during an exercise test at a low exercise workload (< 100 W) or a low heart rate (< 120 bpm) or
 - signs of left ventricular dysfunction are present.
- Angiography, see
- Myocardial perfusion scan
 - The sensitivity is higher than that of an exercise test but the specificity is roughly the same.
 - Can be performed using either physical or pharmacologic stress.

- May be carried out if
 - an exercise test is not diagnostic but CHD is very likely
 - the patient is physically handicapped
 - an ECG abnormality (LBBB, LVH, WPW, electronically paced ventricular rhythm) interferes with the interpretation of the exercise test.
- Exercise echocardiography
 - Ischaemia induces myocardial wall motion abnormalities, which can be detected by echocardiography during exercise compared with the result of resting echocardiography.
 - The test is more sensitive and more accurate than an exercise test but requires an experienced operator.
 - May be considered when ECG abnormalities interfere with the interpretation of an exercise test.
- Coronary CT angiography
 - Noninvasive investigation giving an idea about the state of the coronary arteries.
 - Coronary CT angiography should be reserved for patients whose likelihood of having CHD is small, and in whom the indications for the investigation are therefore more restricted than those for conventional angiography.

Special features in the diagnosis of CHD in women

- In women, CHD is diagnosed on average 5–10 years later than in men.
 - The incidence of CHD increases noticeably after menopause.
 - By the age of 70–79 years, the gender differences in both the incidence and the mortality due to CHD have levelled out.
- The risk factors for CHD are the same for both sexes.
 - Women who develop CHD before menopause have more risk factors than those who develop the disease at a later age.
 - If CHD develops in a middle-aged woman she is likely to have several risk factors: smoking, dyslipidaemia, type 2 diabetes, hypertension.
- It is more difficult to diagnose CHD in women than in men.
 - In women, the chest pain is more often atypical and the disease may only manifest itself as dyspnoea, exhaustion, malaise or nausea on exertion.
 - It is still particularly important to investigate the symptom carefully even if this takes more time.
 - After menopause, the diagnostic accuracy of symptoms improves. In women aged over 65–70 years, the diagnostic accuracy of chest pain is similar to that in men.
 - The predictive value of exercise testing is lower in women. This is due to the greater number of false-positive test results in premenopausal women.
- In women with CHD, coronary arteries are often less (< 50 %) constricted than in men and left ventricular function is better preserved.
- Women with CHD have more symptoms, and there are probably also other mechanisms (microcirculatory disturbances) behind their chest pain.
- Women with non-constrictive CHD and chest pain would appear to be at higher risk of a cardiac event than those without symptoms.

Management of CHD risk factors evd

- Minimizing all risk factors has a marked effect on prognosis as it slows down the progress of atherosclerosis and prevents the development of an MI.
- Stopping smoking is very important. The risk of an MI is 3-fold in smokers and even higher in women.
- In addition, excessive alcohol consumption should be reduced.
- Hypertension should be treated.
 - The target level of below 140/85 mmHg should always be achieved but a lower therapeutic level (130–139/80–85 mmHg) should be the aim in patients with diabetes. In patients with renal disease the aim should be below 130/80 mmHg.
 - See article Investigation and starting the treatment of hypertension > Therapeutic goal .
- The management of hyperlipidaemias

- Statins not only reduce blood lipid concentrations but also have an effect on coronary endothelial function, the inflammatory reactions and blood clotting.
- Recommendations
 - LDL cholesterol < 1.8 mmol/l or decreased by > 50% from the baseline value. According to current scientific knowledge, drug therapy can be considered justified.
 - Other targets should also be considered: cholesterol < 4.0–4.5 mmol/l, cholesterol/HDL < 4.0, HDL > 1.0 mmol/l, triglycerides < 2 mmol/l.
 - For pharmacotherapy of hyperlipidaemias and special reimbursement of antihyperlipidaemic drugs, see article Treatment of dyslipidaemias
- Treating obesity [evd](#)
 - If the patient's BMI is higher than 28 and the waist circumference ≥ 100 cm in men, or ≥ 90 cm in women, weight reduction is indicated.
 - The goal is to achieve a BMI ≥ 25 and a waist circumference ≥ 94 cm in men and ≥ 80 cm in women.
 - Abdominal obesity (high waist-to-hip ratio) signifies a particularly high risk **B**.
- Patients who have had an MI are often found to have a slightly increased fasting blood glucose concentration, impaired glucose tolerance or diabetes.
 - If the 2-hour oral glucose tolerance test is normal, fasting glucose levels should be checked at annual follow-up visits.
 - Should the glucose tolerance test results be abnormal, the patient will need exercise and dietary advice and possibly additionally medication and more frequent follow-up visits.
 - If diabetes is diagnosed, measures should be taken according to the regional chain of care.
- Physical exercise [evd](#)
 - Regular exercise improves not only the sense of well being but also the prognosis, by reducing risk factors. Physical activity also plays a part in primary prevention.
 - Sudden and strenuous physical activity should be avoided.
- According to current scientific knowledge, oestrogen replacement therapy is not recommended for primary or secondary prevention. Existing hormone therapy can be continued based on individual assessment.

Pharmacotherapy: aims and modes of action [evd](#)

- If suspicion of CHD arises, aspirin, a statin and a glyceryl trinitrate product for the symptoms can be started at the very first visit and, additionally, a beta-blocker if sufficient laboratory tests have been performed.

Medication improving the prognosis

- Effective secondary prevention includes aspirin, a beta-blocker, a statin and an ACE inhibitor.
- Aspirin decreases platelet aggregation and thus reduces the risk of acute coronary events. Aspirin is recommended for all patients with CHD permanently from the very first symptoms at a dose of 100–250 mg/day, unless it is contraindicated. Patients allergic to aspirin may use clopidogrel.
- ADP (adenosine diphosphate) receptor blockers (clopidogrel, ticagrelor, prasugrel) also reduce platelet aggregation. As they inhibit platelet function through different surface receptors than aspirin their use in combination with aspirin has an additive effect.
 - ADP receptor blockers are used in the treatment of acute coronary syndrome both in hospital and in the continued treatment (12 months) after the acute event.
 - After the insertion of a coronary stent, ADP receptor blockers significantly decrease the incidence of stent thrombosis and should be included in the medication. The treatment period is shorter when stenting is carried out for occlusion only causing stable angina.
- An ACE inhibitor should be included in the medication of patients who have had an MI or have left ventricular systolic failure or diabetes. An ACE inhibitor is also recommended for patients with CHD or other atherosclerotic vascular disease.
 - ACE inhibitors reduce the symptoms and cardiac events that are associated with ischaemic heart failure.
 - It has been suggested that ACE inhibitors may in some way slow down the progression of atherosclerosis in the arterial walls.

- Either the recommended maintenance doses (ramipril 10 mg/day, enalapril 20[–40] mg/day, lisinopril 20[–40] mg/day) or the highest tolerated doses (adverse effects, hypotension) should be used.
- Statins reduce cholesterol synthesis in the liver, blood LDL cholesterol concentrations and the accumulation of LDL cholesterol on the vascular walls. Moreover, statins improve the functioning of the inner lining of the arteries, suppress blood clotting and interfere with inflammatory and immune responses. In patients with CHD, statins reduce the rate of both reinfarction and death.

Symptom-relieving medication

- Beta-blockers lower heart rate and blood pressure and thus decrease myocardial oxygen consumption during exercise and often relieve chest pain.
 - After MI, beta blockers significantly decrease the risk of reinfarction and death.
 - As beta-blockers alleviate chest pain, their use is often justified for symptom relief, alone.
 - Patients with asymptomatic stable CHD and normal left ventricular function may not benefit from long-term use of beta-blockers. Current scientific evidence is based on a retrospective patient register data, not randomized clinical studies.
 - Adequate beta blockade has been achieved when the heart rate has decreased to 50–60 bpm at rest.
 - Beta blockers are also the first-line antiarrhythmics in patients with CHD.
- Calcium-channel blockers
 - Verapamil and diltiazem can be used to alleviate chest pain, particularly when beta blockers are not suitable. These drugs are contraindicated in patients with left ventricular systolic failure or a conduction defect.
 - Of dihydropyridine derivatives, long-acting nifedipine combined with a beta-blocker, or amlodipine, lercanidipine or felodipine, either alone or combined with a beta-blocker, are suitable for the treatment of both chest pain and hypertension.
- A long-acting nitrate may be included in the medication regime if chest pain is frequent.
 - The nitrate should be administered at the time of day when symptoms occur most frequently, which is often during the daytime. The usual dose is 20–40(–60) mg/day.
 - A nitrate patch can be used to treat nocturnal angina. The patch should be removed in the morning to avoid the development of nitrate tolerance. For the same reason a pause should be incorporated in the administration regime of long-acting nitrates, for example in the evening or at night.
 - Nitrates are only symptomatic therapy and are not needed if the patient is asymptomatic. They improve exercise tolerance but there is no convincing evidence concerning their effect on the prognosis.
- Short-acting glyceryl trinitrate (GTN), either as sublingual tablets or as aerosol spray, is classically used for acute episodes. Remember prophylactic use, as well.

Revascularization (PTCA and CABG) evd

- Coronary artery revascularization may be performed either by balloon angioplasty (percutaneous transluminal coronary angioplasty, PTCA) followed by the placing of a metal stent into the coronary artery (percutaneous coronary intervention, PCI), or by coronary artery bypass graft surgery (CABG).
 - Coronary artery patency is sustained better with stenting than with PTCA alone B. Stenting has reduced the need for coronary surgery.
 - Drug-eluting stents are impregnated with a cell growth inhibitor that is slowly released into the surrounding tissues over several months. Drug-eluting stents have clearly reduced the frequency of restenosis and cardiovascular events compared with metal stents or PTCA alone, but drug-eluting stents may be associated with late thrombosis.
 - A drug-eluting balloon releases a cell growth inhibitor during balloon angioplasty of an occluded coronary artery.
- The decision regarding the need for revascularization and the preferred method (PCI/CABG) is based not only on the severity, location and number of coronary occlusions but also on the assessment of left ventricular function, potential valvular diseases, severity of symptoms and comorbidity (diabetes, renal disease) and risks related to surgery.
- The advantages of PCI include the short duration of treatment, shorter recovery period and faster return back to work. The more frequent need for later revascularization compared with CABG may be

considered a disadvantage.

- In outpatient care, after revascularization it is most important to monitor the patient's state and risk factors and to use pharmacological treatment improving the prognosis, according to the applicable guidelines. Symptom-relieving drug therapy can often be at least reduced.

Related resources

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

References

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