Ischemic heart disease (Coronary artery disease)

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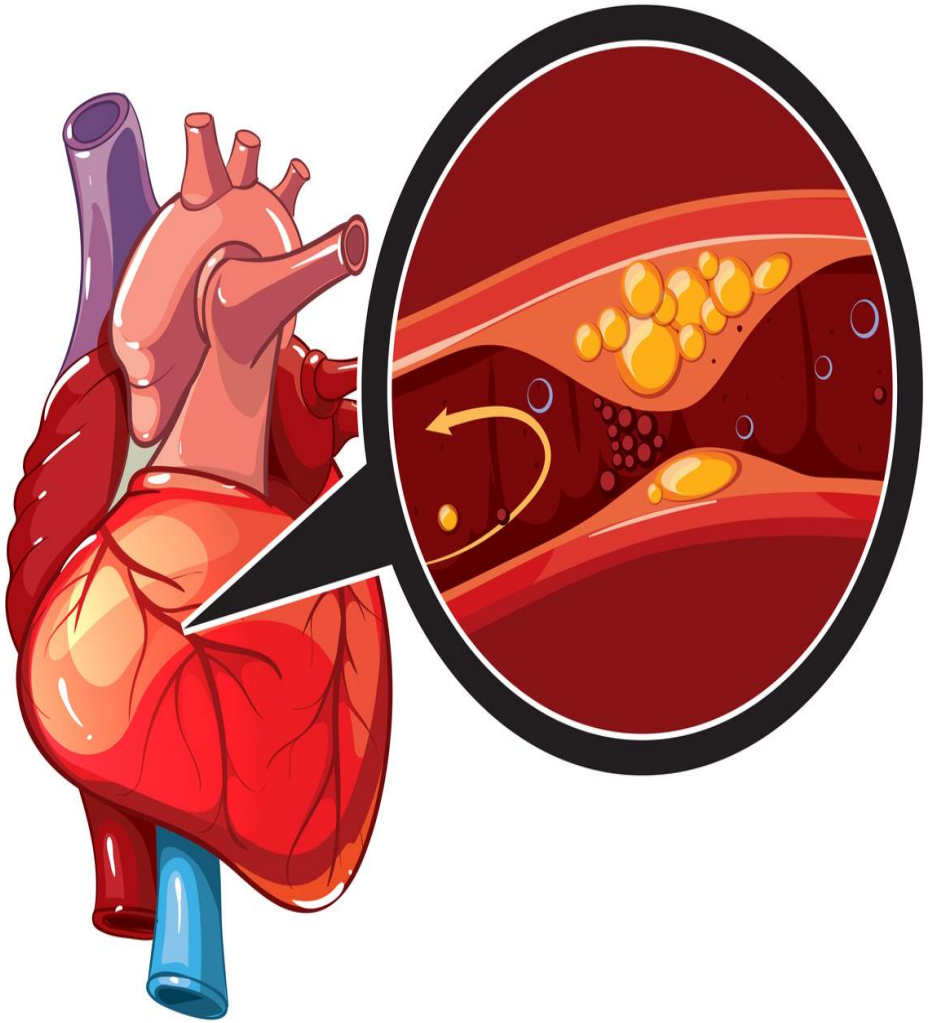
19. 02. 2026



Lecture objectives

Define IHD and distinguish its clinical forms.

Explain key pathophysiological mechanisms (oxygen supply vs. demand).

Summarize typical manifestations of angina pectoris and principles of differential diagnosis of chest pain.

Differentiate acute coronary syndromes (UA/NSTEMI/STEMI) by clinical presentation, ECG, and biomarkers.

List the major complications of myocardial infarction and principles of secondary prevention.

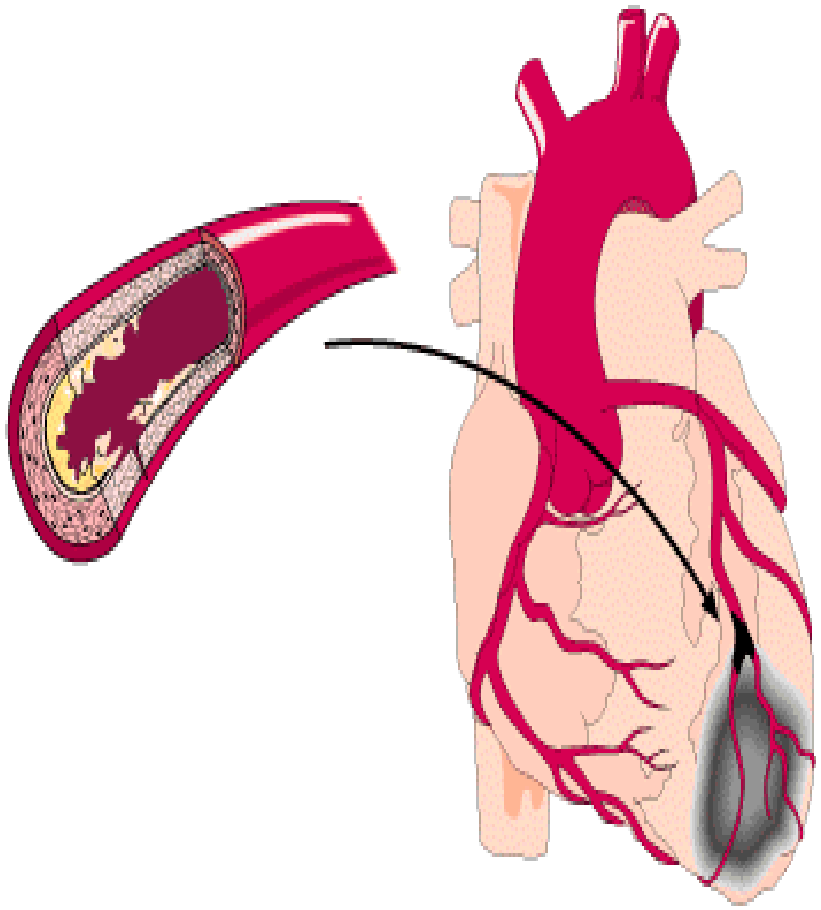
Definition and basic classification

IHD is a clinical syndrome caused by transient or permanent myocardial ischemia.

The key mechanism is an imbalance between myocardial oxygen demand and its supply via coronary blood flow.

The most common cause is atherosclerosis of the coronary arteries (stenosis, plaque rupture/erosion, thrombosis).

Less commonly: vasospasm, microvascular dysfunction, embolization, anemia/hypoxemia, marked tachycardia or hypotension.



Clinical forms of CAD

- Acute forms of CAD (acute coronary syndrome - ACS)
  - Unstable angina pectoris (UA)
  - Acute myocardial infarction without ST elevation (NSTEMI)
  - Acute myocardial infarction with ST elevation (STEMI)
  - Sudden cardiac death (malignant arrhythmias on the background of ischemia)

- Chronic forms of CAD
  - Stable angina pectoris
  - Silent/silent (asymptomatic) myocardial ischemia
  - Prinzmetal's (variant) angina pectoris
  - Chronic heart failure
  - Arrhythmias: Rhythm disorders caused by ischemia.
  - Syndrome X: Painful systole in normal large coronary arteries (involvement of microvessels).

- Clinical forms can overlap and the chronic form can change to acute at any time.

Pathophysiology – imbalance of supply vs. demand for O_2

Oxygen supply

Coronary flow: stenosis, thrombus, vasospasm.

Perfusion pressure and diastolic time (tachycardia shortens diastole).

Blood O_2 content: anemia, hypoxemia.

Potreba kyslíka

Heart rate (↑ HR = ↑ consumption, ↓ diastolic perfusion).

Myocardial contractility.

Wall tension: preload/afterload (BP), hypertrophy.

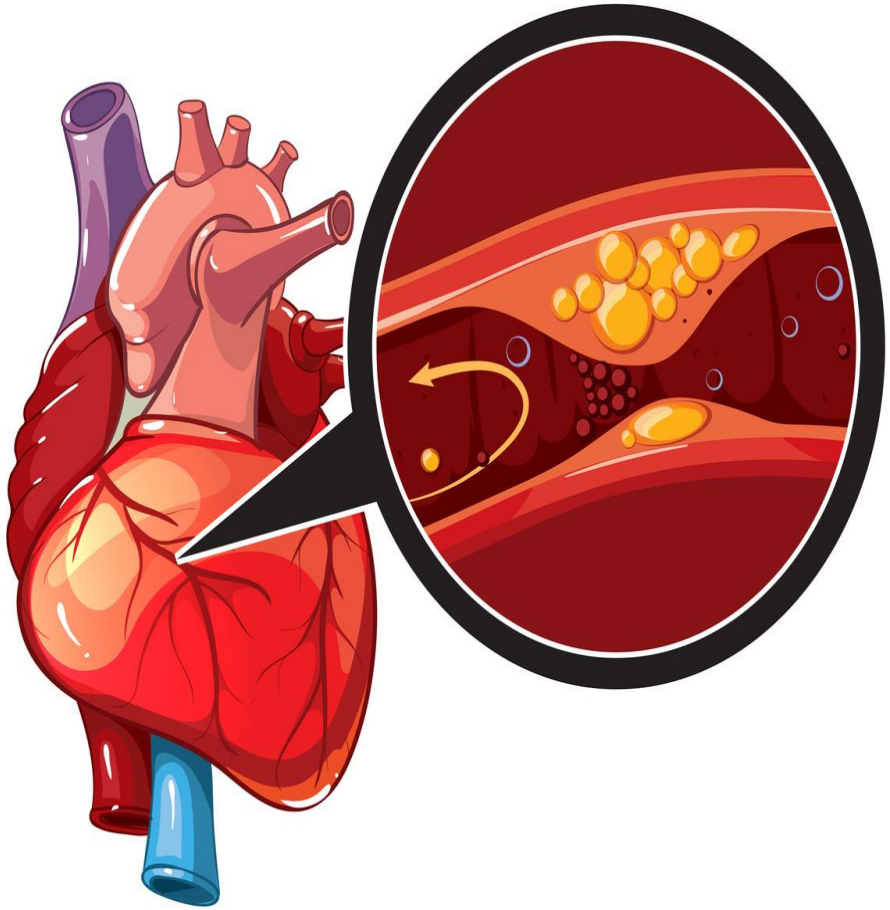
Pathophysiology – atherosclerosis and acute thrombosis

Endothelial dysfunction → inflammation → atherosclerotic plaque formation.

Rupture or erosion of the plaque exposes thrombogenic content → platelet activation and coagulation.

Intraluminal thrombus (subtotal or total occlusion) + vasoconstriction occurs.

Consequence: acute ischemia → electrical instability → necrosis (infarction) depending on the duration and extent of occlusion.



Risk factors of CAD

Nemodifikovateľné	Modifikovateľné (hlavné)
Vek	Dyslipidémia (↑ LDL, ↓ HDL)
Mužské pohlavie / postmenopauza	Arteriálna hypertenzia
Rodinná anamnéza / genetika	Diabetes mellitus / inzulínová rezistencia
	Fajčenie
	Obezita, sedavý životný štýl
	Nevhodná strava, psychosociálny stres

Pozn.: prispievaju also chronic zapalove stavy, CKD, disorders coagulation and zneuzivanie cocaine/amfetaminov.

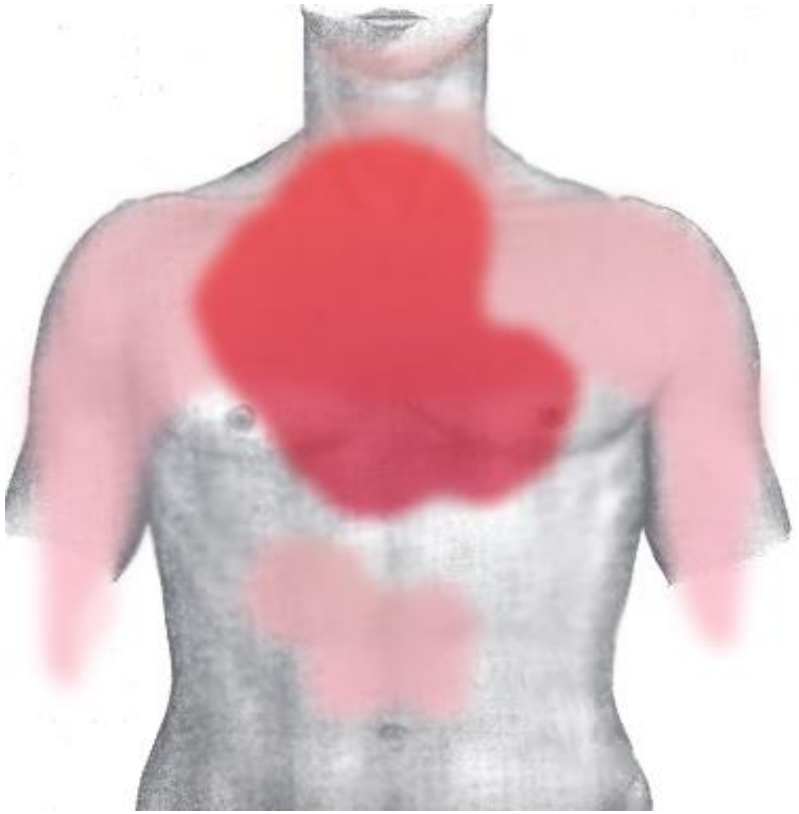
Risk factors of CAD

Non-modifiable	Modifiable (major)
Age	Dyslipidemia (↑ LDL, ↓ HDL)
Male gender/postmenopause	Arterial hypertension
Family history/genetics	Diabetes mellitus/insulin resistance
	Smoking
	Obesity, sedentary lifestyle
	Poor diet, psychosocial stress

Note: chronic inflammatory conditions, CKD, coagulation disorders and cocaine/amphetamine abuse also contribute.

Angina pectoris – typical clinical picture

- Unpleasant pressure/squeezing/burning retrosternal, often diffuse (not “point” pain).
- Radiation: left shoulder and ulnar side of forearm, neck, scapula, epigastrium, interscapular.
- Provocation: physical exertion, cold, emotional stress, postprandial; relief at rest or after nitroglycerine.
- Vegetative accompanying manifestations: sweating, nausea, anxiety; in older/DM may be dominated by dyspnea.



Chest pain – differential diagnosis and „red flags“

- Cardiac: CAD/ACS, pericarditis, aortic dissection.
- Pulmonary: pulmonary embolism, pneumonia, pneumothorax.
- Gastrointestinal: reflux/esophagitis, spasm of the groin, biliary colic.
- Musculoskeletal: costochondritis, vertebrogenic pain.
- "Red flags": persistent pain at rest, syncope, dyspnea, hypotension, new arrhythmia, neurological symptoms, different pulse/BP.

https://www.YouTube.com/watch?in=w8wXdtoW-HQ

chest pain – differential diagnosis and „red flags“

- **pericarditis**
  - long lasting pain independednt of exertion
- **pleuritis**
  - worsening with deep inspiration and breathing
- **esophagitis**
  - burning pain, dysphagia, aftertaste
- **diseases of thorax**
  - changes when coughing, bending forward, turning
- **pulmonary embolism**
  - pleuritic pain, dyspnea, hemoptysis, hypotension, shock
- **dissection aneurysm, peptic ulcer, cholecystitis, pancreatitis**

https://www.YouTube.com/watch?in=w8wXdtoW-HQ

Chronic coronary syndrome (CCS)

- wide spectrum of clinical manifestations of coronary artery disease
- The most common clinical scenarios in patients with suspected or confirmed CCS are:
- Patients with suspected CAD and stable angina and/or dyspnea
- Patients with newly diagnosed heart failure or left ventricular dysfunction and suspected CAD
- Asymptomatic or symptomatic patients with stabilized symptoms after an acute coronary syndrome
- Asymptomatic or symptomatic patients after revascularization
- Patients with angina and suspected vasospastic or microvascular disease
- Asymptomatic patients with CAD detected at screening

Ischemic heart disease

Stable angina pectoris

- a narrowing condition in which the coronary artery is narrowed in some area by atherosclerosis so that blood flow cannot increase to the required level with increased demand, exertion or emotional stress
- a mismatch between oxygen supply and demand = cause of hypoxia
- substernal pain in many modifications
- retrosternal pressure during physical exertion, eating or mental excitement
- radiating chest pain that subsides after administration of nitroglycerin within 1-5 minutes

CAD – Stable AP

Symptoms

- chest pain (ranging from discomfort to a feeling of suffocation)
- referred pain – upper epigastrium, back, neck, jaw, shoulder pain
- possible occurrence of autonomic symptoms – nausea, vomiting, pallor, sweating

Risk factors

- smoking, DM, high cholesterol concentration, high blood pressure, sedentary lifestyle, positive family history, kidney disease, obesity, chronic psychological stress
- AP triggers
- medications (vasodilators, excessive intake of thyroid hormone), vasoconstrictors, polycythemia, hypothermia, hypervolemia, hypovolemia

CAD – Stable AP

- during the attack:
- ↑ BP, pulmonary congestion, there may be heart murmurs, on ECG depression/elevation of the ST segment (subendocardial isch.)
- echocardiography – left ventricular dysfunction
- ECG and echo findings may disappear after the attack (may persist for several hours)
- definition in terms of structural changes:
- a condition in which the lumen of one or more coronary vessels is narrowed by more than 75%
- left ventricular function within normal limits even with such narrowing

Stabilna angina pectoris – klinika and funkcna severity

(Klasifikation Canadian Cardiovascular Society (CCS))

Class 0: Asymptomatic; no symptoms despite mild ischemia.
Class I: Angina only with strenuous, rapid, or prolonged exertion; no angina with ordinary activity.
Class II: Slight limitation of ordinary activity; angina with walking or climbing stairs rapidly, walking uphill, or walking more than 2 blocks on level ground.
Class III: Marked limitation of ordinary physical activity; angina with walking 1-2 blocks or climbing one flight of stairs.
Class IV: Inability to perform any physical activity without discomfort; angina may be present at rest

Atypic forms angina pectoris

- Vasospastic (Prinzmetal's) angina: episodes at rest (often at night/early morning), transient ST elevations; mechanism = coronary spasm.
- Microvascular angina (syndrome X): angina pectoris + objective ischemia in normal/minorly narrowed epicardial arteries; role of endothelial and microvascular dysfunction.
- Silent ischemia: ischemic changes on ECG/monitoring without pain (more common in diabetics, elderly).
- Principles of treatment: control of risk factors + antianginal drugs; in case of vasospasm, CCBs/nitrates should be preferred (carefully with non-selective β-blockers).

CAD

- **Vasospastic angina pectoris (Prinzmetal's, variant)**
- clinical manifestation of myocardial hypoxia even without the presence of atherosclerotic plaque
- occurs in younger people
- fewer risk factors than classic AP, except smoking
- repeated episodes of unexplained chest pain, headache, excessive sweating and/or reduced exercise tolerance
- usually does not result in MI
- occurs without connection to exercise, in the morning or night hours

CAD

- **Risk factors:**
  - recreational drugs – smoking, alcohol, marijuana, cocaine
  - stimulants – epinephrine, dopamine, amphetamines (catecholamine-like stimulants)
  - parasympathomimetics (acetylcholine, methacholine)
  - uterus-contracting drugs
  - some chemotherapeutic agents
  - excessive consumption of energy drinks
  - emotional and physical stress (cold exposure, excessive physical exertion)
- **Mechanism of action:**
  - increased contractility of vascular smooth muscle cells due to defective NO production (NO synthase deficiency) → leads to endothelial dysfunction
    - acetylcholine should stimulate NO production; if NO production is impaired, vasoconstriction occurs
  - thromboxane A2, serotonin, histamine, endothelin
    - abnormal platelet activation causes release of these vasoconstrictors

CAD

- increased activity of **α-adrenergic receptors** in epicardial coronary arteries or excessive release of catecholamines (e.g., **Takotsubo cardiomyopathy**)
- intrinsic hypercontractility of smooth muscle cells in coronary vessels
- decreased parasympathetic activity

Diagnostics

- ECG: **ST-segment elevation** more common than depression
  - sometimes **AV block** or **ventricular extrasystoles**
- widening of the **R wave**
- parasympathomimetics and **methacholine** induce vasospasm
- **β-blockers are contraindicated**
- **Ca^{2+} channel blockers** and **nitrates** cause vasodilation

CAD

Atypical forms of angina pectoris

Silent ischemia (asymptomatic)

- Some individuals have ST segment depression without clinical signs of AP
- Exercise test is usually positive
- Occurs in patients with chronic stable AP
- Holter monitoring reveals episodes of ST segment depression without symptoms
- Occurs mainly in the morning and often correlates with morning BP increase
- AP with normal angiography
- Anginous pain without findings
- Common in young patients, smokers, and women using contraceptives

CAD

- probability of plaque not being visualized by normal methods
- possibility of thrombus in intact vessel
- check hemocoagulation

Coronary syndrome X (microvascular AP)

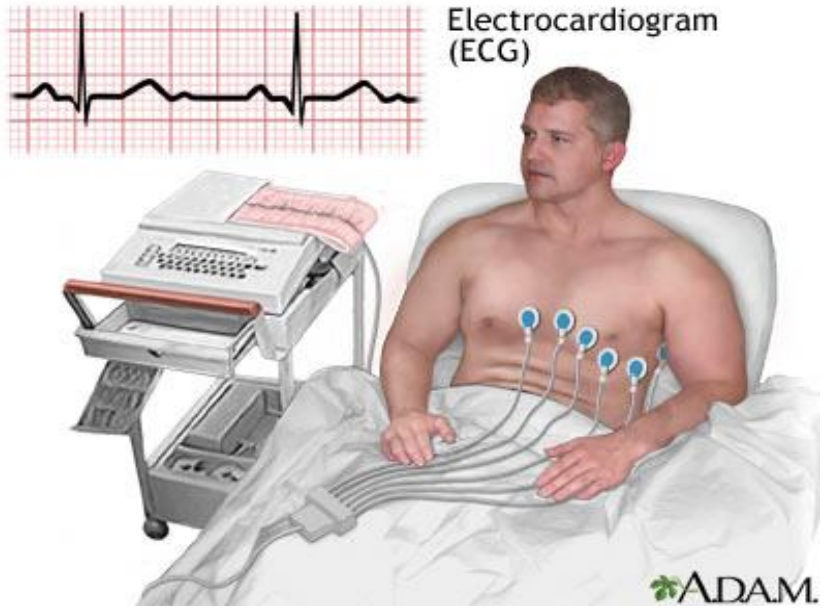
- positive stress test and normal arteriogram
- most common in middle-aged women
- most common myocardial hypoxia in microcirculation (lumen below 100 mcm)
- not visualized angiographically

Kounis syndrome

- acute coronary syndrome caused by allergic or very strong immune reaction (3 types)

Acute coronary syndrome (ACS)

- **ACS represents a sudden deterioration of coronary perfusion – typically after plaque rupture/erosion and thrombosis.**
- **Spectrum: unstable angina (UA), NSTEMI, STEMI.**
- **Differentiation is based on: clinical + ECG + cardiac markers (especially troponins).**
- **In case of persistent pain at rest, always consider ACS until excluded.**



ACS

- acute hypoxic myocardial conditions in relation to the coronary artery

Unstable (crescendo) angina pectoris

- Previous acute coronary insufficiency, intermediate coronary syndrome or preinfarction angina pectoris
- Attacks in various situations, frequent and severe
- Longer duration of pain, also occurs at rest, subsides more slowly (or not at all) after administration of nitrates
- Attack on ECG ST depression and T inversion, more rarely transient bundle branch block and ventricular arrhythmia
- Usually precedes myocardial infarction
- The underlying cause is the formation of microthrombi at the site of narrowing – the cause is an unstable atherosclerotic plaque

ACS

- microscopic dissections form on the plaque → platelet activation → thromboxane A release → vasoconstriction
- damaged endothelium does not produce enough vasodilators
- thrombus emerges from the plaque fissure
- microemboli and vasoconstriction cause endothelial dysfunction
- patients with unstable AP and NSTEMI have subendocardial ischemia and necrosis
- reduced flow is caused by platelet aggregation or occlusive thrombus without plaque rupture
- after platelet activation, vasoconstriction occurs and plaque rupture may occur → ischemia or myocardial infarction

ACS

Acute myocardial infarction without ST segment elevation (NSTEMI)

- also subendocardial or non-Q myocardial infarction
- together with unstable AP is considered to be the result of subtotal occlusion of the coronary artery by thrombus
- ST segment depression and/or T inversion

Acute myocardial infarction with ST segment elevation (STEMI)

- transmural or Q myocardial infarction
- elevation in the relevant limb leads exceeds 1mV, in the thoracic leads 2mV
- clinical symptoms suggest complete thrombotic occlusion of the coronary artery

Acute myocardial infarction

- CVDs account for half of all deaths in developed countries
- 1/3 of CVD patients die from the disease
- 50% die of arrhythmia in the first hour
- A dynamic state in which cessation of perfusion causes irreversible damage
- Anemic necrosis is most often caused by thrombotic occlusion of an atherosclerotic artery or its branch
- Myocyte necrosis occurs 15-40 minutes after vessel occlusion
- Infarction is most often initiated by plaque rupture

Acute myocardial infarction

- plaque rupture → exposure of subendothelial blood components → platelet activation and aggregation → thrombus → myocardial necrosis
- The rupture process is influenced by many factors
- T lymphocytes (part of inflammation) produce soluble cytokines → activation of smooth muscle cell apoptosis → thinning of the fibrous cap
- Increased formation of metalloproteinases (mainly at the site of plaque transition to the arterial wall) during inflammation → increased formation of metalloproteinases → plaque thinning

Acute myocardial infarction

- **Triggering factors**
- **Rarer causes:**
  - **1. acute coronary thrombosis without atherosclerosis**
  - **2. embolic occlusion of the coronary artery**
  - **3. inflammation-induced coronary artery stenosis in coronaryitis**

<u>Heart attack without atherosclerotic changes in the coronary arteries:</u>

- ***1. normal coronary arteries***
- unclear etiology (approx. 5%, age under 35, smokers)
- myocardial contusion (arterial contusion)
- disproportion between oxygen supply and delivery
- cocaine abuse
- erythrocytosis. polycythemia vera, thrombocytosis, DIC, hypercoagulation

Acute myocardial infarction

– ***2. coronary artery embolization***

- infective endocarditis
- thrombus in the left atrium or ventricle
- rheumatic valvular disease (multiple mitral) with atrial fibrillation
- artificial heart valves
- cardiopulmonary bypass
- coronary angiography
- mitral valve prolapse
- nonbacterial thrombotic endocarditis
- thrombi from intracardiac catheters
- myxoma in the left heart

Acute myocardial infarction

– ***3. non-atherosclerotic changes in the coronary arteries***

- arteritis
- thickening of the coronary artery wall in metabolic diseases or intimal proliferation, coronary fibrosis caused by radiation damage, disease of small coronary arteries (below 0.1 mm)
- narrowing of the lumen by other mechanisms (aortic or coronary vessel dissection, coronary vessel spasm)
- congenital coronary abnormalities

Acute myocardial infarction

- MI most often spreads from the subendocardium (most sensitive) to the subepicardium
- the size of the lesion is determined by:
  - the size of the area of blood supply from the occluded coronary vessel
  - the duration of occlusion
  - oxygen consumption at the time of occlusion
  - the amount of resting perfusion through the collateral circulation (most important)
- variable myocardial tolerance to ischemia

Acute myocardial infarction

- **early phase**

➢ first 6 hours from onset of symptoms

➢ therapeutic intervention has the highest probability of success

➢ serious vascular changes in the infarct area

➢ atheromatous material and platelet fibrin mixture can act as an embolizing factor in the bloodstream

➢ endothelial cells are edematous

➢ platelets, Lc and fibrin occlude small vessels → capillary compression → complications in the application of collaterals

➢ increased capillary permeability → even hemorrhages

Acute myocardial infarction

- **heart adaptation phase**
  - approximately from the 6th hour after the first symptoms
  - the left ventricle undergoes geometric reconstruction of the architecture – remodeling (2 processes)
  - infarction expansion
  - late left ventricular remodeling

➢ **infarction expansion**

  - dilation of the ventricle due to acute stretching, thinning, dilation and damage to the myocardial segment
  - causes a decrease in ejection volume (approximately 15% of cases)
  - plays an important role in the further development of the infarction
  - later, the possible development of permanent dilation, aneurysm, rupture of the ventricular septum or free wall of the left ventricle depends on it
  - the wall of the left ventricle at the apex (the thinnest) is very vulnerable
  - in occlusion of the left coronary artery, expansion most often occurs here

Acute myocardial infarction

- expansion and rupture occur more often in transmural infarction, in patients with a first infarction and in hypertensive patients
- expansion occurs in the first hours to days

– **late remodeling of the left ventricle**

- gradual ventricular dilation of the unaffected segment
- from a functional point of view, it is decompensation of loss of function
- ↑ load on the healthy segment → compensatory hypertrophy → can be the cause of heart rhythm disorders, worsen oxygenation (an unfavorable factor for the possible development of heart failure)
- significant impact on later ventricular function and prognosis
- three factors significantly influence the development of remodeling:
- size of the lesion
- ventricular load
- patency of the infarcted coronary artery

Acute myocardial infarction

extension of infarction (enlargement of the infarction)

- further damage to the left ventricle
- assumed based on the re-increase in CK-MB (new necrosis)
- increases hospital mortality

reinfarction

- occurrence of a new infarction
- within a few days to weeks after the first infarction
- from a pathophysiological point of view:

1. myocardial stunning phenomenon (stunned/stiffened myocardium)

- persistence of dysfunction after restoration of perfusion (functional, structural and bioch. changes) days to weeks until full restoration of function after infarction

Acute myocardial infarction

- **2. phenomenon of hibernating myocardium (hypothermia/frozen myocardium)**
- develops during chronic reduction of perfusion or increase in oxygen requirements
- the myocardium suppresses (down-regulates) its function according to oxygen supply
- a new equilibrium (labile) arises, which does not progress to necrosis
- allows a new therapeutic approach for MI up to heart failure
- **3. phenomenon of reperfusion injury (4 types)**
- a) lethal reperfusion injury of myocytes that were viable at the time of reperfusion initiation
- b) vascular reperfusion injury (occurrence of the "no-reflow" phenomenon)
- c) stunned myocardium
- d) reperfusion arrhythmias (ventricular tachycardia and/or fibrillation) a few seconds after reperfusion)

Acute myocardial infarction

processes during acute MI:

- loss of myocardial cell integrity → significant electrophysiological changes → arrhythmias
- significant sympathetic stimulation (also increased β-receptors as a result of ischemia)
- local catecholamines have an arrhythmogenic effect (increased Ca ion influx into the cell by stimulating adenyl cyclase)
- partial inactivation of fast Na channel → decrease in resting potential → decrease in conduction velocity in the infarct area→ arrhythmogenicity due to formation of substitute ectopic sites

metabolic changes leading to arrhythmogenicity:

- increased lactate production → shortening of the refractory period + decrease in resting potential

Acute myocardial infarction

- the spontaneous depolarization phase tends to be shortened
- free fatty acids in the presence of hypoxia or ischemia can reduce the resting membrane potential and accelerate spontaneous depolarization
- there is a relationship between the concentration of free fatty acids in the serum of a patient with AMI and the incidence of ventricular arrhythmias
- insufficient perfusion causes contractility disorders
- there are:
  - dyssynchrony (disorder of the timing of contraction)
  - hypokinesia (decrease in systolic contraction)
  - akinesia (no contraction)
  - dyskinesia (paradoxical systolic bulge)
- the consequences for overall function depend on the size of the area used and the functional state of the intact myocardium

Acute myocardial infarction

– **the first reaction of a healthy myocardium is hyperkinesis**
– compensatory nature
– lasts 2-3 weeks
– more pronounced after successful reperfusion and disappearance of stunning
– if it does not occur, it is a negative prognostic indicator

From the point of view of performance, the following are monitored:

– decrease in ejection fraction
– decrease in stroke volume
– decrease in minute volume
– decrease in blood pressure
– end-diastolic volume of the LV is mostly increased

Acute myocardial infarction

- in a few days, hardening of the affected part – ingrowth of connective tissue → reduction of compliance → disappearance of dyskinesia → improvement of ventricular function
- in the next period, remodeling with an increase in ventricular volume → increase in oxygen demand
- linear relationship between LV function parameters and clinical symptoms of dysfunction
  - 8-10% involvement = decrease in LV distensibility
  - 15% = decrease in ejection fraction, ↑ diastolic pressure and LV volume
  - 25% and more = clinical manifestations of LV insufficiency
  - over 40% = cardiogenic shock, mortality up to 75%
- large infarction → ↓ LV function, ↑ ventricular filling pressure → decrease in ejection volume reduces aortic pressure and coronary perfusion pressure → worsening of ischemia → increase in preload (LV dilation), but ↓ ejection fraction

Myocardial infarction – cellular changes

- If the condition persists, **vacuolization** occurs, with **edema of lysosomes and mitochondria** (damage to the mitochondrial membrane).
- **Ca^{2+} activates many enzymes** (proteases, nitric oxide synthase, phospholipases, endonucleases), leading to **damage of the cytoskeleton** and **injury to membranes, proteins, and DNA**.
- The **inflammatory response and apoptosis** are activated.
- **Leakage of intracellular enzymes** is used for **diagnosis of tissue damage**.
- **Restoration of oxygen supply** leads to **reperfusion injury**.

Key myocyte changes in ischemia

Onset of ATP depletion	Seconds
Loss of contractility	<2 min
ATP reduced	
to 50% of normal	10 min
to 10% of normal	40 min
Irreversible cell injury	20–40 min
Microvascular injury	>1 hr

ATP, adenosine triphosphate.

Reperfusion injury

- arises after restoration of circulation in the damaged part
- this process includes ROS production, influx of Ca2+ and changes in pH
- leads to increased permeability of mitochondrial membranes and cell death
- oxidative stress
- at ischemia lasting more than an hour hypoxanthine arises as a breakdown product of ATP metabolism
- enzyme xanthine dehydrogenase acts at higher oxygen availability as xanthine oxidase = development of superoxide and hydroxyl radicals

Reperfusion injury

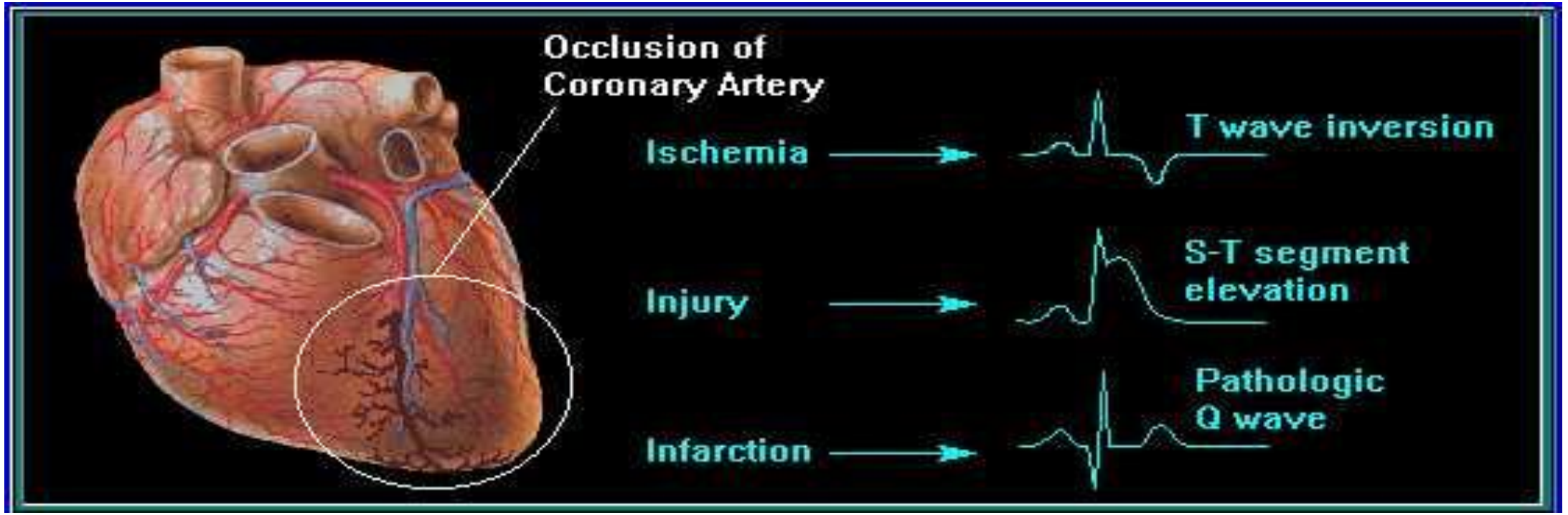
- Xanthine oxidase also produces uric acid (can act as prooxidant or scavenger)
- reperfusion injury can cause hyperkalemia
- increased intracellular calcium
- as a result of ischemic injury to the sarcoplasmic membrane
- increases myocyte lethality – hypercontractility and opening of mitochondrial permeability transition pores (mPTP)
- sudden pH adjustment
- lagging lactate from cells
- leads to opening of mPTP

Reperfusion injury

- inflammation accumulation of neutrophils and migration to myocardium
- release of ROS and proteolytic enzymes

- presupposes coupling of the kinase pathway (RISK) and activation of mPTP
- causes release of calcium to cells and calcium overload

ECG at ischemia and infarctio

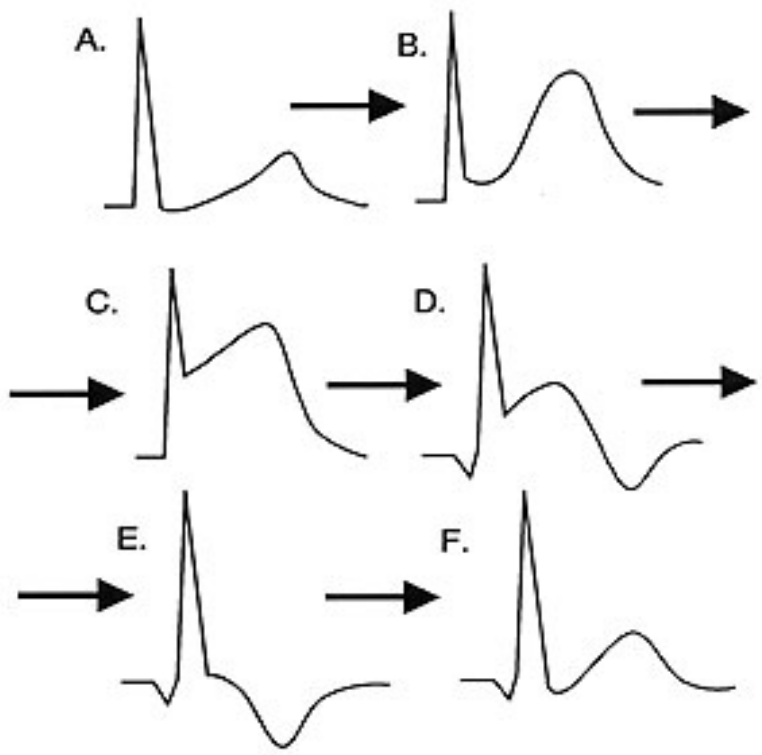


Ischemia: inverzia T / horizontalna or zostupna depression ST.

Acute injury: elevation ST (typic STEMI) – hodnotit in klinickom kontexte.

Necrosis: patologic vlna Q (neskorsi znak).

Evolution of Acute MI (schematic)



Evolution of Acute MI

Hyperacute T waves → ST elevation → Q wave development (not always).

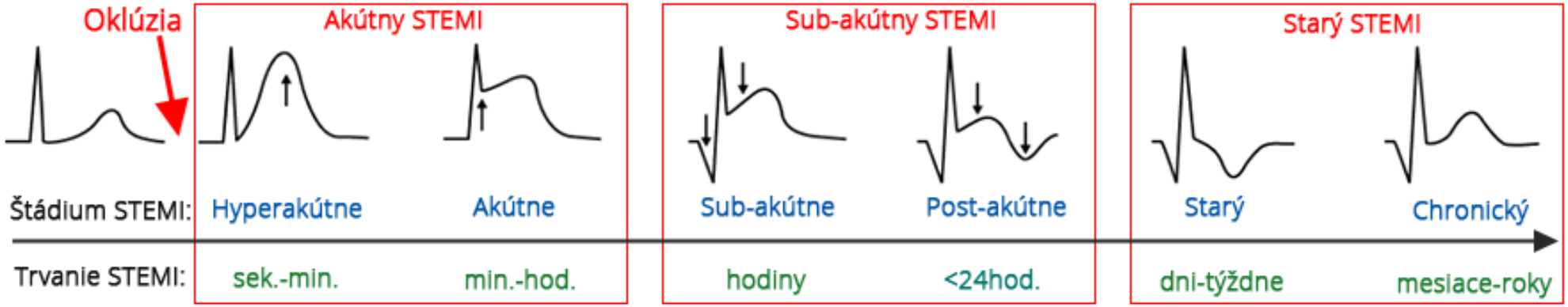
Gradual ST normalization; persistence of T inversion may persist.

Reperfusion may change dynamics (faster ST normalization, earlier “washout” of biomarkers).

Stadia STEMI according to ECG obrazu

https://www.techmed.sk/infarction-myocardium-stemi/

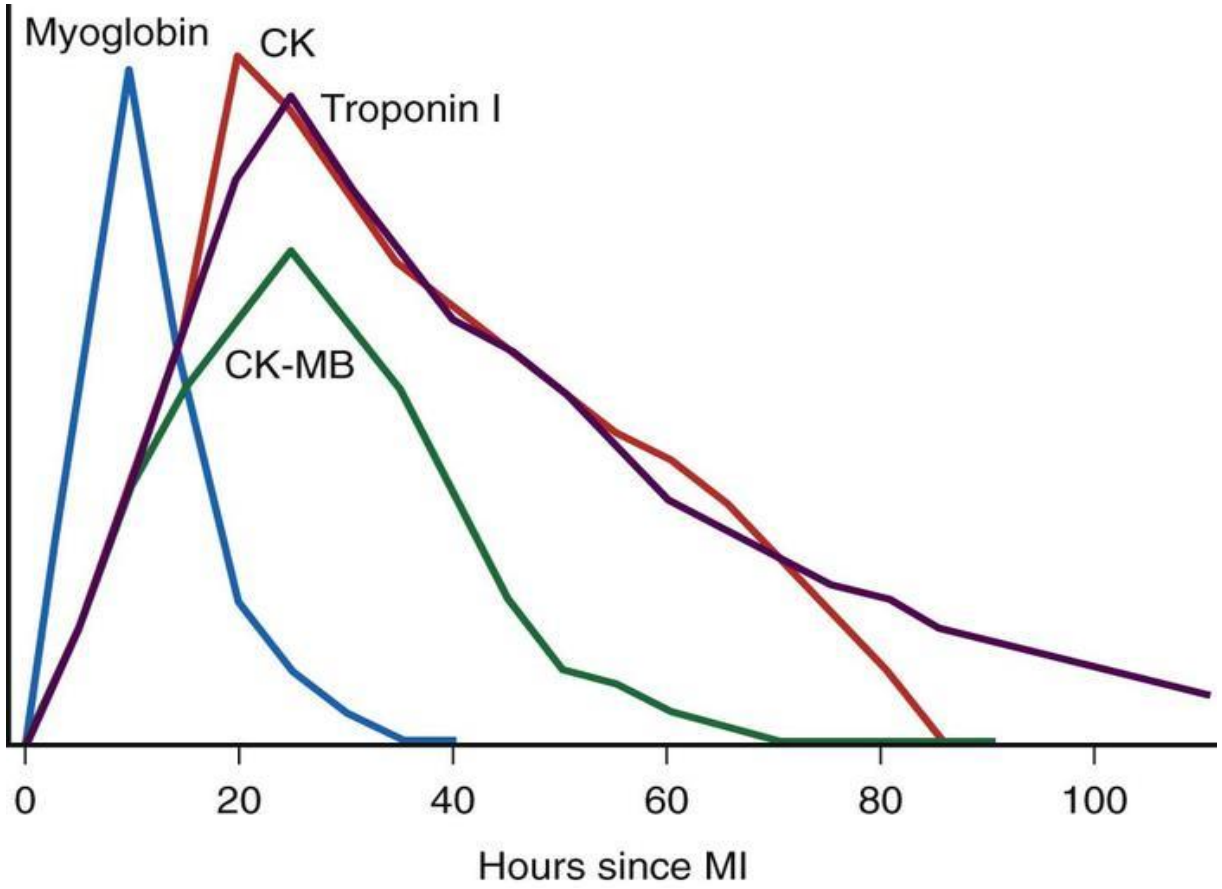
Dynamika ST elevácii pri STEMI

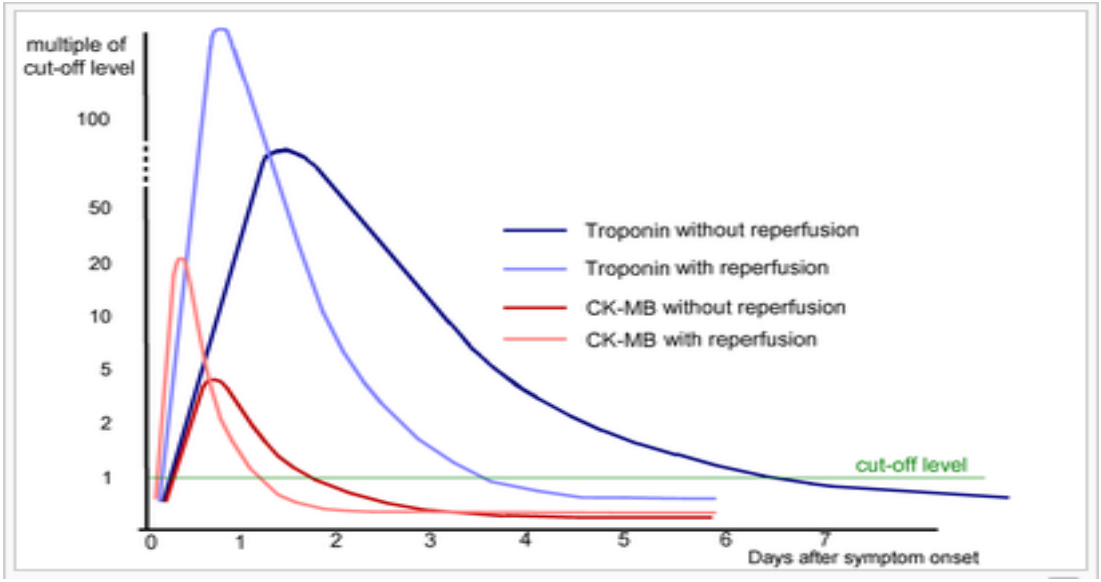


Rozdelenie STEMI podľa štádia

- V klinickej praxi sa STEMI podľa EKG obrazu rozdeľuje na **3 štádia:**
  - **Akútny STEMI**
    - Má **ST elevácie bez Q kmitu**
    - Trvanie: **minúty - hodiny**
  - **Subakútny STEMI**
    - Má **ST elevácie a Q kmit**
    - Trvanie: **hodiny - dni**
  - **Starý STEMI**
    - Má **Q kmit bez ST elevácii**
    - Trvanie: **> týždeň**

Cardiomarkers





Preferred marker of necrosis: (highly sensitive) troponins I/T – assess dynamics (increase/decrease).

CK-MB is important especially in suspected reinfarction or in specific situations.

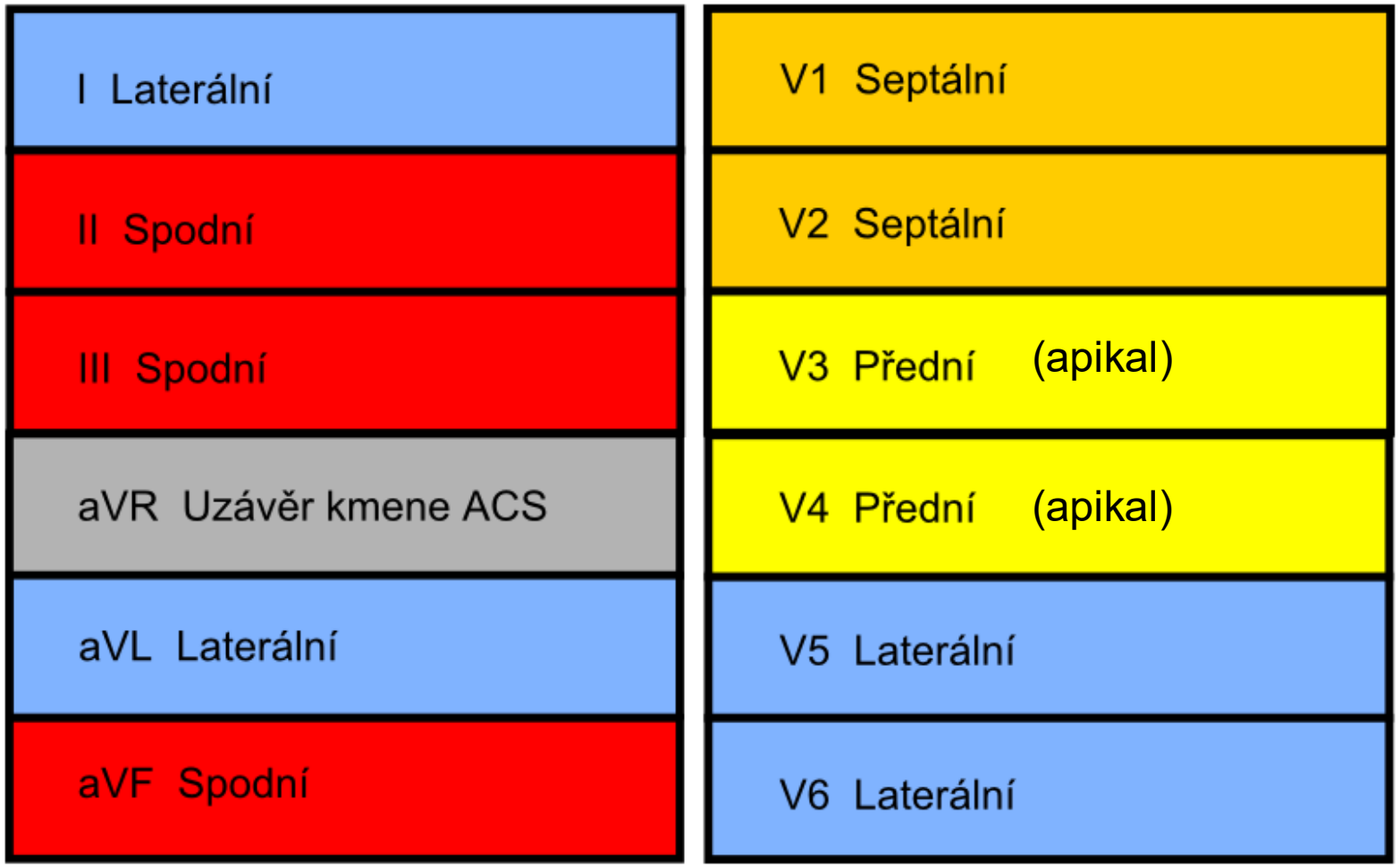
acute myocardial infarction

Akutní koronární syndrom (AKS)			
	STEMI	NSTEMI	Nestabilní AP
Anamnéza	bolest na hrudi	bolest na hrudi	bolest na hrudi
EKG	ST elevace alespoň 2 mm ve svodech V1–V3 nebo alespoň 1 mm ve V4–V6, I, aVL, II, III, aVF. ST elevace musí být patrné alespoň ve dvou sousedních svodech.[7] Čerstvě zjištěný LBBB nebo bifascikulární blok (RBBB + LAH, RBBB + LPH).[7]	ST deprese alespoň 1 mm a/nebo změny T vln (inverze, oploštění) na EKG.[7] Velmi rizikové jsou ST deprese pod 2 mm.[7]	ST deprese a/nebo změny T vln na EKG
Biochemie	pozitivní troponiny	pozitivní troponiny	negativní troponiny

acute myocardial infarction

Lokalizace AIM dle EKG změn. Převzato z [9]. Upraveno dle [7][5].			
Lokalizace	**ST elevace**	**Zrcadlová (reciproká) ST deprese**	**koronární tepna**
Přední (anteroseptální) IM	V1–V4	Ne	RIA
Anteroextenzivní (anterolaterální) IM	V1–V6	Ne/II, III, aVF	RIA
Laterální (boční) IM	I, aVL, V5, V6	II,III, aVF	RCx, RD, RMS, RPLD
Spodní (diafragmatický) IM	II, III, aVF	I, aVL, V1–V4	ACD (85 %), RCx (15 %)
IM zadní stěny	V7, V8, V9	vysoké R ve V1–V3 s ST depresemi ve V1–V3 > 2mm („zrcadlový pohled")	RCx
IM pravé komory	V1, V4R	I, aVL	ACD

acute myocardial infarction



Suspicion on ACS – diagnostic minimum

Quick history (pain character, duration, provocation, risk factors) + physical examination and vital function.

12-lead ECG repeated based on clinical presentation (dynamics of changes can be crucial).

Serial cardiac markers (troponin) – interpreted together with ECG and clinic.

Echocardiography in case of suspected complications or regional kinetic disorder.

Exclusion of alternatives, which require urgent treatment (aortic dissection, pulmonary embolism, pneumothorax).

Myocardial infarction – morphologic changes over time (overview)

Time since occlusion	Dominant finding (typical)
0–24 h	Beginning **coagulative necrosis**, edema, “**wavy fibers**”; **risk of arrhythmias**.
1–3 days	**Neutrophilic infiltration**; progression of necrosis.
3–7 days	**Macrophages**, removal of necrotic tissue; **highest risk of rupture**.
1–2 weeks	**Granulation tissue**, neovascularization, start of scarring.
> 2 weeks	**Mature fibrous scar**, ventricular remodeling.

Complications of acute MI

- Electrical: ventricular tachyarrhythmia/fibrillation, AV block (according to location).
- Hemodynamic: acute cardiac failure, pulmonary edema, cardiogenic shock.
- Mechanical: free wall rupture (tamponade), septal rupture (VSD), papillary muscle rupture (acute MR).
- Inflammatory: pericarditis (early/late), postinfarction syndrome (Dressler syndrome)
- Thromboembolic: LV mural thrombus, systemic embolization; aneurysm development.

secondary prevention

Consistent control of risk factors: LDL, blood pressure, glycemia, weight; non-smoking.

pharmacotherapy according to risk: antiplatelet treatment, statins, drugs to control BP and symptoms (individualization).

Cardiac rehabilitation: gradual return to exercise, education, adherence.

Management of comorbidities (CKD, obstructive sleep apnea, depression) and regular check-ups.

Zhrnutie – „take-home messages“

- IHD/CAD is a direct consequence of an imbalance in O_2 supply and demand; most often on the basis of atherosclerosis.
- angina pectoris is a clinical manifestation of ischemia; stable vs. unstable form has a fundamentally different risk.
- ACS is diagnosed by a combination of clinical features, ECG dynamics and serial troponins.
- Complications of MI can be electrical, hemodynamic also mechanical – temporal incidence is predictable.
- The prognosis is improved most by control of risk factors and long-term secondary prevention.

Odporucane zdroje

ESC Guidelines – management of acute coronary syndromes (updated recommendations; see escardio.org).

ESC Guidelines – chronic coronary syndromes.

Selected chapters from cardiology and pathophysiology (textbooks/scripts according to VL curriculum).

Current local hospital/protocol procedures for ACS (diagnosis and treatment).