Treatment of ischemic heart disease

Ischemic heart disease Ischemic heart disease is a group of diseases which are all characterised by myocardium ischemia(ie, the mismatch between oxygen demand and supply).

Treatment goals

The goal of coronary heart disease treatment is mainly:

- improving the quality of life, thus treating the symptoms of the disease;
- improving the patient's prognosis.

Treatment strategies

- Stopping or slowing down the process of atherogenesis.
- Optimization of the ratio of oxygen consumption and supply in the myocardium.
- Preventing the creation of thrombi.
- Influencing risk factors.

Basic drugs

Organic nitrates

These drugs belong to the group of vasodilators. **They are the drugs of first choice in the treatment of angina pectoris.** Nitrates are converted by the action of free SH-groups of glutathione into nitrosothiol, from which **nitric oxide** (so-called endothelial relaxation factor - EDRF) is released. NO release leads to vasodilation and inhibition of platelet adhesion and aggregation (antithrombotic effect). In atherosclerotic arteries, the production of EDRF is reduced, which explains the beneficial effect of nitrates resulting from the release of NO in atherosclerotic areas.

In the coronary arteries, nitrates cause vasodilation, especially of the atherosclerotized vessels in the epicardium. In the systemic circulation, nitrates induce venodilation with a consequent decrease in venous return and decrease in myocardial oxygen and metabolic demands. náhled|250px|Tepna postižená aterosklerózou The phenomenon of **tachyphylaxis** is of great practical importance . The reduction of the nitrates effect is based on the depletion of free SH groups necessary for the formation of S-nitrosothiol followed by a decrease in NO release. Therefore, nitrates are given at a higher dose in the morning, followed by another dose at noon or afternoon. If the patient does not have nocturnal angina pectoris, they are not given a dose for the night. The organism then has time to synthesize substances containing SH-groups whech were consumed that day.

They are used for angina pectoris, silent myocardial ischemia, acute myocardial infarction, hypertensive crisis and heart failure.

Adverse events include headaches or orthostatic hypotension.

Contraindications of nitrates: **hypotension, increase in intracranial pressure, obstructive cardiomyopathy, aortic stenosis, glaucoma.**

Nitrates are available in various forms for the treatment of acute attacks (iv, sublingual, sprays) or for prophylactic use (controlled-release tablets maintaining stable levels for several hours, patches). Basic substances used:

- ***nitroglycerin***,
- ***isosorbide dinitrate***,
- ***isosorbite mononitrate*** (has a longer biological half-life, due to the slower onset of action it is not suitable for acute use).

Molsidomine does not belong to nitrates, but the mechanism of action is identical, in addition it stimulates fibrinolysis. Treatment with nitrates and molsidomine improves the quality of life, but does not improve the prognosis of the disease. Therefore, all patients with no contraindications should also be treated with beta-blockers.

ACE-i/ARB

Angiotensin-converting enzyme (ACE-i) inhibitors and **angiotensin II receptor (ARB) inhibitors** are used for:

- lowering blood pressure
- reduction of overall cardiovascular mortality

The presumed mechanism of action is slowing down the myocardial remodeling process after infarction, when a post-infarction scar is formed. [1]

Beta-blockers

They provide pain relief in approximately 60% of patients and reduce the number of ischemic periods by more than 75%. They significantly improve the prognosis of patients with coronary heart disease and should therefore be given to all patients who do not have a contraindication.

🔎 *For more information see Cardioinhibitors.*

Calcium channel blockers

The general mechanism of action is blockage of the calcium channels in smooth muscle of vascular walls and in the contractile and conductive cells of the myocardium. The result is: dilatation of the coronary arteries, dilatation of arterioles in the systemic circulation, reduction of contractility, irritability and conductivity of the cardiac muscle. The disadvantage is, that dilatation of coronary arteries is not limited to the affected arteries (as in nitrates), but it also affects healthy arteries, which can lead to redistribution of blood flow from ischemic areas (steal phenomenon) and worsening of ischemic problems. Therefore, the use of these substances is limited to cases, where coronary heart disease is accompanied by peripheral vasospastic disease or by so-called Prinzmetal's angina pectoris (arising from the spasms of otherwise unaffected coronary arteries).

🔎 *For more information see Cardioinhibitors.*

Prevention of thrombus formation

The greatest experience are with **acetylsalicylic acid**. It blocks cyclooxygenase, interrupting the production of thromboxane A2. Of the other antiplatelet agents, promising results have been published with ticlopidine treatment. Due to the relatively high price, it remains reserved for the treatment of patients with salicylate intolerance. Ticlopidine interferes with ADP-induced platelet aggregation, potentiating the effect of acetylsalicylic acid.

Aldosterone blockers

They are recommended for specific group of patients after a heart attack:

- they do not have significant renal dysfunction
- they do not have hyperkalemia
- they receive a therapeutic dose of ACE-i and a beta-blocker
- ejection fraction ≤ 40%
- have diabetes or heart failure [2]

Anticoagulant and fibrinolytic therapy

In acute forms of ischemic coronary heart disease.

Influence of atherogenesis

Hypolipidemic treatment.

Treatment of chronic ischemic heart diesease

Stable angina pectoris

It is based on a combination of β-blocker, nitrates and acetylsalicylic acid.

Spastic AP

Calcium channel blockers are first choice drugs.

Unstable angina pectoris and myocardial infarction

Treatment of all patients should be in cardiac intensive care units and, if this is not possible, in a department where the vital signs can be continually monitored medication immediatelly adjusted.

The usual care

- Bed rest 24 hours,
- i.v. application of 5% dextrose, as a prevention of dehydration,
- "Fasting" for 8 hours (if the pain subsides, a light meal can be served).

Drug treatment

1. **Analgesic therapy** – *morphine sulfate* 2–5mg i.v. every 30 minutes up to a maximum dose of 15mg / h for 3 hours.
2. **Fibrinolytic treatment** – 200mg *hydrocortisone*, , initial bolus of *streptokinase* for 15 minutes, we continue with a continuous infusion of 1 million units over 75 minutes. This is followed by heparinization (3-7 days) and then (3-7 months) antiplatelet therapy.
3. **Sedation** with *oxazepame*.
4. **Oxygen** with a mask or nasal tube 2–4 l / min.
5. Stool softeners

Specific Cardiology medication

1. Nitrates administered i.v. (dose reduction if the systolic pressure falls below 100 mmHg).
2. β-blockers - unless contraindicated.

Sources

Related articles

- Ischemic heart disease
- Vascular supply of the heart
- Chronic ischemic disease of lower extremities
- Myocardium infarction
- Bypass

Used literature

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