Treatment of coronary heart disease

Ischemic heart disease (IHD) is a group of disease states that share ischemia (i.e., a mismatch between oxygen demand and supply) in common.

Treatment Goals

The main goal of CHD treatment is:

- improving the quality of life, i.e. affecting the symptoms of the disease;
- improving the patient's prognosis.

Treatment Strategy

- Stopping or slowing down the process of atherogenesis.
- Optimization of the ratio of consumption and supply of oxygen in the myocardium.
- Prevention of occlusion thrombus.
- Influence risk factors.

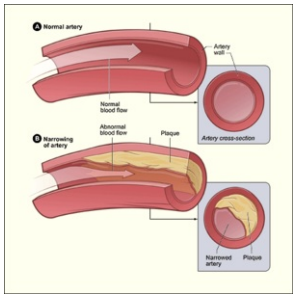
Essential Medicines

Organic nitrates

The drugs belong to the group of vasodilators. **They are the drugs of first choice in the treatment of angina pectoris.** Nitrates are converted to nitrosothiol by the action of free SH-groups of glutathione, from which nitric oxide is released in the endothelium (the so-called endothelial relaxing factor - EDRF), which 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, especially in this location.

In the coronary bed, nitrates cause vasodilation, especially of atherosclerotic vessels in the epicardium. In the systemic circulation, nitrates induce venodilation with a subsequent decrease in venous return and a decrease in the metabolic demands of the myocardium.

The phenomenon of **tachyphylaxis** is of great practical importance. The basis for the reduction of the effect is the depletion of free SH groups necessary for the formation of S-nitrosothiol associated with a decrease in the release of NO. Therefore, nitrates are administered in a higher dose in the morning, with another dose following in the midday or afternoon hours. If the patient does not have nocturnal angina, the night interval without nitrates is left. The organism thus has time to synthesize the substances containing SH-groups consumed that day.



Artery affected by atherosclerosis

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

From the **undesired effects**, we should mention headaches, the occurrence of orthostatic hypotension.

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

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

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

Molsidomine structure does not belong to nitrates, but the mechanism of action is identical, in addition it stimulates fibrinolysis. Treatment with nitrates and molsidomine can be expected to improve the quality of life, but not to improve the prognosis of the disease. Therefore, all patients for whom reasons for contraindications are not found should also be treated with beta-blockers.

ACE-i/ARB

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

- reduction of blood pressure
- reduction of total cardiovascular mortality

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

Beta-blockers

They bring relief from pain in approximately 60% of patients and reduce the number of ischemic periods in more than 75%. They significantly improve the prognosis of patients with CHD and should therefore be administered to all patients who do not have contraindications.

🔎 *For more information see Cardioinhibitors.*

Calcium channel blockers

The general effect is the blockade of the calcium channel in the smooth muscle of the vascular wall and in the contractile and conducting cells of the myocardium. The consequence is: dilation of coronary arteries in their epicardial course, dilation of arterioles in the systemic blood stream, reduction of myocardial contractility, reduction of excitability and conductivity. The disadvantage is that the dilation of coronary arteries is not limited to the affected (as with nitrates) but 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 CHD is accompanied by peripheral vasospastic disease or it is a so-called **Prinzmetal's angina pectoris** (arising on the basis of spasms of otherwise unaffected coronary arteries).

🔎 *For more information see Cardioinhibitors.*

Prevention of thrombus formation

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

Aldosterone blockers

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

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

Anticoagulation and fibrinolytic therapy

In acute forms of CHD.

Influence on atherogenesis

Treatment of hypolipidemics.

Treatment of CHD

Stable - exertional angina pectoris

The basis is a combination of β-lytic, nitrate and acetylsalicylic acid.

Spastic form of AP

Calcium channel blockers are the basis.

Unstable angina pectoris and myocardial infarction

The treatment of all patients should be provided in cardiology intensive care units and, if this is not possible, then in such hospital workplaces, where the equipment and staffing allow continuous monitoring of vital functions and immediate adjustment of medication.

Usual Care

- Rest in bed 24 hours,
- i.v. application of 5% dextrose, as prevention of dehydration,
- *fasting* for 8 hours (if the pain subsides, a light meal can be given).

Drug treatment

1. **Analgesic therapy** - *morphine sulfate* 2–5 mg i.v. every 30 minutes up to a maximum dose of 15mg/h for 3 hours.
2. **Fibrinolytic treatment** - 200 mg of 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 *oxazepam*.
4. **Oxygen** by mask or nasal probe 2-4 l/min.
5. Stool softening preparations.

Specific cardiology medication

1. Nitrates given i.v. (dose reduction if the systolic state falls below 100 mmHg).
2. β-blockers - if not contraindicated.

Links

Related Articles

- Ischemic heart disease
- Vascular supply of the myocardium
- Chronic ischemic disease of the lower limbs
- Heart-attack
- Bypass

References

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