ETHIOPIAN INTERNATIONAL JOURNAL OF MULTIDISCIPLINARY RESEARCH

eISSN: 2349-5715 pISSN: 2349-5707

Volume: 10, Issue 12, Dec-2023 SJIF 2019: 4.702 2020: 4.737 2021: 5.071 2022: 4.919 2023: 6.980

MODERN TREATMENT MECHANISMS IN PATIENTS WITH PRINZMETAL ANGINA PECTORIS (Literary review)

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Annotation: Although coronary artery stenting and CABG are often used today, vasospasm must be kept in mind as a possible cause of CAD. For this group of patients, it is preferable to prescribe calcium antagonists and nitrates. Taking β-blockers in this pathology can lead to a deterioration in the condition, so their appointment, if possible, should be avoided.

Key words: vasospastic angina, nitrates, calcium antagonists.

Relevance. Prinzmetal's angina (synonyms: vasospastic angina (VS), variant angina, spontaneous angina) is a rare type of angina pectoris caused by spasm of blood vessels supplying the heart and accompanied by an elevation of the ST segment. The clinical manifestation of spasm of the coronary arteries is the sudden onset of severe pain at rest or during sleep in the morning, less often in the daytime, with no obvious connection with physical stress, often developing in one and the same time of day. The nature of the pain can be unbearable, the patient is covered with sweat, tachycardia and hypotension may occur. Pain is accompanied by changes on the electrocardiogram (ECG) in the form of ST segment elevation/depression by 2 mm or more (0.2 mV), with the preservation of these signs for 5-10 minutes and is reversible. Any attack of VS can turn into acute myocardial infarction.

A key role in the occurrence of vasospasm is played by local hyperreactivity of the muscle layer of a large epicardial coronary artery [1].

A certain contribution to the development of spasm is made by endothelial dysfunction. The vascular endothelium has a modulating effect on the function of the smooth muscle layer, providing a response of smooth muscle cells to various stimuli. With the intact function of the endothelium and its production of the vasodilating agent NO, the effect of acetylcholine on the vascular wall leads to the expansion of the coronary artery. With endothelial dysfunction due to insufficient activity of NO synthase, NO deficiency develops, and acetylcholine causes vasoconstriction or vasospasm. This mechanism of spasm with the development of ischemia is dominant in microvascular angina (previously called X syndrome), but it is also possible in VS [2, 3].

The above is confirmed by the identification of mutations in β-adrenergic receptors and genes responsible for the activity of NO synthase in patients with VS [4].

One of the diseases affecting the endothelium and leading to its dysfunction is atherosclerosis. On the other hand, vasospasm leads to damage to the endothelium, contributing to a local increase in atherogenesis. The next mechanism of VS is an imbalance of the autonomic nervous system, which is detected in patients with VS, for example, when analyzing heart rate variability with episodes of hypervagotonia and hypersympathicotonia. It determines the development of vasospasm at night, with psychoemotional stress, and the effectiveness of plexectomy, which eliminates the influence of the sympathetic nervous system mediated by the stellate ganglion and helps to stop VS attacks [8].

When diagnosing VS, it is especially important to analyze the symptoms and anamnesis of the patient. Anginal attack is often characterized by patients as a feeling of discomfort, pressure, coma in the throat, heartburn, but it can also be a typical pain that occurs at rest. Meanwhile, each patient may

eISSN: 2349-5715 pISSN: 2349-5707
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have an individual daily periodicity, attacks often occur at rest at night and in the early morning hours, often occur in the form of series with a total duration of 30–60 minutes. The pain syndrome in VS is characterized by a prolonged increase in pain with a faster resolution. As a rule, taking nitroglycerin relieves pain [9].

In the diagnosis of VS, ECG is important - a study at the time of an angina attack and in dynamics - when it stops [10]. For VS, transient ST-segment elevations are most typical, which may be accompanied by reciprocal depressions. After the cessation of the attack, the ECG returns to normal. The diurnal dynamics is also characteristic, with the maximum number of elevations at night. Dynamics of the ECG in VS can be painful and painless. Depression is possible as the only manifestation of vasospasm. Often, despite the typical clinic and the presence of ischemia, ECG dynamics is not recorded, which makes diagnosis difficult. In this regard, and also taking into account the short duration of attacks, 24-hour Holter ECG monitoring (HM-ECG) is of particular importance. Long-term monitoring for 48-72 hours is advisable.

The exercise test for VS is not diagnostic, but sometimes (in 10–30% of patients) exercise can provoke vasospasm [11]. In this case, the test will be positive. ST-segment elevation is most characteristic of VS, which reflects transmural ischemia (as was the case in patient A). Also, the exercise test may be positive with a combination of VS and coronary atherosclerosis. On the ECG in this case, ST segment depression typical of subendocardial ischemia is more often recorded.

Coronary angiography (CAG) is a method that allows visualizing coronary spasm, which can be single-vessel and multi-vessel. The characteristic features of vasospasm in VS are its small extent, proximal localization, development in arteries more than 1.5 mm in diameter. According to the frequency of spasm localization, the RCA leads, then the LCA, and then the circumflex artery [1]. In this case, most often the focus of the spasm is fixed (the proximal segment of the RCA is most typical), as in patient A., less often the spasm migrates along the artery. Multivessel spasm is extremely rare. This spasm is mostly diffuse. It often leads to the development of acute myocardial infarction, more extended, hyperreactivity of the arteries not affected by spasm is characteristic, and the basal tone of the coronary arteries is increased.

Due to a special pathogenetic mechanism, which is mainly due to arterial spasm, VS requires special treatment, in which the main place is occupied by agents that cause relaxation of the muscle elements of the coronary vessel. Such drugs today are calcium antagonists and nitrates. In terms of effectiveness, non-dihydropyridine calcium antagonists, and first of all verapamil, are in the first place.

β-blockers in VS can often provoke vasospasm and worsen the course of the disease.

Diagnostic tests can be used to diagnose VS during CAG. They are divided into permissive and provocative. From a safety point of view, resolving samples are the most preferable. Most often, intracoronary administration of nitroglycerin is performed, followed by resolution of vasospasm. However, spontaneous vasospasm in CAH is rarely detected, in this case, if VS is suspected, provocative tests are necessary. In international recommendations, four methods most often appear: two pharmacological ones, with the introduction of ergonovine (ergometrine, methylergometrine) or acetylcholine, as well as cold and hyperventilation tests. The last two are safer in relation to the development of refractory vasospasm, but they are inferior to pharmacological ones in terms of information content.

A test with ergometrine or ergonovine makes it possible to differentiate VS from cardiac syndrome X, in which the provocation of an angina attack also occurs, is accompanied by ischemic dynamics, and spasm of the coronary artery is not visualized. The ergonovine test has the highest

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sensitivity, but is associated with a high risk of complications. These include refractory spasm with the development of myocardial infarction, life-threatening bradyarrhythmias and ventricular arrhythmias. Vasoconstrictor reactions are possible in the area of atherosclerotic plaques [14]. The test is contraindicated in widespread and multivessel atherosclerosis, reduced left ventricular ejection fraction, aortic stenosis, recent myocardial infarction, life-threatening rhythm and conduction disturbances.

Less sensitive, but safer test with intracoronary administration of acetylcholine. Due to the risk of atrioventricular block, temporary pacing may be justified. Attitude to provocative samples is ambiguous. In the Bulletin of KazNMU, No. 2-2015 83 www.kaznmu.kz The use of ergometrine is prohibited in the United States, and ergonovine is limited. In the 2013 EKO recommendations, CAG with the use of nitrates or calcium antagonists corresponds to class I recommendations, a test with vasospasm provocateurs corresponds to class IIa, in the 2011 ACA recommendations with the use of nitrates it corresponds to class I, and a test with vasospasm provocateurs corresponds to class IIb or III (depending on the presence and severity of concomitant atherosclerosis) [10, 15].

In addition to the characteristic clinic, the absence of classical risk factors for atherosclerosis, except for smoking, a relatively young age, as well as the presence of diseases often combined with it: Raynaud's disease, migraine, "aspirin" asthma, Brugada's syndrome, insulin resistance, inflammatory diseases, helps to suspect a patient with VS. For cardiologists, it is especially important to know the provocative role of β-blockers for the development of coronary artery spasm in VS. It is these drugs that are the drug of choice for the treatment of coronary artery disease due to their negative chronotropic and inotropic effect, which saves the myocardium from lack of oxygen in conditions of ischemia due to atherosclerosis of the supply vessels. In patients with VS, the vascular response to the use of β-blockers is specific. The specificity of patients with VS is that the structure and function of β2 receptors responsible for vasodilation are changed in them. Vasospasm in most patients with VS is increased when β-blockers block the already weakened β2-receptors, while α-receptors, which promote vasoconstriction, are activated [9]. Non-selective β-blockers and monotherapy with β-blockers are not recommended for VS. If β-blockers do not worsen the course of the disease, and especially if there is an atherosclerotic lesion of the coronary bed, their use may be justified.

It is known that ASA reduces the synthesis of prostacyclin and prostaglandins, which can lead to increased coronary spasm in some patients with VS. Due to the presence of atherosclerosis and percutaneous transluminal balloon coronary angioplasty (TBCA), ASA treatment in our patients was continued and did not cause vasospasm provocation.

Thus, although coronary artery stenting and CABG are often used today, vasospasm must be kept in mind as a possible cause of CAD. For this group of patients, it is preferable to prescribe calcium and nitrate antagonists. Taking β-blockers in this pathology can lead to a deterioration in the condition, so their appointment, if possible, should be avoided.

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