

CAUSES, DIAGNOSIS AND MODERN CLINICAL DIAGNOSTIC METHODS OF ANGINA PECTORIS

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Introduction: Angina is rarely described by the patients themselves as "pain". In most cases, patients complain of a feeling of discomfort behind the sternum, and the localization of these sensations can also vary. Treatment may include antiplatelet drugs, nitrates, beta-blockers, calcium channel blockers, angiotensin-converting enzyme inhibitors, statins, coronary angioplasty or coronary artery bypass grafting. Angina can manifest as a mild, non-distracting pain or can quickly develop into a strong, intense feeling of pressure in the precordial region.

Sometimes this feeling of discomfort is localized in the upper abdomen. It is characteristic that with stable angina, pathological sensations are never localized above the ears and below the navel. Angina pectoris is a clinical syndrome characterized by discomfort or tightness in the precordial region, which is caused by transient myocardial ischemia without the development of infarction. In most cases, angina attacks develop against the background of physical or emotional stress and pass at rest or after sublingual administration of nitroglycerin.

The feeling of discomfort can spread to the left shoulder and extend to the fingertips of the left hand. Pain may occur in the back, throat, lower jaw and teeth, radiating to the inner surface of the right arm. The diagnosis of the disease is established on the basis of clinical manifestations, ECG changes and various methods of visualization of myocardial ischemia.

Research methods and materials: In addition, endothelial dysfunction may contribute to changes in arterial tone: for example, in the endothelium affected by atherosclerosis, a "catecholamine surge" causes more vasoconstriction than vasodilation (the normal response).

The narrowing of blood vessels in atherosclerosis is not completely static, the size of the vascular lumen is affected by changes in vascular tone, which is usually present in all people; It has been found that in most patients, angina attacks occur in the morning hours, when there is an increase in vascular tone.

When myocardial ischemia occurs, a decrease in blood pH is observed in the coronary sinus, the release of potassium ions into the extracellular space, the accumulation of lactate, changes in the ECG are noted, and a decrease in ventricular contractility (systolic and diastolic) is noted. During an attack of angina pectoris, an increase in diastolic pressure in the LV is usually observed, which is sometimes accompanied by congestion in the lungs and shortness of breath.

The exact mechanism responsible for the feeling of shortness of breath during an angina attack is unknown, but stimulation of nerve endings by metabolites formed during hypoxia may be involved.

Results: The frequency of attacks can increase (called progressive angina), which leads to myocardial infarction or death. The frequency of attacks can vary from several attacks per day to prolonged periods of absence of clinical symptoms lasting weeks, months, or years.

Conversely, attacks can gradually decrease or disappear, if adequate collateral coronary circulation develops, a necrotic focus appears at the site of the ischemic area, or heart failure or intermittent claudication develops, limiting activity. Nocturnal angina attacks can also be a manifestation of left ventricular failure, which is equivalent to nocturnal attacks of shortness of breath. In the supine position, venous return is increased, which leads to myocardial stretching and increased myocardial tension, which in turn increases myocardial oxygen demand.

Nocturnal angina attacks are caused by changes in breathing, heart rate, and blood pressure that occur during sleep. Rest angina is angina that occurs spontaneously in the supine position, but not necessarily at night. It is usually accompanied by a slight increase in heart rate and sometimes a significant increase in blood pressure, which, accordingly, increases the demand for myocardial oxygen. On the other hand, an increase in blood pressure and heart rate can provoke the development of an angina attack, and they can be the result of myocardial ischemia in response to rupture of atherosclerotic plaque and formation of a thrombus in a coronary artery. If an angina attack lasts a long time, the imbalance between myocardial oxygen demand and supply increases, which increases the likelihood of myocardial infarction. If the patient has a normal resting ECG and is able to exercise, an exercise stress ECG is performed. In men with chest discomfort suggestive of angina, the sensitivity of the stress ECG is approximately 70% and the specificity is approximately 70% (1). These values are somewhat lower for women. In addition, women with coronary artery disease are more likely to have resting ECG changes than men (32% vs. 33%).

Despite the high sensitivity of the exercise stress test, false-negative results are possible in severe forms of coronary heart disease (main or three-vessel disease). A positive test is the basis for further investigation. In patients with an atypical clinical presentation, a negative exercise test usually excludes angina and cardiovascular disease.

The choice of imaging technique depends on its availability in the clinic and the experience of the investigators. Imaging techniques allow assessment of LV contractile function at rest and in response to stress, identification of areas of ischemia, myocardial infarction, and viable myocardium, and localization and distribution of the risk zone.

Stress echocardiography also allows the diagnosis of mitral regurgitation associated with ischemic papillary muscle dysfunction. Stress myocardial imaging is performed in conditions where the resting ECG is abnormal, as false-positive ST-segment elevations are common on the stress ECG. Exercise or pharmacological therapy (eg, dobutamine and dipyridamole infusions) may be used. Imaging techniques include stress echocardiography, myocardial perfusion imaging with single-photon emission CT (SPECT) or PET, and stress MRI.

Conclusion: However, because calcium can be detected in the absence of significant stenosis, this index does not correlate well with the need for PCI or CABG. Based on these findings, the American Heart Association recommends that CT be performed only in a limited population of patients and in conjunction with clinical and medical history to assess the risk of fatal or nonfatal MI (4). Electron beam CT allows us to measure the amount of calcium in atherosclerotic plaque in the coronary artery. The use of electron beam CT is essential to exclude serious coronary disease in patients presenting to the emergency department with atypical symptoms, normal troponin levels, and a low probability of hemodynamically significant coronary artery disease. The calcium index is associated with the risk of developing coronary artery disease.

These groups may include asymptomatic patients with an intermediate 9-year risk estimate for atherosclerotic cardiovascular disease (10–20%) and symptomatic patients with inconclusive stress test results.

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