Journal of Science in Medicine and Life
Volume: 3 Issue: 1 Year: 2025
ISSN: 2992-9202 | http://journals.proindex.uz

Types of Angina, Modern Methods of Diagnosis, Origin and Prevention

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Abstract: Angina 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 disappear after rest or sublingual administration of nitroglycerin. The diagnosis of the disease is established on the basis of clinical manifestations, ECG changes, and various imaging methods of myocardial ischemia. Treatment may include antiplatelet drugs, nitrates, beta-blockers, calcium channel blockers, angiotensin-converting enzyme inhibitors, statins, coronary angioplasty, or coronary artery bypass grafting.

Key points: Etiology, Pathophysiology, Clinical manifestations, Diagnostics, Treatment.

Introduction

The workload of the heart exceeds the ability of the coronary arteries to deliver the required amount of oxygenated blood. This mismatch between the amount of oxygenated blood delivered and the amount of oxygen needed can occur when the arteries narrow. Constriction usually occurs as a result of

Acute coronary artery thrombosis can cause angina even in cases of partial or temporary obstruction, but in most cases it leads to the development of acute myocardial infarction (MI).

Since the myocardial oxygen demand is determined mainly by factors such as heart rate, systolic tension of the left ventricular wall, and its contractility, angina attacks develop mainly during physical exertion and pass with rest.

In addition to physical activity, conditions such as arterial hypertension, aortic stenosis, aortic insufficiency, or hypertrophic cardiomyopathy also increase the load on the myocardium and can cause angina. In such situations, angina pectoris can occur regardless of the presence of atherosclerotic lesions of the coronary arteries. In addition, in the presence of these conditions, myocardial perfusion is reduced due to the development of diastolic dysfunction, accompanied by an increase in myocardial mass and a decrease in coronary myocardial perfusion during diastole.

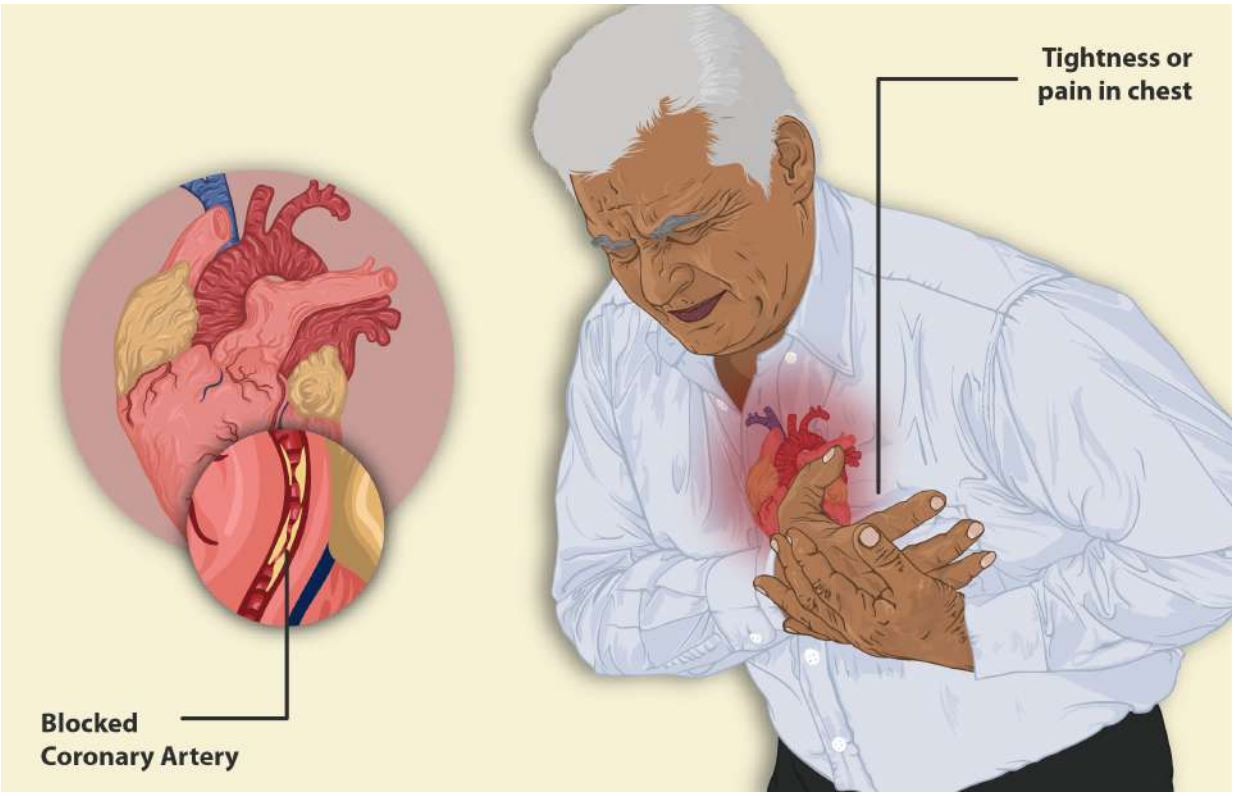
Reduced oxygen delivery to the myocardium is also common in conditions such as anemia and hypoxia, which can initiate and worsen angina.

Pathophysiology of angina pectoris

- Stable
- Unstable

With stable angina, the relationship between the level of load, the increase in myocardial oxygen demand, and the threshold for the development of myocardial ischemia is relatively predictable.

Unstable angina is a clinically worsening angina (e.g. angina at rest or with increasing frequency and/or intensity of episodes).



In atherosclerosis, vasoconstriction is not completely static, the size of the vessel lumen is affected by changes in vascular tone, which is usually present in all people. It has been found that angina attacks in most patients occur in the morning, when vascular tone is increased. In addition, endothelial dysfunction may contribute to changes in arterial tone: for example, in the atherosclerotic endothelium, a "catecholamine surge" leads to greater constriction than to vasodilation (the normal response).

When myocardial ischemia occurs, the pH of the blood in the coronary sinus decreases, potassium ions enter the extracellular space, lactate accumulates, changes are recorded on the ECG, and a decrease in ventricular contraction (systolic and diastolic) is noted. During an angina attack, there is usually an increase in left ventricular diastolic pressure, which is sometimes accompanied by pulmonary congestion and shortness of breath. The exact mechanism responsible for the feeling of shortness of breath in an angina attack is unknown, but stimulation of nerve endings by metabolites formed during hypoxia may be important;

Signs and symptoms of angina

Angina can manifest as a mild, non-disturbing pain or quickly develop into a strong, intense feeling of pressure in the precordial region. Angina is rarely described by patients themselves as “pain”. In most cases, patients complain of a feeling of discomfort in the chest, and the location of these sensations can also vary. 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. 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.

In some patients, atypical angina may be accompanied by bloating, gas, abdominal pain, or burning and tenderness in the back, shoulder, arm, or jaw, and is more common in women. Such patients often associate the symptoms with indigestion; chewing may even relieve the symptoms. In other

patients, dyspnea is characteristic, which occurs due to a sharp transient increase in LV filling pressure in response to their ischemia. Often, patients' complaints are characterized by vagueness, vagueness, and it is very difficult to differentiate between shortness of breath, angina pectoris, or a combination of these clinical manifestations. Because the clinical manifestations of myocardial ischemia completely regress within a minute or so, short-term, rapidly passing chest pain is rarely associated with angina pectoris.

In the interictal period and even against the background of a current attack of angina, physical examination data may be normal. However, at the height of the attack, an increase in heart rate, blood pressure, increased heart sounds, and apical impulse expansion are noted. Splitting of the second heart sound (S2) may occur due to an increase in the duration of LV systole during the attack. In many patients, the 4th heart sound (S4) is less pronounced than the 3rd heart sound. A systolic murmur, whistling or "blowout" occurring in the middle or end of the tone at the apex of the heart - but not very loud - may be associated with local ischemic dysfunction of the papillary muscle, which in turn leads to the development of mitral insufficiency.

Angina attacks usually occur with physical exertion or strong emotions, last for a few minutes, and are relieved by rest. The exercise capacity of a patient with stable angina is predictable, but the amount of exercise may vary due to changes in vascular tone. Angina attacks may be exacerbated by physical exertion after meals, as well as in cold weather. In addition, anginal pain occurs when walking against the wind, as well as when leaving a warm room for cold weather. The severity of angina is usually classified by the level of physical activity that provokes an angina attack (see table: Canadian Cardiovascular Classification System of Angina Pectoris).

The frequency of attacks can vary from a few attacks per day to prolonged clinical episodes lasting weeks, months, or years. The frequency of attacks may increase (called progressive angina), leading to myocardial infarction or death. Conversely, attacks may gradually decrease or disappear if adequate collateral coronary circulation develops, a focal necrosis develops in the ischemic area, or heart failure or intermittent claudication develops, limiting activity.

Nocturnal angina attacks are caused by changes in breathing, heart rate, and blood pressure that occur during sleep. 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, there is an increase in venous return, which leads to stretching of the myocardium and an increase in its tension, which in turn increases the myocardial oxygen demand.

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 anginal attack, and they can be the result of myocardial ischemia in response to rupture of an atherosclerotic plaque and the formation of a blood clot in the coronary artery. If an anginal attack is prolonged, the imbalance between the need for and supply of oxygen to the myocardium increases, which increases the likelihood of developing myocardial infarction.

Unstable angina

Because the characteristics of angina are usually predictable for each patient, any change (e.g., rest angina, new-onset angina, worsening angina, new-onset nocturnal angina, or new-onset postural angina) should be considered serious, especially if the angina is severe (i.e., grade 3 or 4 according to the Canadian Cardiology Society classification). Such changes are called unstable angina and require immediate diagnosis and treatment.

Diagnosis of angina pectoris

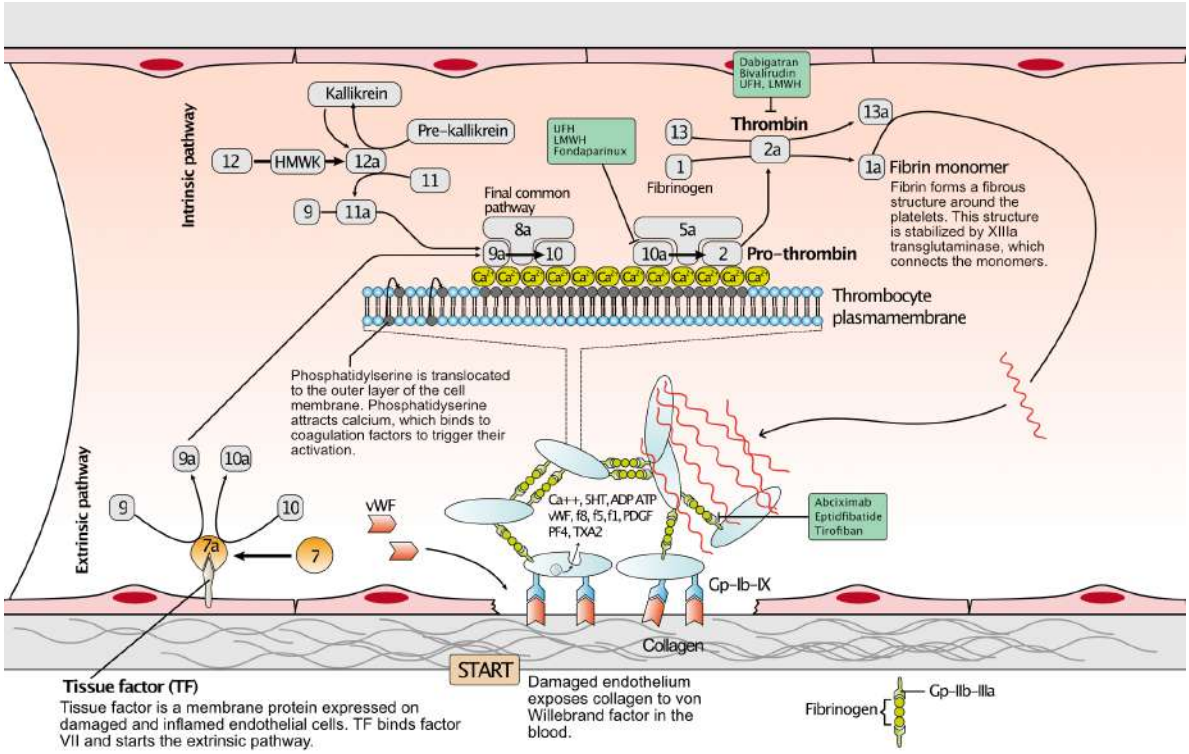
a. Characteristic symptoms

b. Electrocardiography (ECG)

c. Stress testing using an ECG or imaging (echocardiography, radionuclide imaging, positron emission tomography [PET], or MRI)

d. Fractional flow reserve (FFR) assessment based on CT angiography or CT data

e. Coronary angiography in the presence of severe symptoms, a positive stress test, or significant lesions detected on CT-CPR.



The diagnosis of angina is usually suspected when chest discomfort is present, which is triggered by physical activity and relieved by rest. A history of significant risk factors for coronary heart disease (CHD) may exacerbate existing symptoms. If this discomfort persists for more than 20 minutes or occurs at rest, or in patients with episodes of loss of consciousness or heart failure, an evaluation for acute coronary syndrome should be considered.

The occurrence of chest discomfort can also be associated with disorders of the gastrointestinal tract (for example, reflux esophagitis, esophageal spasm, dyspeptic symptoms, cholelithiasis), rib pathology, anxiety states, panic attacks, hyperventilation, as well as other heart diseases (for example, aortic dissection, pericarditis, mitral valve prolapse, supraventricular tachycardia, atrial fibrillation), in which there are no changes in coronary blood flow.

An ECG is always performed. More specific tests are a stress test with an ECG or myocardial imaging (e.g., echocardiography, radionuclide study, MRI) and coronary angiography. Noninvasive studies are performed first.

ECG

ECG recording is indicated in the presence of typical clinical manifestations - attacks of discomfort behind the sternum during physical exertion. Since anginal attacks stop at rest, it may be difficult to record an ECG directly during an attack, except during a stress test.

If an ECG is performed during an angina attack, it may show reversible ischemic changes:

Discoordination of the QRS complex with the T wave

ST segment depression (most common)

ST segment elevation

Decreased R wave height

Intraventricular or bundle branch block conduction disorders

Arrhythmia (usually ventricular extrasystoles)

Between angina attacks, the ECG (and usually LV function) remains normal at rest in 30% of patients with angina, even in the presence of three-vessel coronary artery disease. In 70% of patients, the presence of a previous myocardial infarction, signs of hypertrophy, and nonspecific changes in the ST segment and T wave on the ECG is not sufficient to make or rule out the presumed diagnosis.

Stress test

a. For this, you need to conduct a stress test.

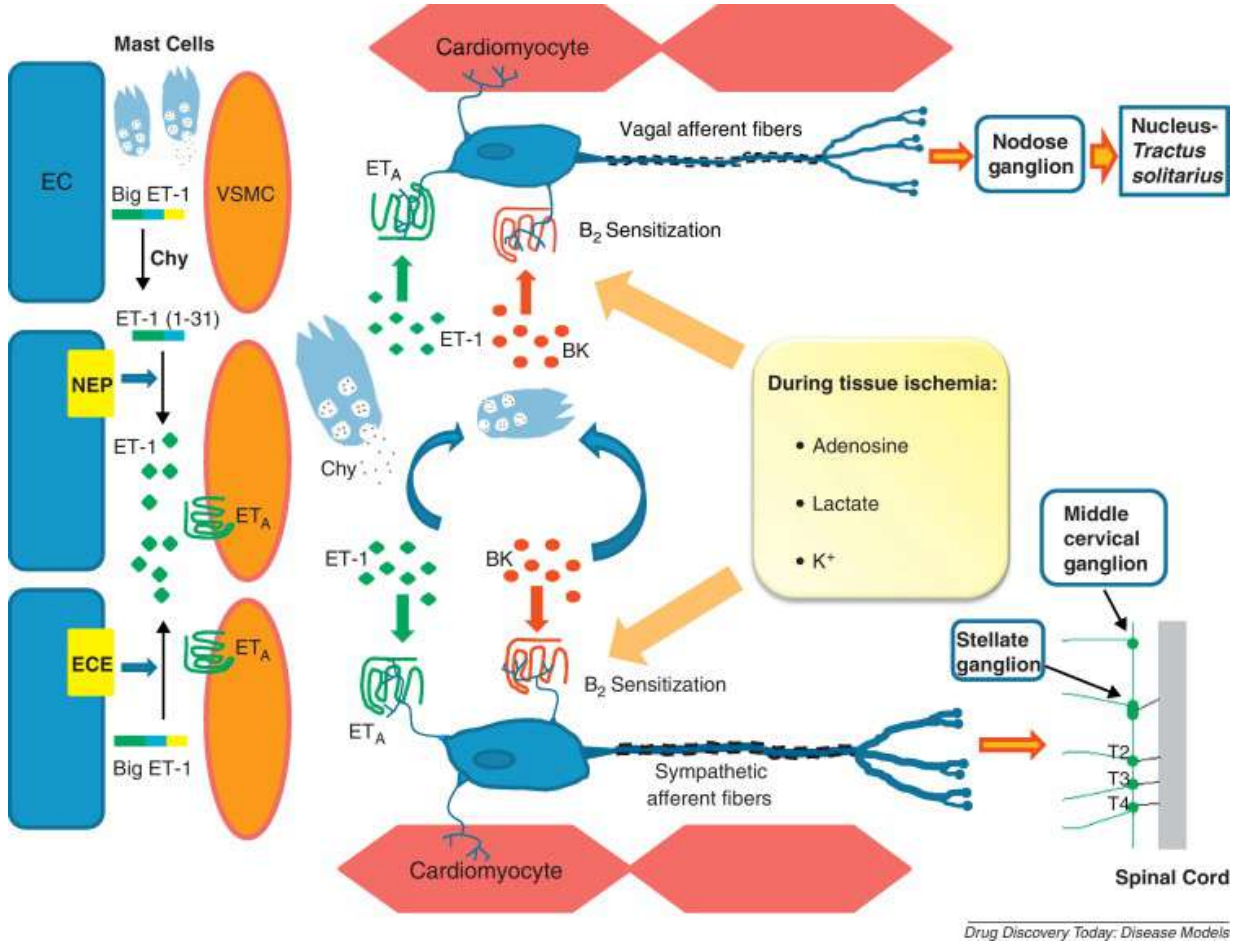
b. Confirming the diagnosis

c. Assessing the severity of the disease

d. Determining the appropriate level of physical activity for the patient

e. Help in the initial assessment of the prognosis of the disease

f. If a clinical or working diagnosis of unstable angina is established, early stress testing is contraindicated.



Exercise ECG is performed when the patient has normal resting ECG and is able to exercise. In men with chest discomfort suggestive of angina, the sensitivity of the stress ECG is approximately 70% and the specificity is approximately 80% (1). These figures are somewhat lower for women. However, women with coronary artery disease have more frequent ECG changes than men (32% and 23%, respectively). Despite the high sensitivity of the exercise test, false-negative results are possible in severe forms of coronary artery disease (main or three-vessel disease). A positive test is the basis for further investigation. In patients with an atypical clinical presentation, a negative stress test usually excludes angina pectoris and ischemic heart disease.

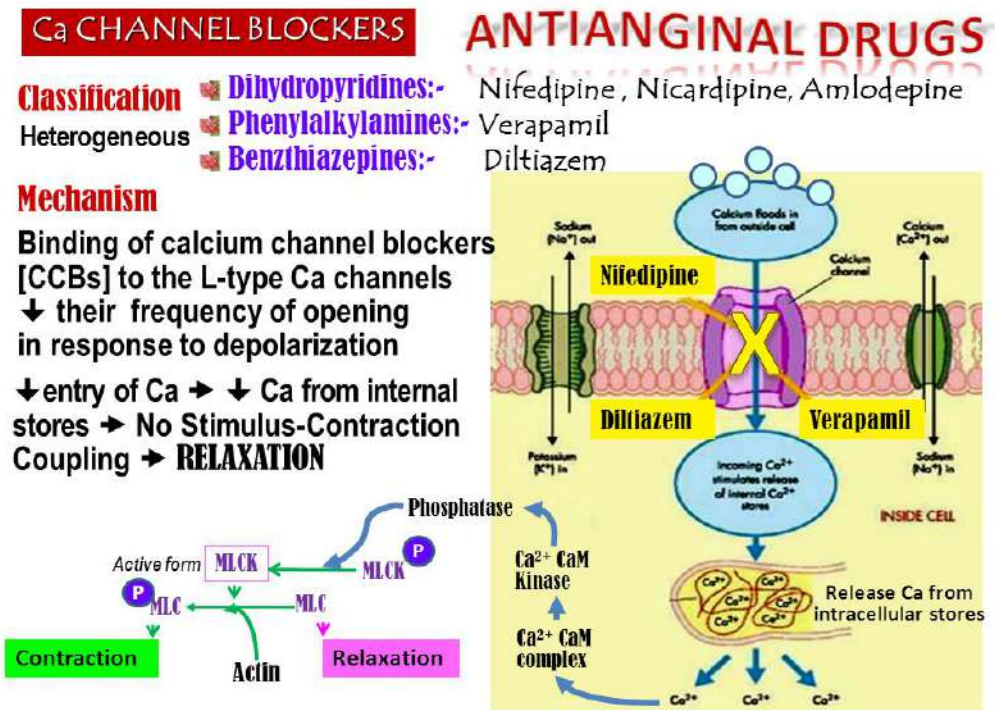
Myocardial stress imaging is performed in conditions where the resting ECG is abnormal, as false-positive ST-segment elevations are common on exercise ECGs. Exercise or pharmacological stress (eg, dobutamine and dipyridamole infusions) may be used. Imaging techniques include stress echocardiography, myocardial perfusion imaging with single-photon emission CT (SPECT) or PET, and stress MRI. The choice of imaging technique depends on its availability in the clinic and the experience of the investigators. Imaging techniques allow the assessment of LV contractile function at rest and in response to stress, the detection of ischemia, myocardial infarction, and viable myocardium, and the location and extent of the risk zone. Stress echocardiography, in addition, allows the diagnosis of mitral regurgitation associated with ischemic papillary muscle dysfunction.

Coronary angiography

Coronary angiography is the gold standard for diagnosing coronary artery disease, but it is not always necessary to confirm the diagnosis. It is primarily indicated to determine the location and severity of coronary lesions; this information is necessary for planning the tactics of myocardial revascularization (percutaneous coronary intervention [PCI] or coronary artery bypass grafting [CABG]). CG is also necessary to obtain information about the coronary artery during an occupational examination or to develop recommendations for lifestyle changes (stopping work, playing professional sports). Although angiography does not directly demonstrate the hemodynamic significance of coronary lesions, if the lumen diameter is reduced by >70%, the obstruction is considered to be of physiological significance. When the diameter is reduced by <70%, in the absence of spasm or thrombosis, angina usually does not develop.

Intravascular ultrasound (IVUS) provides images of the structure of the coronary arteries. For this, an ultrasound probe is placed inside the lumen of the coronary artery during angiography. This test provides more information about the condition of the coronary arteries than other methods; it is indicated if the nature of the lesions is unclear or the severity of the disease does not correspond to its symptoms. In combination with PCI, it helps to ensure optimal placement of stents.

A catheter guide with a blood flow or pressure sensor can be used to assess blood flow at stenotic sites. This is expressed as the fractional flow reserve (FFR), which is the ratio of the peak flow at the stenotic site to the normal peak flow. These blood flow measurements are most useful in assessing the need for angioplasty or CABG in patients with indeterminate lesions (40-70% stenosis). An FFR of 1.0 is considered normal, while an FFR of <0.75-0.8 indicates myocardial ischemia. Stent placement is considered inappropriate for lesions with an FFR >0.8.



Optical coherence tomography (OCT) is another imaging modality that can be used during coronary angiography. This modality uses near-infrared light to produce high-resolution cross-sectional images of the coronary arteries, which is superior to IVUS (2 , 3). OCT has been used to optimize stent placement and sizing during PCI. However, its role in the routine clinical care of patients with CAD undergoing angiography is not fully understood.

Coronary artery imaging

Non-invasive imaging techniques performed at rest can assess the condition of the coronary arteries.

Electron-beam CT can measure the amount of calcium in atherosclerotic plaque in the coronary arteries. The calcium index is associated with the risk of coronary artery disease, but 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 CT scanning only in limited patient groups and in conjunction with clinical and historical data to assess the risk of fatal or nonfatal MI (4). These groups may include asymptomatic patients with a 10-year intermediate risk of atherosclerotic cardiovascular disease (10–20%) and symptomatic patients with questionable stress test results. 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.

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