

Original Article: Angina Pectoris



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ABSTRACT

The most common cause of angina is atherosclerosis of the coronary arteries. Signs and symptoms of angina pectoris appear when one or more coronary arteries are more than 75% blocked. Angina pectoris is derived from the Greek word meaning chest compression. The presence of angina indicates cardiac ischemia. Ischemia was associated with short-term angina. It does not lead to permanent damage to the heart muscle tissue, but it is nevertheless a life-threatening factor and can further lead to dysrhythmia and myocardial infarction. Angina pectoris is caused by a temporary ischemia caused by an imbalance between the supply and demand of oxygen required by the heart muscle. Angina pain is often relieved by rest and consumption of nitroglycerin and its accompanying symptoms include: shortness of breath, tachycardia, palpitations, nausea, vomiting, fatigue, sweating, paleness, weakness and syncope may be associated with Angina to be seen. Contact with cold and drinking cold liquids causes the arteries to constrict, reduces coronary blood flow, and increases the myocardial need for oxygen. Eating too much food reduces coronary blood flow and increases myocardial oxygen demand due to the diversion of blood to the gastrointestinal tract. Stress and anxiety, accompanied by the release of catecholamines into the bloodstream, increase blood pressure and increase heart rate and increase myocardial oxygen demand.

Introduction

A variety of factors affect the diameter of the coronary arteries and are able to upset the balance between oxygen supply and demand and lead to angina pain [1-3]. These factors fall into two categories:

A) Factors that reduce the diameter of these vessels by a direct effect on the coronary artery:

- ✓ Coronary artery spasm [4-6].

- ✓ Coronary artery arthritis [7].

- ✓ Coronary hypertrophy [8-10].

B) Factors that lead to angina pain of non-coronary origin:

- ✓ Aortic stenosis [11].
- ✓ Aortic insufficiency [12].
- ✓ Mitral examination [13].
- ✓ Mitral stenosis [14].

Characteristics of angina pain

Angina pain has the following characteristics:

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A) Diffuse pain: Angina pain may spread to the left shoulder and then extend from the inside of the left arm to the elbow, wrist or fingers [15].

B) Initiating factors: Any activity that increases the demand for heart muscle, such as exercise, exposure to cold [16-18], heavy food consumption, sexual intercourse may cause angina pain to begin [19-21].

Clinical manifestations

Myocardial ischemia causes chest pain that varies in severity from light and superficial pressure under the sternum to severe pain with panic and death and imminent stimulation [22-25]. The pain is usually felt deep in and behind the chest in the upper or middle third of the sternum. The pain is mostly focal, but can spread to the neck, shoulders, and inner surface of the upper limbs [26-28]. The patient often has a feeling of cramping with a quality of constant pressure. Feeling weak or swollen wrists, arms may be accompanied by pain [29-31].

Note: One of the important characteristics of angina pain is the elimination of the causative agent [32-34].

Note: In people with diabetic neuropathy or the elderly with advanced atherosclerotic stenosis, like other symptoms without pain [35-37]. Older people get angina pectoris due to the reduction of subcutaneous fat with the slightest cold [38].

Division of angina based on clinical symptoms

Angina pectoris is classified according to clinical signs as follows:

A) Stable angina: This angina is caused by activity. Exercise increases the oxygen consumption of the heart muscle [39-41]. This pain is relieved by resting, lowering blood pressure, and lowering the number of heartbeats [42-44]. Eventually each stable angina becomes unstable and eventually MI, but this time varies greatly [45].

B) Unstable angina: In the past, this type of angina was called angina before a heart attack. Angina, which used to be caused by a specific activity, does not require much activity now and occurs at rest and even at night while sleeping [46-48]. The characteristics of angina pain have changed, its severity and duration have increased, i.e., the patient used to have pain every few months or every few weeks, but now he does not need much activity to cause pain and pain occurs every day [49-51]. After a long rest, it takes some time for the pain to heal. There is also pain at night and it wakes the patient. There is pain almost all day and all night [52-54]. Previously, he did not have nausea, vomiting and cold sweats when he was in pain, but now, with the onset of heart pain, he has nausea, vomiting and cold sweats [55-57]. On the other hand, sometimes taking 4-5 tablets of sublingual nitroglycerin takes a quarter to half an hour to reduce the pain a little [58-60]. In some patients, the transition from stable to unstable angina may take years, but in some it may take less than a month [61-63].

Note: This type of angina is the most dangerous type of angina, because its mortality is very high, if it is not diagnosed or admitted to the CCU, there is a possibility of sudden death due to a very high arrhythmia [64-66].

Note: ECG changes in patients with unstable angina may be normal, but a patient with a normal ECG should not be called healthy. Clinical symptoms and the quality of pain are the most important criteria for diagnosis [67-69].

Note: QT prolongation, ST fall, sometimes U-wave appears when in pain. All ECG changes resolve after the end of angina pain [70].

C) Nocturnal angina: In this angina, the patient wakes up at night due to pain and mentions the specific symptoms of frontal chest pain with

spread to the neck, left hand, right hand and epigastrium. Angina usually occurs in the REM phase of sleep with dreaming [71-73]. At this stage, due to the sympathetic increase, the number of pulses and blood pressure increases. In other patients, the cause of the abnormal decrease in aortic diastolic pressure and increased venous return due to increased fluid during sleep is the result of increased heart diameter and increased myocardial oxygen consumption [74-76], which is the most important cause of nocturnal angina. The onset of anterior myocardial infarction with nocturnal angina indicates a worsening of the disease and is more common in people with all three coronary arteries or left main coronary arteries. This angina often indicates left ventricular failure [77-79]. At night, the venous return to the heart increases due to the supine position. This increase in venous return has increased the left ventricular diameter. As a result, diastolic end pressure and diastolic end volume increase. As a result, the elasticity of myocardial fibers increases [80-82]. This increase in traction increases myocardial oxygen consumption and pain appears. The best medicine for this angina is a drug that reduces the diameter of the left ventricle, thus reducing traction and reducing myocardial oxygen consumption [83-85]. Digoxin is the drug of choice for nocturnal angina.

D) Angina pectoris: Angina, in which pain attacks occur when bending or lying down, is a similar cause to nocturnal angina. It occurs mostly at night when sleeping, causing the person to wake up, and is now accompanied by shortness of breath [86-88], nocturnal attacks, and resolves shortly after sitting or standing. The cause of this type of angina is increased venous return to the heart [89].

E) Prinzmetal angina: It is a variant of angina. Its clinical symptoms are completely different

from other angina and it lacks the two properties of classical angina [90].

- ✓ Chest pain is caused by both exercise and rest.
- ✓ It is often associated with changes (ST segment) as the ST segment rises during pain and normalizes after pain on the ECG.

The pain usually occurs periodically at certain times each day. Occurs especially when waking up or early in the day (morning). This is because when you wake up, the level of catecholamine increases, which in turn increases the activity of the heart and the heart muscle's need for oxygen. This angina is caused by spasm of the coronary arteries and can lead to the sudden death of unstable angina and myocardial infarction [91].

The various mechanisms of angina pectoris include: When the resting state is converted to the activity of the sympathetic system, the level of catecholamine increases and spasm occurs in the coronary artery, eventually leading to pain. The drug of choice for this type of angina are drugs that dilate the smooth wall of blood vessels, such as calcium block calcium.

F) Angina variant: Angina variant is not much different from Prinzmetal angina, but in terms of division, both are in the same category. Today, angina is more commonly used for coronary spasms that occur on primary atherosclerotic plaques, but the word Prinzmetal is used to describe spasms in healthy coronary arteries, but in both cases the word can be used interchangeably.

G) Resistant or rebellious angina: Chronic severe resistant angina with recurrent attacks that do not respond to medical treatment.

H) Angina after infarction: After myocardial infarction due to residual ischemia around the site of necrosis, chest pain occurs periodically,

which can be relieved with proper medical treatment.

The purpose of treatment measures in angina pectoris

- ✓ Improve acute attack.
- ✓ Prevent subsequent attacks to reduce the risk of myocardial infarction.

Types of angina treatment:

There are usually two ways to treat angina.

A) Medical or non-invasive surgery:

B) Surgery:

A) Medical or non-invasive surgery: non-invasive non-surgical method is performed. The goals of angina medical care and treatment include reducing the heart muscle's need for oxygen. Medically, drug therapy and control of risk factors are used to achieve these goals. Drug treatment includes: nitrates, beta-blockers, calcium channel blockers. Antiplatelet drugs Nitroglycerin is the best drug used to treat angina and acute attacks of coronary heart disease. Nitroglycerin reduces venous return and preload due to dilation of coronary arteries and decreased venous resistance. It reduces afterload by dilating the aortic artery and reducing venous return and diastolic end volume. It reduces overload and preload and allows more blood to flow through the collateral by directly dilating the large arteries.

Note: The best way to take this drug is sublingually, which enters the bloodstream quickly through the sublingual veins.

Most of the oral type is metabolized by the liver, and a small amount of it passes through the liver, acts within 1 hour, and becomes ineffective within 5 hours. Normally, 4 tablets are taken daily. It is useful to spray it in the mouth. The type of ointment is mostly used during sleep. Adequate nitrate reaches the patient through the skin throughout the night and the patient

does not have ischemia during sleep. Ointments work by gradual absorption and are gradually metabolized and excreted. In the case of ointment, the amount is usually increased until a headache or excessive effects of blood pressure or heart rate appear. It is then reduced to a maximum that does not cause these side effects. The ointment should be applied to the skin and replaced regularly.

The skin under the ointment sheet should be examined for redness and swelling. Sublingual nitroglycerin works within 1 minute and becomes inactive within 1 hour. Patients with angina, especially the unstable type, should always have the drug with them. Immediately put under the tongue as soon as you feel pressure in the chest, neck, hands or pain in the epigastrium. Sometimes the drug must be taken prophylactically.

For example, they should take medication before having sex or when leaving the house in cold weather or in any situation that may cause pain. A hospitalized patient with signs and symptoms of recurrent ischemia or after continuous or intermittent intravenous infusion of nitroglycerin may be prescribed.

The amount of nitroglycerin administered is based on the symptoms of the disease to prevent side effects such as hypotension. Usually, 90 mg or less should not be given if systolic blood pressure is present. Nitroglycerin may be used topically for 24 hours.

Educate the patient if he or she is using a sublingual tablet so that he or she does not suck saliva before dissolving. If the pain is severe, the tablet can be crushed with your teeth to speed up sublingual absorption. Nitroglycerin tablets should be stored in a dark glass container. Nitroglycerin is a flammable substance, so it loses its effect over time due to heat, moisture, light.

If the nitroglycerin tablet is fresh, the patient may feel burning under the tongue and often feel full or throbbing in the head. Nitroglycerin

tablets should be renewed every 6 months. If there is still pain and discomfort after taking nitroglycerin, the possibility of MI is imminent. If the pain does not go away after taking 3 sublingual tablets 5 minutes apart, the patient should go to the nearest emergency room. The patient should be advised to sit after taking nitroglycerin tablets to prevent hypotension and syncope.

The most common complication of headache is due to vasodilation of cerebral arteries. Other side effects include dizziness, fainting, flushing, palpitations, low blood pressure, and tachycardia. Beta-adrenergic blocking drugs reduce myocardial oxygen consumption by slowing the heart rate. On the other hand, they reduce the strength of contraction, lower blood pressure and reduce oxygen demand. Propranolol can be prescribed with isosorbide to prevent angina and coronary spasm.

Beta-blockers reduce the prevalence of recurrent angina and myocardial infarction. Cardiac side effects include hypotension, bradycardia, advanced ventricular block, and exacerbation of congestive heart failure. If the drug is administered intravenously for acute cardiac events, the ECG should be carefully monitored for the patient's rate, heart rate, and blood pressure after administration. Because some beta-blockers stimulate beta-adrenergic receptors that narrow the bronchi, the drug is contraindicated in patients with obstructive pulmonary diseases such as asthma.

Other side effects include worsening hyperlipidemia, depression, fatigue, decreased libido, and hiding the symptoms of hypoglycemia. They should avoid abrupt discontinuation of the drug, as it aggravates angina pain and myocardial infarction.

The dose should be gradually reduced for a few days before stopping the drug. Patients with diabetes who are taking beta-blockers should be instructed to monitor their blood glucose levels regularly to determine if hypoglycemia may be a

side effect of the drug. Beta-blockers exacerbate it in people who have nightmares. It should not be used in people with impotence. Other complications of bradycardia include musculoskeletal weakness, hypotension, and decreased mental activity.

Conclusion

These drugs reduce myocardial muscle contraction and dilate coronary arteries. By dilating the arterioles, myocardial oxygenation increases, and by lowering systemic arterial blood pressure, the ischemic myocardial need for oxygen decreases.

Nifedine: Nifedine is able to reduce blood pressure by reducing the smooth muscle tone of the effect of high environmental dilation. Complications include hypotension, hot flashes, dizziness, and headache, and sometimes the patient discontinues the medication due to hot flashes and dizziness.

Verapamil: Verapamil is used with anti-arrhythmic properties (reduces the automatic power of the atrial sinus node, prolongs the transmission time of the impulse from the atrial node, thus reducing the heart rate). This drug slows the heart rate but should always be used with caution with beta-blockers.

Diltiazem: Diltiazem has the properties of both drugs and is the best drug to control pain. It is contraindicated in congestive heart failure or should be used with caution because it impairs myocardial contractility. These drugs should be administered at intervals of 4-6 hours.

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