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MITRAL VALVE STENOSIS

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ABSTRACT

Mitral valve stenosis (Latin: stenosis ostii atrioventricularis sinistri stenosis, also known as mitral stenosis or narrowing of the left atrioventricular orifice) is a common heart disease. It leads to impaired diastolic blood flow from the left atrium to the left ventricle. The disease is characterized by a slow progression. The onset of symptoms is most often between the ages of 40 and 50. The disease is more common in women. There is a complaint of shortness of breath during physical exertion due to high pressure in the pulmonary arteries.

This article will discuss the causes, symptoms and consequences of mitral valve stenosis, as well as methods of treatment and prevention.

Key words: Pulmonary hypertension, stenosis, shortness of breath, cardiac asthma, cat wheezing, gibbus cordis, habitus gracilis, acrocyanosis, facies mitralis, electrocardiography, quail rhythm.

INTRODUCTION

Mitral stenosis is a narrowing of the left atrioventricular orifice, which makes it difficult for blood to flow out of the left atrium and leads to an increase in diastolic pressure between it and the left ventricle. The most common cause of mitral stenosis is rheumatic endocarditis. In most cases, it is latent and not diagnosed in time. Although the disease usually begins to develop in childhood or adolescence, complaints that force the patient to consult a doctor (shortness of breath, decreased work capacity, etc.) appear at a young age (25-40). It is 2-3 times more common in women than in men. Rheumatic stenosis of the left atrial septum is most often observed in combination with mitral valve insufficiency[15-22]. Although there is no obvious stenosis in endocarditis due to other causes, in some cases, with timely treatment of infective endocarditis, a mild narrowing of the atrioventricular orifice may be detected without impaired intracardiac hemodynamics. In very rare cases, mitral stenosis is observed in atherosclerosis. In some patients, acquired or congenital mitral stenosis is accompanied by a defect in the interatrial septum and is called Lutambashe syndrome. The following are characteristic signs of mitral valve damage in rheumatic endocarditis:

- Thickening of the mitral valve leaflets (swelling due to inflammation, and subsequently the development of fibrous tissue)
- Fusion of the leaflets
- Fusion and shortening of the leaflet chordae
- Calstinosis of the leaflets, fibrous ring, and subvalvular structures.

HEMODYNAMICS

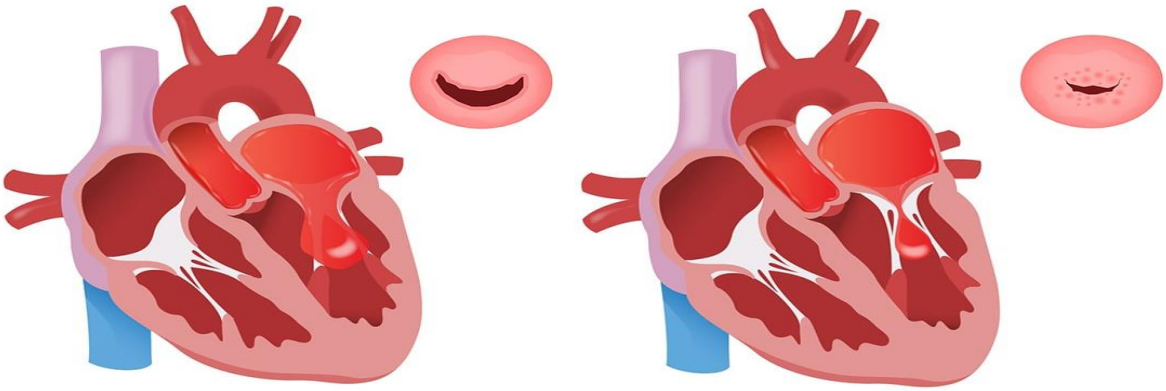
Hemodynamic changes characteristic of mitral stenosis occur as a result of a significant narrowing of the opening between the left atrium and the ventricle.

* Left atrial hypertrophy and dilatation. The area of the left atrioventricular orifice is normally 4-6 cm². An orifice area of 4 cm² or less creates an obstacle to the flow of blood from the left atrium to the left ventricle during diastole ("first obstacle") and can be overcome only by increasing pressure in the atrium. As a result of increasing pressure in the left atrium, its hypertrophy and subsequent dilatation are formed. Pulmonary hypertension. The next hemodynamic change in mitral stenosis is associated with stagnation of blood in the pulmonary circulation

and an increase in pressure in the pulmonary artery. Two forms of pulmonary hypertension are distinguished:

"Venous ("slow") pulmonary hypertension. An increase in pressure in the left ventricle (normally 8-12 mm Hg) (25-30 mm Hg) makes it difficult for venous blood to flow through the small circulation. As a result, the venous vessels fill with blood and blood stagnates in the lungs. High pressure in the pulmonary veins. Naturally, through the capillaries, it is transmitted to the pulmonary artery, and as a result, the so-called "venous" or "slow" pulmonary hypertension develops;

"Arterial" (active) pulmonary hypertension. In patients with a very narrow mitral valve, a sharp increase in pressure in the left ventricle (more than 25-30 mm Hg) is observed, which increases the risk of rupture of the pulmonary capillaries or the development of alveolar edema in the lungs. To prevent these complications, a reflex defense occurs - a spasm of the pulmonary arterioles (Kitaev reflex). As a result, the amount of blood flowing from the right ventricle to the pulmonary capillaries decreases sharply, but at the same time, the pressure in the pulmonary artery increases sharply (active pulmonary hypertension).



Clinic: Clinical signs of mitral stenosis are related to the degree of narrowing of the left atrioventricular orifice, the pressure difference between the left atrium and the left ventricle, and the degree of pulmonary hypertension. Based on the size of the mitral orifice, Goldstein distinguishes three degrees of mitral stenosis:

-- Mild mitral stenosis - orifice area from 2.0 cm^2 to 4.0 cm^2, no clinical symptoms or shortness of breath during physical exertion (FS II);

-- Moderate mitral stenosis - orifice area from 1.0 cm^2 to 2.0 cm^2, shortness of breath during physical exertion, fatigue, and right ventricular failure (FS II, III;)

Severe mitral stenosis - orifice area less than 1.0 cm^2, shortness of breath during physical exertion, pulmonary edema, fatigue, and right ventricular failure.

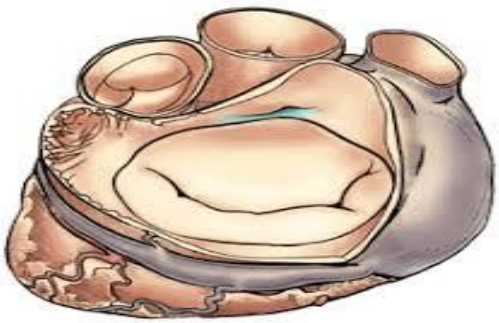
Despite the fact that this defect is formed at a young age, the first symptoms that force the patient to consult a doctor appear after 10-15 years.

Complaints: Compensated mitral stenosis does not cause any unpleasant subjective sensations in patients, sometimes only palpitations may occur. Later, symptoms associated with pulmonary hypertension appear: shortness of breath, attacks of cardiac asthma, dry cough, which can later turn into a wet cough with blood, palpitations[1-15]. Later, there may be short-term pain around the heart due to prolonged or low-intensity physical exertion. In rare cases, patients who are in motion for a long time may be disturbed by dysphonia and dysphagia. Symptoms of right ventricular failure are observed in the form of edema in the legs, a feeling of heaviness under the right rib cage, an increase in the abdomen due to ascites, and a decrease in diuresis.

Examination: Patients with mild mitral stenosis are not particularly important. As the stenosis increases, pulmonary hypertension increases, the appearance of a pallor of the face, a bright red color of both cheeks with a cyanotic tinge, cyanosis of the lips and tip of the nose (facies mitralis), and in the stage of pronounced decompensation - acrocyanosis is observed. With the development of right ventricular failure, edema, up to anasarca, is observed. When examining the heart, we observe a shift of the apex impulse to the right and up. With an increase in the left ventricle, the apex impulse may not be felt, since the left ventricle is compressed by the hypertrophic right ventricle, but the apex of the heart and epigastric pulsation can be observed.

On palpation of the heart: Sometimes, especially when palpating the patient on the left side, a low-frequency diastolic murmur (‘‘cat’’) is detected at the apex of the heart, which is a palpatory sign of a diastolic murmur characteristic of mitral stenosis.

On percussion of the heart: It is determined that the border of the apex of the heart is displaced to the left (due to left ventricular hypertrophy), and upwards (due to the left atrium), the heart has a flattened ‘‘mitral’’ configuration.



ELECTROCARDIOGRAPHIC STUDY

The ECG reveals hypertrophy of the left atrium and right ventricle, heart rhythm disturbances (atrial fibrillation, extrasystole, paroxysmal tachycardia), and blockade of the branches of the right bundle.

EchoCG	With the help of echocardiography, a decrease in the area of the mitral orifice, thickening of the anterior and posterior leaflets of the mitral valve, and enlargement of the left atrium can be detected.
Radiography	Radiological studies (chest radiography, contrast heart radiography of the esophagus) are characterized by a dilated pulmonary artery arch, left atrial and right ventricular dilatation, mitral configuration of the heart, and enlargement of the abdominal cavity shadows.
Invasive diagnostics	When examining the heart chambers, an increase in pressure in the left atrium and right parts of the heart and an increase in the transmitral pressure gradient are detected. Left ventricular and atriography, as well as coronary angiography, are indicated for mitral valve replacement.

Mitral stenosis treatment

Drug therapy is necessary to prevent infective endocarditis (antibiotics), reduce the severity of heart failure (cardiac glycosides, diuretics) and eliminate arrhythmias (beta blockers). If there is a history of thromboembolism, subcutaneous heparin and antiplatelet agents are prescribed under APTT monitoring.

Surgical treatment is carried out in stages II, III, IV of hemodynamic disorders. In the absence of deformation, calcification or damage to the papillary muscles and chordae, balloon valvuloplasty can be performed. In other cases, closed or open commissurotomy is indicated, during which adhesions are separated, the mitral valve leaflets are cleaned of calcification, blood clots are removed from the left atrium, and annuloplasty is performed for mitral insufficiency.

Prognosis and prevention

The five-year survival rate for the natural course of mitral stenosis is 50%. Even a small asymptomatic defect is prone to progression due to repeated attacks of rheumatic carditis. The 5-year survival rate after surgery is 85-95%. Restenosis after surgery develops in approximately 30% of patients within 10 years, requiring mitral recommissurotomy.

Mitral stenosis prevention consists of prophylaxis against recurrence of rheumatism, sanitation of foci of chronic streptococcal infection. Patients should be monitored by a cardiologist and rheumatologist and regularly undergo a

complete clinical and instrumental examination to exclude the development of a decrease in the diameter of the mitral orifice.

CONCLUSION

The current surgical method for mitral valve stenosis is the installation of an artificial valve. In cases where there is no calcification of the mitral valve and the mobility of the leaflets is preserved, valve reconstruction is performed plastically. It is necessary for everyone to take complex congenital and acquired heart defects seriously, not to be indifferent to the symptoms of the disease that arise in them. The patient should immediately consult a doctor and take preventive measures.

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