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Mitral Stenosis

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Introduction

Mitral stenosis (MS) is a form of valvular heart disease. Mitral stenosis is characterized by narrowing of the mitral valve orifice.

Etiology

The most common cause of mitral stenosis is rheumatic fever. Uncommon causes of mitral stenosis are calcification of the mitral valve leaflets and congenital heart disease. Other causes of mitral stenosis include infective endocarditis, mitral annular calcification, endomyocardial fibroelastosis, malignant carcinoid syndrome, systemic lupus erythematosus, Whipple disease, Fabry disease, and rheumatoid arthritis.

Pathophysiology

The mitral valve is a tri-leaflet valve positioned between the left atrium and left ventricle. The normal mitral orifice area is 4 to 6 square centimeters. Under normal physiologic conditions, the mitral valve opens during left ventricular diastole to allow blood to flow from the left atrium to the left ventricle. The pressure in the left atrium and the left ventricle during diastole are equal. The left ventricle gets filled with blood during early ventricular diastole. There is only a small amount of blood that remains in the left atrium. With the contraction of the left atrium (the "atrial kick") during late ventricular diastole, this small amount of blood fills the left ventricle.

Mitral valve areas less than 2 square centimeters causes impediment of the blood flow from the left atrium into the left ventricle. This creates a pressure gradient across the mitral valve. As the gradient across the mitral valve increases, the left ventricle requires the atrial kick to fill with blood.

Mitral valve area less than 1 square centimeter causes an increase in left atrial pressure. The normal left ventricular diastolic pressure is 5 mmHg. A pressure gradient across the mitral valve of 20 mmHg due to severe mitral stenosis will cause a left atrial pressure of about 25 mmHg. This left atrial pressure is transmitted to the pulmonary vasculature resulting in pulmonary hypertension.

As left atrial pressure remains elevated, the left atrium will increase in size. As the left atrium increases in size, there is a greater chance of developing atrial fibrillation. If atrial fibrillation develops, the atrial kick is lost.

Thus, in severe mitral stenosis, the left ventricular filling is dependent on the atrial kick. With the loss of the atrial kick, there is a decrease in cardiac output and sudden development of congestive heart failure.

Mitral stenosis progresses slowly from initial signs of mitral stenosis to NYHA functional class II symptoms to atrial fibrillation to NYHA functional class III or IV symptoms.

History and Physical

Mitral stenosis presents 20 to 40 years after an episode of rheumatic fever. The most common symptoms are orthopnea and paroxysmal nocturnal dyspnea. Patients may have symptoms of palpitations, chest pain, hemoptysis, thromboembolism when the left atrial volume is increased, ascites, edema, and hepatomegaly (if right side heart failure develops).

There is also an increase in symptoms of fatigue and weakness with exercise and pregnancy.

On auscultation, the first heart sound is usually loud and may be palpable due to increased force in the closing of the mitral valve.

The P2 (pulmonic) component of the second heart sound (S2) will be loud if severe pulmonary hypertension is due to mitral stenosis.

An opening snap (OS) is an additional sound that may be heard after A2 component of the second heart sound (S2). This is the forceful opening of the mitral valve when the pressure in the left atrium is greater than the pressure in the left ventricle.

A mid-diastolic rumbling murmur with presystolic accentuation is heard after the opening snap. This murmur is a low pitch sound. It is best heard with the bell of the stethoscope at the apex. The murmur accentuates in the left lateral decubitus position and with isometric exercise.

Advanced mitral stenosis, presents with signs of right-sided heart failure (jugular venous distension, parasternal heave, hepatomegaly, ascites) and/or pulmonary hypertension.

Other signs include, atrial fibrillation, left parasternal heave (right ventricular hypertrophy due to pulmonary hypertension) and tapping the apical beat.

Evaluation

Mitral stenosis is evaluated using noninvasive and invasive measures. Noninvasive tests are the electrocardiogram (ECG), chest x-ray, echocardiogram, and exercise echocardiogram. An invasive test for mitral stenosis would include a cardiac catheterization.

On the ECG, the P wave changes suggest left atrial enlargement. A presence of right axis deviation and right ventricular hypertrophy suggest severe pulmonary hypertension. ECG frequently detects atrial arrhythmias such as atrial fibrillation.

On the chest x-ray, the early stages of mitral stenosis findings are normal heart size, straightening of the left border of the cardiac silhouette, prominent main pulmonary arteries, dilatation of the upper pulmonary veins, and displacement of the esophagus by an enlarged left atrium. During the severe chronic stage of mitral stenosis, the chest x-ray will have enlargement of all the chambers, pulmonary arteries, and pulmonary veins.

The echocardiogram is a very useful tool to assess the mitral stenosis etiology, morphology, severity, and treatment intervention. The analysis of the morphology of mitral valve apparatus includes leaflet mobility and flexibility, leaflet thickness, leaflet calcification, subvalvular fusion, and the appearance of commissures. The Wilkins score grades each of the components of the mitral apparatus from 1 to 4: leaflet mobility, thickness, calcification, and impairment of the subvalvular apparatus. The Padial score grades the leaflet thickening (each separately), the commissural calcification, and the subvalvular disease from 1 to 4. The Wilkins score less than 8, a Padial score less than 10, and less than moderate regurgitation have better outcomes.

An exercise echocardiogram is performed using an upright treadmill or supine bicycle with Doppler recording of transmitral and tricuspid valve velocities. This measures the transmitral gradient and pulmonary artery systolic pressure at rest and with exercise.

Cardiac catheterization is an invasive procedure. Cardiac catheterization should be performed for assessment of the severity of mitral stenosis when noninvasive tests are inconclusive or when there is a discrepancy between noninvasive tests and clinical findings regarding the severity of mitral stenosis (Class I, Level of Evidence C).

Classification of Severity of Mitral Valve Stenosis

Mild

- Mean gradient (mmHg) less than 5
- Pulmonary artery systolic pressure (mmHg) less than 30
- Valve area (cm2) less than 1.5

Moderate

- Mean gradient (mmHg) 5 to 10
- Pulmonary artery systolic pressure (mmHg) 30 to 50
- Valve area (cm2) 1.0 to 1.5

Severe

- Mean gradient (mmHg) less than 10
- Pulmonary artery systolic pressure (mmHg) greater than 50
- Valve area (cm2) less than 1.0

Mitral Valve Anatomy According to the Wilkins Score

Grade 1

- Mobility: Highly mobile valve with only leaflet tips restricted
- Thickening: Leaflet near normal in thickness (4 mm to 5 mm)
- Calcification: A single area of increased echo brightness
- Subvalvular Thickening: Minimal thickening just below the mitral leaflets

Grade 2

- Mobility: Leaflet mid to base portions have normal mobility
- Thickening: Mid leaflets normal, considerable thickening of margins (5-8 mm)
- Calcification: Scattered areas of brightness confirmed to leaflet margins
- Subvalvular Thickening: Thickening of chordal structures extending to one of the chordal length

Grade 3

- Mobility: Valve continues to move forward in diastole, mainly from the base
- Thickening: Thickening extending through the entire leaflet (5 mm to 8 mm)
- Calcification: Brightness extending into the mid portions of the leaflets
- Subvalvular Thickening: Thickening extended to distal third to the chords

Grade 4:

- Mobility: No or minimal forward movement of the leaflets in diastole
- Thickening: Considerable thickening of all leaflet tissue (more than 8 mm to 10 mm)
- Calcification: Extensive brightness throughout much of the leaflet tissue

- Subvalvular Thickeing: Extensive thickening and shortening of all chordal structures extending down to the papillary muscles.

Treatment / Management

Treatment for mitral stenosis involves medical therapy, percutaneous mitral valvuloplasty, and surgical therapy. Currently, no medical therapy can relieve a fixed obstruction of the mitral valve. Medical therapy is focused on preventing endocarditis, decreasing new cases of rheumatic fever, improving symptoms, and decreasing the thromboembolic risk.

Endocarditis prophylaxis should only be given to high-risk patients before dental procedures that involve manipulation of gingival tissue or perforation of the oral mucosa. High-risk patients are those patients with a prosthetic heart valve or prosthetic material used for valve repair, previous history of infective endocarditis, and cardiac valvuloplasty.

Rheumatic fever prophylaxis with Benzathine penicillin is the primary prevention treatment in patients with streptococcal pharyngitis.

If the rhythm is normal sinus, medical therapy is used to improve symptoms. Diuretics are utilized to help relieve congestion. Beta-blockers and/or calcium channel blockers help with exertional symptoms associated with elevated heart rate.

If the rhythm is atrial fibrillation, the first step is to control the rate using AV node blocking agents such as beta blockers, calcium channel blockers, and/or digitalis. In an unstable patient, perform direct current cardioversion. If you cannot convert atrial fibrillation to normal sinus rhythm, then the primary goal is rate control. In a stable patient, restoration of normal sinus rhythm is preferred over rate control to improve functional capacity and quality of life.

Anticoagulation prevents thromboembolic events. Anticoagulation is indicated in patients with mitral stenosis and atrial fibrillation (paroxysmal, persistent, or permanent), previous embolic events, and the presence of left atrial thrombus. At present, Warfarin is the anticoagulation of choice. Aspirin or other antiplatelet drugs are not approved to decrease thromboembolic risk in mitral stenosis. Warfarin should be monitored using international normalized ratio (INR) to target 2.5.

Percutaneous mitral balloon valvuloplasty (PMBV) is an invasive procedure used to manage mitral stenosis. PMBV improves symptoms by increasing mitral valve area and reduce mitral valve gradient. PMBV is indicated in symptomatic patients (New York Heart Association functional class greater than II), or asymptomatic patients with pulmonary hypertension with moderate or severe stenosis, and favorable valve morphology in the absence of left atrial thrombus, or moderate to severe mitral regurgitation.

Mitral valve replacement surgery is indicated in patients with symptomatic moderate or severe mitral stenosis when percutaneous mitral balloon valvuloplasty is contraindicated or unfavorable valve morphology (Class I, Level of Evidence B).

Pearls and Other Issues

Pregnancy during mitral stenosis will increase the patient's symptoms by one New York Heart Association class. Medical therapy is attempted first to improve symptoms. If symptoms do not improve with medical treatment, then refer the patient for percutaneous mitral balloon valvuloplasty.

Questions

To access free multiple choice questions on this topic, click here.

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