Mitral and tricuspid valve disease

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Abstract

Mitral stenosis (MS) has become less common in developed countries following the declining incidence of rheumatic fever, but it remains a major health problem worldwide. Atrial fibrillation is a common accompaniment and may trigger the onset of pulmonary oedema. Medical treatment is aimed at control of atrial fibrillation and prevention of thromboembolism. Patients with symptoms and a valve area <1.5 cm^2 should be considered for balloon valvuloplasty or surgical valve replacement. Mitral regurgitation (MR) is common and most often caused by degenerative disease of the valve, or secondary to underlying left ventricular dysfunction. Management depends upon the aetiology. Patients with severe degenerative MR should be offered valve repair before the onset of limiting symptoms or left ventricular dysfunction. Exercise testing can have a role in surveillance and decision-making. Surgery for ischaemic MR is less successful, but should be considered in patients with moderate or severe MR undergoing coronary artery bypass grafting, and in patients with severe MR and symptoms of breathlessness. Percutaneous and minimally invasive approaches are emerging as alternatives in some patients.

Keywords Ischaemic mitral regurgitation; mitral balloon valvuloplasty; mitral regurgitation; mitral stenosis; mitral valve repair; mitral valve replacement; rheumatic mitral valve disease; tricuspid regurgitation; tricuspid stenosis

Mitral stenosis

Aetiology

Mitral stenosis (MS) is almost always caused by chronic rheumatic heart disease, although 30% of patients have no clear history of rheumatic fever. Rheumatic MS is twice as common in women as in men. The incidence has fallen dramatically in developed countries but in Africa, the Middle East and Asia, symptomatic mitral valve disease may affect patients from teenage years upwards.[1,2] Other causes of mitral stenosis include congenital mitral stenosis and mitral annular calcification.

Pathophysiology

Rheumatic fever is triggered by oropharyngeal infection with Group A haemolytic streptococci. The infection induces an immune reaction that predominantly affects the left-sided heart valves. This is progressive and in time produces a thickened, fibrotic valve with fusion of the commissures, reduced leaflet mobility and a small orifice. The chords may also be matted together and the valve may ultimately become heavily calcified.

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Natural history

As the mitral valve narrows, a diastolic pressure gradient develops between the left atrium and left ventricle. Left atrial pressure rises, leading to a passive rise in pulmonary venous pressure. Any situation that increases heart rate or cardiac output, such as exercise, infection or pregnancy, will further increase the transmitral gradient and pulmonary venous pressure, producing breathlessness or even pulmonary oedema. Chronic pulmonary venous hypertension leads to secondary pulmonary arterial hypertension, right heart dilatation, tricuspid regurgitation and right heart failure.

There is usually a latent period of 20–40 years from rheumatic fever to the onset of symptoms. In developing countries, this delay is shorter, with symptoms often appearing before the age of 20. Once limiting symptoms develop, progression is more rapid and prognosis is poor.

Symptoms

Breathlessness and fatigue of gradual onset are often the only symptoms. Development of atrial fibrillation may precipitate acute pulmonary oedema. Some patients present with symptoms of right heart failure or with systemic thromboembolism. Haemoptysis resulting from severe pulmonary venous hypertension is less common.[1,2]

Physical examination

Table 1 outlines the physical findings in MS.

Investigations

Echocardiography is used in patients with suspected MS to estimate valve area and gradients. A valve area of 1.5–2.0 cm^2 indicates a mildly stenotic valve, 1.0–1.5 cm^2 moderate stenosis and <1.0 cm^2 severe stenosis. Echocardiography also provides important information about the morphology of the other valves, atrial and ventricular dimensions and function, and pulmonary arterial pressure (Figure 1).

A 2D (±3D) transoesophageal echocardiography (TOE) is required if intervention is contemplated.[1] It allows for a detailed anatomical assessment of leaflet mobility, thickness and calcification, subvalvular fusion, commissural fusion, and calcification.[3] These features are used to establish the feasibility of percutaneous balloon mitral valvuloplasty (PBMV).[4,5]

Exercise echocardiography can be helpful in the assessment of asymptomatic patients, or in patients with a disparity between the severity of symptoms and stenosis.[6,7]

ECG: atrial fibrillation is very common in MS. If sinus rhythm persists, a broad notched P-wave may be present.

Chest X-ray: appearances change with progression of MS but may show left atrial enlargement and evidence of pulmonary hypertension or right heart enlargement in advanced disease.

Physical examination in mitral stenosis (MS)

Inspection and palpation

Malar flush in chronic severe disease

Often atrial fibrillation

Apex beat tapping (palpable first heart sound)

- No displacement of apex in isolated MS

Parasternal heave if pulmonary hypertension present

Systolic waves in jugular venous pressure

- When tricuspid regurgitation present

Auscultation

Loud S1

- Indicates a pliable valve
- Not with severely calcified immobile valves

Loud pulmonary component of the second sound

- If pulmonary pressure raised

Opening snap (OS)

- After S2
- Indicates a pliable valve
- The tighter the stenosis, the closer the OS to S2

Diastolic murmur

- Starts after the OS (if present)
- The longer the murmur, the tighter the MS
- Best heard at apex with patient tilted to the left

Systolic murmur

- With accompanying mitral regurgitation and/or tricuspid regurgitation

Table 1

Cardiac catheterization: now rarely needed except to evaluate the presence or absence of coronary artery disease in candidates for valve surgery.

Management

Asymptomatic patients with MS can be reviewed periodically with repeat echocardiography. They should be advised to seek medical attention if symptoms develop in the interim.

Anticoagulation: all patients with atrial fibrillation should be anticoagulated unless there is a clear contraindication. The European Society of Cardiology recommends anticoagulation in sinus rhythm if the left atrium is enlarged (>5 cm), or if there is dense spontaneous contrast on TOE or a history of thromboembolism (class IIa, C).

Control of atrial fibrillation: inappropriate tachycardia shortens diastolic filling time and may aggravate symptoms. Atrial fibrillation of recent onset in patients with mild-to-moderate MS may respond to DC cardioversion. Otherwise, optimal control of the heart rate during activity is the priority, using β-adrenoceptor blockers or rate-limiting calcium channel blockers, which are usually more effective than digoxin.[2]

Symptomatic treatment: breathlessness or other symptoms of congestion may be relieved with diuretic therapy and long-acting nitrates.

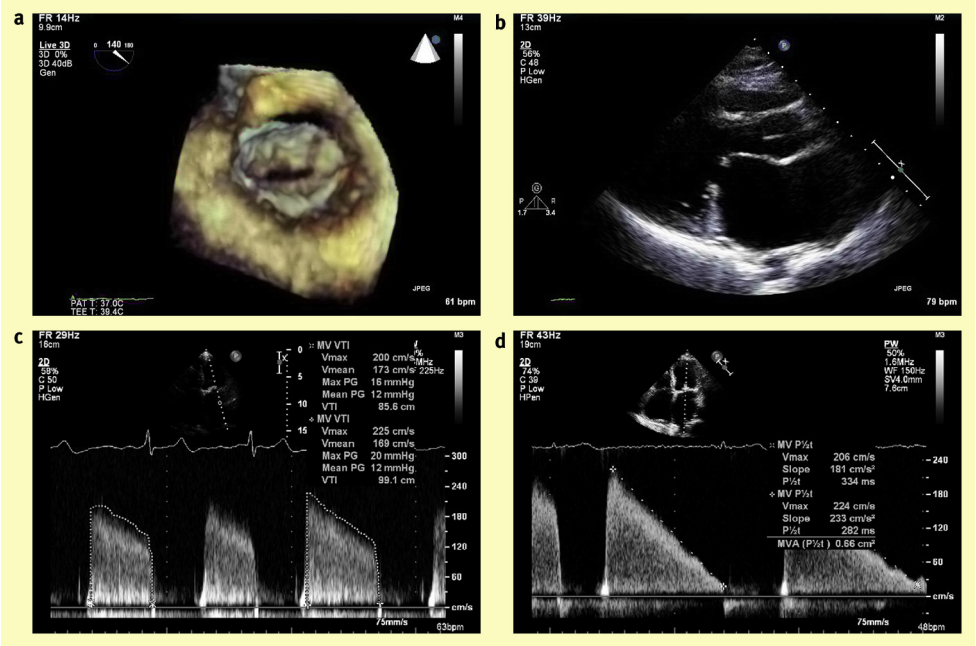

Figure 1 The echocardiographic assessment of mitral stenosis. (**a**) A 3D TOE image of the mitral valve from which valve area can be assessed by planimetry. (**b**) A transthoracic parasternal long axis view showing characteristic thickening of the mitral valve, particularly at the leaflet tips, and the 'hockey stick' appearance of the anterior mitral valve leaflet. (**c**) A continuous wave Doppler trace through the mitral valve. Mean gradient through the valve is 12 mmHg, which is consistent with severe mitral stenosis. (**d**) Pulsed-wave Doppler at the leaflet tips for pressure half-time (PHT) assessment. PHT is a measure of the rate of fall in pressure across the valve — the slower the rate of change, the more severe the stenosis. The relationship between mitral valve area and pressure half time is linear. A mitral valve area of 1 cm^2 has an approximate PHT of 220 ms. A pressure half time of 334 ms, as above, has an estimated valve area of 220 ÷ 334, or 0.66 cm^2.