

Mitral valve disease

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- Mitral regurgitation Any pathological process affecting the mitral valve apparatus may lead to mitral regurgitation. As such, there are many causes of regurgitation and they can be broadly classified into four headings. They are shown in Table 59.4 . Pathophysiology There is an important distinction between acute and chronic mitral regurgitation. The former is usually the result of

Prosthetic valve Streptococcus spp. Aggregatibacter spp., Cardio spp.) Coagulase-negative Staphylococcus spp. Staphylococcus aureus Enterococci Candida Non-tuberculous mycobacteria

ischaemic papillary muscle rupture or following infective endocarditis, whereas the latter is the result of longstanding myxomatous degeneration or fibroelastic changes in the leaflets. In acute mitral regurgitation, the left ventricle ejects blood back into a small, poorly compliant left atrium, imposing a sudden volume load on the left atrium during ventricular systole. This leads to an abrupt rise in left atrial pressure followed a rise in pulmonary venous pressure and pulmonary oedema. Chronic mitral regurgitation progresses slowly , allowing compensatory left ventricular dilatation and hypertrophy , and atrial dilatation without significant increase in pressure, protecting the pulmonary circulation. As the disease advances left atrial pressure begins to rise, leading to a rise in pulmonary venous pressure and progressive pulmonary congestion, with eventual congestive cardiac failure. Clinical features In acute mitral regurgitation, the patient is usually unwell, presenting with clinical and radiological evidence of acute ↑ John Brereton Barlow , 1924–2008, South African cardiologist. Bernard Jean Antonin Marfan , 1858–1942, physician, L'Hôpital des Enfants-Malades, Paris, France, described this syndrome in 1896. Edward Ehlers , 1863–1937, Professor of Clinical Dermatology , Copenhagen, Denmark. Henri Alexandre Danlos , 1844–1912, dermatologist, Hôpital St Louis, Paris, France. Christian Johann Doppler , 1803–1853, Professor of Experimental Physics, Vienna, Austria, enunciated the 'Doppler principle' in 1842. pulmonary oedema and a loud apical pansystolic murmur. Patients with mild chronic mitral regurgitation are usually asymptomatic. With progressive pulmonary congestion and left ventricular failure, the patient develops fatigue, exertional - dyspnoea and orthopnoea. The development of AF with left atrial dilatation is common. The enlarged left ventricle leads to by a heaving apical impulse and a pansystolic murmur. Investigations /uni25CF ECG : may show left atrial hypertrophy (bifid P waves, - known as 'P mitrale'), left ventricular hypertrophy and AF . /uni25CF Chest radiography : there may be cardiomegaly with prominent pulmonary vasculature. /uni25CF Echocardiography : this is often combined with colour flow Doppler imaging, which shows the severity of the regurgitant jet of mitral regurgitation. /uni25CF Coronary angiography : in patients >40 years of age to investigate the coronary arteries. /uni25CF Cardiac MRI : increasingly popular as it can give detailed information on structure and function. Indications for surgery Indications for surgery in patients with primary mitral regurgitation include severe symptoms or associated changes in left ventricular function or dimension (e.g. left ventricular end-systolic

diameter). Evidence suggests that changes in this setting are usually associated with significant mortality if not corrected (Figure 59.13). It is also recommended to treat severe mitral disease if a patient is undergoing cardiac surgery for a different reason. Surgical treatment of primary mitral regurgitation usually involves valve repair. When repair is not feasible, valve replacement with attempts to preserve the subvalvular apparatus should be considered. The treatment of ischaemic mitral regurgitation remains controversial and current evidence suggests that patients with

Degenerative causes Ventricular causes Barlow's disease (myxomatous degeneration) dynamic regurgitation Calcification of the leaflets or annulus Myocardial infarction resulting in papillary muscle rupture Marfan/Ehlers-Danlos syndromes Cardiomyopathy and annular and other connective tissue disorders dilatation 120/80 mmHg + + + 120/ + mmHg Figure 59.13 Features and pathophysiology of mitral regurgitation. There is a loud parasystolic murmur and the left atrium enlarges. The left ventricle enlarges as a consequence of volume overload. Autoimmune and infective Other causes causes Infective endocarditis Trauma (rarely) Rheumatic fever (post- Congenital defects such as streptococcal throat infection) isolated mitral cleft Associated with certain medications (those containing ergotamine) Radiotherapy

replacement, while patients with moderate regurgitation should usually undergo repair along with CABG if indicated. Mitral stenosis The most common cause of mitral stenosis worldwide remains rheumatic fever, despite the fact that the incidence of overt rheumatic fever in resource-rich countries has decreased. During the healing phase of acute rheumatic fever, the valve leaflets become adherent to each other at their free border so that the commissures become obliterated, narrowing the valve orifice. Symptoms of mitral stenosis usually develop more than 10 years after the acute attack. Pathophysiology Mitral stenosis slows diastolic ventricular filling and left atrial pressure rises to maintain cardiac output. This leads to atrial hypertrophy and dilatation. Pulmonary congestion results from the rise in left atrial pressure with time. Although the lungs are protected against pulmonary oedema by constriction of the pulmonary vessels, this adaptive response, along with the passive 'back pressure' generated by the rise in left atrial pressure, leads to pulmonary hypertension (>25 mmHg). This leads to an increased demand on the right ventricle with eventual right heart failure and tricuspid regurgitation. The development of AF is common and can lead to a significant reduction in cardiac output. AF predisposes to thrombiforming in the left atrium, which may embolise to the systemic circulation. Summary box 59.9 Causes of mitral valve disease /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Clinical features Some patients may remain asymptomatic for years and then present with symptoms when the heart is stressed by events such as pregnancy , fever, chest infection or with the onset of AF . The common symptoms are fatigue and dyspnoea on exertion, which result from the combination of reduced forward flow and increased back pressure. The resulting pulmonary congestion adds to breathlessness and may produce a cough or haemoptysis. In severe mitral stenosis, there may also be a right ventricular heave due to right ventricular hypertrophy in response to pulmonary hypertension. Auscultation may reveal an opening - snap soon after the second heart sound, as the diseased valve is opened forcibly by the high pressure in the left atrium. The reverse happens when the valve closes and there is a loud 'tap - ping' first heart sound. In addition, a rumbling mid-diastolic murmur can be heard. The duration of the murmur is related to the severity of the mitral stenosis, increasing in length as the stenosis becomes more severe. Investigations /uni25CF ECG may show left atrial enlargement or AF , right axis deviation or

other signs of right ventricular hypertrophy (tall QRS complexes in the right ventricular leads V1-3).
Chest radiography : there is a small aortic outline and a prominent pulmonary artery . The left atrium is enlarged (sometimes to an enormous degree) along with upper lobe diversion as a result of the raised pulmonary venous pressure. The right ventricle also appears enlarged (Figure 59.14).
Echocardiography , in combination with colour flow Doppler imaging, allows assessment of the flow across the valve and, therefore, the degree of stenosis. Transoesophageal echocardiography (TOE) may be better at assessing valve morphology and excluding the presence of an atrial thrombus.
Coronary angiography : to investigate the coronary arteries. -
Cardiac MRI .
Right heart catheterisation . Indications for surgery Medical management includes anticoagulation in patients - with AF or left atrial enlargement. Tachyarrhythmias should

Stenosis Rheumatic heart disease (common) Calcification of valve or chordae tendinae
Congenital (rare) Regurgitation Rheumatic heart disease Valve prolapse Left ventricular dilatation or hypertrophy Ischaemia Bacterial endocarditis Figure 59.14 Chest radiograph of longstanding mitral stenosis, showing a massive left atrium.

be treated using pharmacological agents such as digoxin to avoid decompensation and cardiac failure. Diuretics may also provide some benefit. The first-line invasive intervention is balloon valvuloplasty (PMBV); surgery is indicated for severely symptomatic patients who are unsuitable for PMBV or in whom PMBV failed. The prognosis is determined by the severity of the stenosis, the size of the atrium, the presence of AF and rising pulmonary artery pressure (Figure 59.15 Surgical options include mitral valve repair or mitral valve replacement. Formerly common surgical procedures such as closed or open commissurotomy are now rarely performed. Mitral valve operations Approaches to the mitral valve vary; commonly a median sternotomy or, occasionally , right thoracotomy is performed ('mini'-mitral surgery). The valve can be approached directly through the left atrium in the interatrial groove, through the right atrium and then the interatrial septum or through the left atrial appendage. Mitral valve repair Restoration of normal valve function and preservation of the mitral apparatus is preferable to replacement in specific groups of patients, as it can be associated with improved long-term ventricular remodelling and function. This approach reduces the bleeding complications associated with anticoagulants. The functional classification system developed by Carpentier serves as a guideline in valve reconstruction. It classifies mitral insufficiency into one of three groups according to the amplitude of the leaflet motion and provides a useful framework for the mechanisms of failure of the mitral valve. As a rule, several valvular lesions or abnormalities are involved in a functional abnormality , with specific techniques developed to correct each lesion. At surgery , the anatomy of the valvular apparatus and sub valvular structures is carefully inspected. The extent of annular dilatation, leaflet prolapse and chordal dysfunction is assessed. Repair should respect rather than resect tissues, restoring a good coaptation surface between the two leaflets. The mitral valve repair can employ various techniques, including insertion of a prosthetic ring annuloplasty (Figure 59.16); triangular or quadrangular resection of the leaflet; use of a sliding plasty; chordal shortening; chordal transposition; and neochordea implantation. Many techniques exist, indicating that no one technique) addresses all possible findings in mitral regurgitation. Valve repair offers better preservation of ventricular function and avoids prolonged anticoagulation, and valve-related complications such as PVE or structural dysfunction. Recent advances in surgical techniques and

the development of different types of rings has led to increased use of mitral valve repair with excellent results, making it the standard operation. The operative mortality is 1–3%. One of the major issues related to mitral repair is the incidence of regurgitation recurrence, which varies between series but can be up to 30% at 5 years. This is related to which leaflet is repaired and the amount of foreign material used in the repair (patch). Mitral valve replacement When valve repair is not feasible, mitral valve replacement is necessary. This usually involves a median sternotomy and access to the left atrium on CPB. The diseased valve is excised and a suitably sized mechanical or bioprosthetic valve is implanted. The atriotomy is closed following de-airing of the left heart. Intraoperative TOE can be used to assess adequate valve function. - The mortality rate for elective mitral valve replacement may be up to 5%, depending largely on the state of the myocardium and the general condition of the patient. Common serious in-hospital complications include stroke (<3%) and renal or patients following failure (3%). The longer term prognosis of mitral valve replacement is generally good in comparison with the natural history of mitral valve disease. Indeed, more recent evidence suggests that patients with ischaemic severe mitral regurgitation can benefit more from valve replacement than from repair.

120/0–5 mmHg Enlarged 20+ left atrium mmHg Thrombus Figure 59.15 Pathophysiology of mitral stenosis. The aorta and left ventricle are relatively small because of chronically reduced cardiac output. The atrium is enlarged and may fibrillate, become stagnant and contain a thrombus. The ventricle fibrillates with a turbulent jet that may be detected as a diastolic murmur or a thrill at the apex. Figure 59.16 Operative view of the mitral valve repair using a Carpentier–Edwards annuloplasty ring (courtesy of A Murday, FRCS).

® The MitraClip is a device used to reduce mitral valve regurgitation. The method involves suturing of the leaflets of the mitral valve together so that regurgitation into the left atrium is prevented. The valve continues to open through the sides of the suture and therefore blood continues to flow into the left ventricle. Access is usually from the groin where a catheter is inserted in the femoral vein to the right atrium. The left atrium is accessed by making a septal puncture. Although this method is less invasive and associated with rapid recovery and reduced in-hospital stay, it is however technically demanding and long term durability of the results of the device is unknown. ® Data suggest that the MitraClip may be suitable for a small subset of high-risk patients (e.g. chronic heart failure), but the vast majority are better served by surgery that leaves them with substantially less mitral regurgitation.

Revision #1

Created 2025-12-31 15:22:12 UTC by Omar Ayman

Updated 2025-12-31 15:22:12 UTC by Omar Ayman