



Valvular Heart Disease : Diagnosis and Management

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Abstract

Valvular heart disease is a leading cause of morbidity and mortality in India. Advances in both surgical and percutaneous techniques and a better understanding of timing for intervention accounts for the current increased rates of survival. Echocardiography remains the gold standard for diagnosis and periodic assessment of patients with valvular heart disease. Generally, patients with stenotic valvular lesions can be monitored clinically until symptoms appear and most can now benefit from percutaneous techniques. In contrast, patients with regurgitant valvular lesions require careful echocardiographic monitoring for left ventricular function and may require surgery even if no symptoms are present. Percutaneous therapy of valvular regurgitant lesions is yet to evolve fully. ©

INTRODUCTION

The developed countries witnessed a dramatic decline in the incidence of rheumatic fever (RF) and the prevalence of rheumatic heart disease (RHD) in the 20th century; however, this has continued to be major health care concern in the developing countries. In India, the prevalence of RF/RHD among school children is 2-11 per 1000 with a mean of 6 per 1000.¹ Recently, a study conducted by Jacob Jose in 2002 has shown the prevalence to be 0.68 per 1000 children.² The adult average ranges between 123 and 200 per 100,000 population,³ when compared to industrialized nations the incidence of RF is 0.5/100,000 population and prevalence less than 0.05/1000.⁴

Diagnosis of valvular heart disease

Cardiac auscultation remains the most widely used method of screening for valvular heart disease.⁵ In instances like valvular aortic stenosis detection of a cardiac murmur gives a clue to the detection of a cardiac disease that may be important even when asymptomatic or that may define the reason for cardiac symptoms whereas, a diastolic murmur virtually always represent pathological conditions and require further cardiac evaluation, as do most continuous murmurs.

ECG is important in providing negative information at a low cost like the absence of ventricular hypertrophy, atrial enlargement, arrhythmias, conduction abnormalities, prior myocardial infarction, and evidence of active ischemia.

Non-invasive investigation like an echocardiography with color flow and spectral Doppler evaluation is a very important tool for assessing the significance of cardiac

murmurs. Information regarding valve morphology and function, chamber size, wall thickness, ventricular function, pulmonary and hepatic vein flow, and estimates of pulmonary artery pressures can be readily integrated with the help of an echo.

Invasive test with a cardiac catheterization may be required in assessing the presence and severity of a lesion, hemodynamic assessment and in some patients in whom there is a discrepancy between the echocardiographic and clinical findings.

SPECIFIC VALVE LESIONS

Mitral stenosis

Approximately 25% of all patients with rheumatic heart disease have pure MS, and an additional 40% have combined MS and mitral regurgitation (MR).⁶⁻⁸ Congenital malformation of the MV occurs rarely and is observed mainly in infants and children. Rare causes of acquired causes of MV obstruction other than rheumatic heart disease include left atrial myxoma, ball-valve thrombus, mucopolysaccharidosis, and severe mitral annular calcification. The pathological process by which the rheumatic fever causes MS includes leaflet thickening and calcification, commissural fusion, chordal fusion, or a combination of these processes.^{9,10}

The normal MV area is 4.0 to 5.0 cm². When the orifice is reduced to 2 cm², blood flow occurs from LA to LV only if propelled by a pressure gradient. This results in elevation of left atrial pressure, which is reflected back into the pulmonary venous circulation, increases pulmonary venous pressure and leads to distension of the pulmonary veins and capillaries.^{11,12}

Pulmonary hypertension in MS results from

- (1) elevated left atrial pressure resulting in passive backward transmission into the pulmonary vasculature.

ature

- (2) reactive pulmonary hypertension due to pulmonary arteriolar constriction
- (3) organic obliterative changes in the pulmonary vascular bed due to longstanding and severe MS.

Subsequently, over a period of time, severe pulmonary hypertension results in right-sided heart failure, with dilatation of the right ventricle and its annulus and secondary tricuspid and sometimes pulmonic regurgitation. Reduction in pulmonary compliance results in dyspnoea and also there is a redistribution of pulmonary blood flow from the base to apex of the lungs.

Hemoptysis results in patients with MS due to (1) pulmonary apoplexy-resulting from the rupture of thin-walled, dilated bronchial veins (2) attacks of paroxysmal nocturnal dyspnoea (3) acute pulmonary oedema with rupture of alveolar capillaries (4) Pulmonary infarction, a late complication of MS associated with heart failure (5) Chronic bronchitis resulting from oedematous bronchial mucosa

Mitral stenosis shows a slow and stable course in the early years followed by a progressive acceleration later in life.¹³⁻¹⁶ In US and Western Europe, patients who develop acute rheumatic fever have an asymptomatic period of approximately 15 to 20 years before symptoms of MS develop. It then takes approximately 5 to 10 years for most patients to progress from mild disability (NYHA class II) to severe disability (NYHA class III-IV). In India, critical MS may be found in children as young as 6 to 12 years old. In the asymptomatic or minimally symptomatic patient, survival is greater than 80% at 10 years, with 60% of patients having no progression of symptoms.¹⁴⁻¹⁶ However, once significant limiting symptoms occur, there is a dismal 0% to 15% 10-year survival rate (14-17). Once there is severe pulmonary hypertension, mean survival drops to less than 3 years.¹⁸ The mortality of untreated patients with MS is due to progressive pulmonary and systemic congestion in 60% to 70%, systemic embolism in 20% to 30%, and pulmonary embolism in 10%, and infection in 1% to 5%.^{9,15} Serial hemodynamic and Doppler-echocardiographic studies have reported annual loss of MV area ranging from 0.09 to 0.32 cm².^{19,20}

Severity of mitral stenosis: echocardiographic assessment

Severity	Valve area	Mean gradient	PASP (mm of Hg)
Mild	Greater than 1.5 cm ²	Less than 5 mm Hg	Less than 30 mm Hg
Moderate	1.0 to 1.5 cm ²	5 to 10 mm Hg	30 to 50 mm Hg
Severe	Less than 1.0 cm ²	Greater than 10 mm Hg	Greater than 50 mm Hg

Suitability for BMV can be assessed by Echo using a

Wilkin's score, which takes into account 4 parameters, which includes the leaflet thickness, mobility, calcification and the morphology of subvalvular apparatus. Each of these parameters has 4 grades. Echo score of less than 8 out of 16 has been associated with a favourable outcome after BMV.^{20a}

In patients with symptomatic MS NYHA class II, with a MVA >1.5cm² who do not manifest elevation in either pulmonary artery, pulmonary artery wedge, or transmitral pressures coincident with development of exertional symptoms most likely would not benefit from intervention on the MV. In patients with NYHA class III/IV with mild MS, on exercise, if there is no increase in MV gradient of >15 mm Hg, or PAWP of not more than 25 mm HG, or increase in PASP of not more than 60 mm HG, other causes of symptoms should be looked for.

INDICATIONS FOR BMV

BMV is indicated in moderate or severe MS patients without the presence of left atrial thrombus or moderate to severe MR who are:

- (1) symptomatic (NYHA functional class II, III, or IV) with valve morphology favorable for PBMV
- (2) asymptomatic who have pulmonary hypertension (pulmonary artery systolic pressure greater than 50 mm Hg at rest or greater than 60 mm Hg with exercise)
- (3) symptomatic patients who are either not candidates for surgery or are at high risk for surgery.

The **benefit of BMV is less well established** in patients with moderate to severe MS who are

- (1) asymptomatic with new onset of atrial fibrillation
- (2) symptomatic patients with MV area greater than 1.5 cm² if there is evidence of hemodynamically significant MS based on pulmonary artery systolic pressure greater than 60 mm Hg, pulmonary artery wedge pressure of 25 mm Hg or more, or mean MV gradient greater than 15 mm Hg during exercise.
- (3) As an alternative to surgery for who have a nonpliable calcified valve and are in NYHA class III-IV

The immediate results of percutaneous mitral valvotomy are similar to those of mitral commissurotomy.²²⁻²⁵ Follow up of patients showed an event-free survival (freedom from death, repeat valvotomy, or MV replacement) overall is 50% to 65% over 3 to 7 years, with an event-free survival of 80% to 90% in patients with favorable MV morphology.^{24,26,27} When comparing patients undergoing BMV with surgery, there was no significant difference in acute hemodynamic results or complication rate, and early follow-up data indicate no difference in hemodynamics, clinical improvement, or exercise time. However, longer-term follow-up studies at 3 to 7 years.^{28,29} indicate more favorable hemodynamic

and symptomatic results with percutaneous balloon valvotomy than with closed commissurotomy. Of the 2 studies that compared percutaneous balloon valvotomy with open commissurotomy, one reported equivalent results²⁹ and the other showed more favorable results with open commissurotomy.³⁰

Indications for mitral valve surgery in symptomatic patients with moderate or severe MS when

- (1) percutaneous mitral balloon valvotomy is unavailable,
- (2) percutaneous mitral balloon valvotomy is contraindicated because of left atrial thrombus despite anticoagulation or because concomitant moderate to severe MR is present, or
- (3) the valve morphology is not favorable for percutaneous mitral balloon valvotomy in a patient with acceptable operative risk.
- (4) Severe pulmonary hypertension (pulmonary artery systolic pressure greater than 60) with NYHA functional class I–II symptoms who are not considered candidates for percutaneous mitral balloon valvotomy or surgical MV repair

ATRIAL FIBRILLATION IN MS

30 to 40% of patients with MS develop atrial fibrillation (AF).^{13,14} Atrial fibrillation occurs more commonly in older patients¹³ and is associated with a poorer prognosis, with a 10-year survival rate of 25% compared with 46% in patients who remain in sinus rhythm.¹⁵ Development of AF correlates independently with the severity of MS and the height of left atrial pressure.³¹ The risk of arterial embolization, especially stroke, is significantly increased in patients with atrial fibrillation.^{13,14,32-34} Systemic embolization may occur in 10% to 20% of patients with MS.^{13,14,32} One third of embolic events occur within 1 month of the onset of atrial fibrillation, and two thirds occur within 1 year. The frequency of embolic events does not seem to be related to the severity of MS, cardiac output, size of the left atrium, or even the presence or absence of heart failure symptoms. It correlates with the age of the patient and size of left atrial appendage.^{14,33,35} An embolic event may thus be the initial manifestation of MS.⁵² In patients who have experienced an embolic event, the frequency of recurrence is as high as 15 to 40 events per 100 patient-months.^{32-34,36}

Acute management consists of intravenous heparin and control of heart rate with digoxin, calcium channel blockers and beta-blockers. Intravenous or oral amiodarone can also be used when beta-blockers or heart rate-regulating calcium channel blockers cannot be used. Electrical cardioversion should be undertaken urgently, with intravenous heparin before, during, and after the procedure, if there is hemodynamic instability. If the decision has been made to proceed with elective cardioversion in a patient who has had documented

atrial fibrillation for longer than 24 to 48 h and who has not been on long-term anticoagulation, either the patient can be put on warfarin for more than 3 weeks, followed by elective cardioversion³⁷ or to cardiovert after ruling out a left atrial thrombus on trans-oesophageal echo along with intravenous heparin before, during and after the procedure.³⁸ It is important to continue long-term anticoagulation after cardioversion.

Class IC and amiodarone helps in maintaining the patients of paroxysmal atrial fibrillation in sinus rhythm. In patients who are resistant to conversion to sinus rhythm may be maintained on a rate controlling drugs like digoxin, beta-blockers or calcium channel blockers. If there is no contraindication to anticoagulation these patients on chronic atrial fibrillation should be put on anticoagulants to prevent embolic events.

MITRAL REGURGITATION

The common causes of organic MR include MVP syndrome, rheumatic heart disease, CAD, infective endocarditis, certain drugs, and collagen vascular disease. MR may also occur secondary to a dilated annulus from dilatation of the left ventricle. In some cases, such as ruptured chordae tendineae, ruptured papillary muscle, or infective endocarditis, MR may be acute and severe.

Mitral valve prolapse refers to the valve prolapse of 2 mm or more above the mitral annulus into the left atrium with or without MR and with or without mitral valve thickening (5mm or greater).³⁹ The prevalence of this entity is 1% to 2.5% of the population.⁴⁰ The basic microscopic feature of primary MVP is marked proliferation of the spongiosa, the delicate myxomatous connective tissue between the atrialis (a thick layer of collagen and elastic tissue that forms the atrial aspect of the leaflet) and the fibrosa or ventricularis (dense layer of collagen that forms the basic support of the leaflet).

In patients with acute severe MR, a sudden volume overload is imposed on the left atrium and left ventricle which increases LV preload, allowing for a modest increase in total LV stroke volume.⁴¹ However, forward stroke volume and cardiac output are reduced, as the LV has no time to develop eccentric hypertrophy, which is a compensatory mechanism to overcome the volume overload. This is reflected back into the pulmonary venous system resulting in pulmonary congestion. In this phase of the disease, the patient has both reduced forward output (even shock) and simultaneous pulmonary congestion. In severe MR, the hemodynamic overload often cannot be tolerated, and MV repair or replacement must often be performed urgently.

CHRONIC MITRAL REGURGITATION

Patients with mild to moderate MR may remain asymptomatic with little or no hemodynamic compromise for many years. Numerous studies indicate

that patients with chronic severe MR have a high likelihood of developing symptoms or LV dysfunction over the course of 6 to 10 years.⁴²⁻⁴⁵ The mortality rate in patients with severe MR caused by flail leaflets is 6% to 7% per year. Munoz and colleagues found that medically treated patients with severe MR had a 5-year survival of 45 percent and 30 percent surviving 10 years after the diagnosis,⁴⁶ whereas Horstkotte and associates reported a 5-year survival of only 30 percent in patients who were candidates for but who declined operation.⁴⁷ At 10 years follow-up, Ling et al found that heart failure occurred in 63%, atrial fibrillation in 30% of patients and 90% of patients are dead or require MV operation.⁴² However, patients at risk of death are predominantly those with LV ejection fractions less than 0.60 or with NYHA functional class III-IV symptoms, and less so those who are asymptomatic and have normal LV function.^{42,48} Severe symptoms also predict a poor outcome after MV repair or replacement.⁴⁸

Severe MR develops slowly in patients with MVP after a long delay and is rare before the age of 50 years. It occurs in about 15 percent of patients over a 10-15 year period and more so in men. Serious complications including cardiac death need for cardiac surgery, acute infective endocarditis, or cerebral events were seen in 1 per 100 patient years.^{49,50}

MV operations are of three types: (1) MV repair; 2) MV replacement with preservation of part or all of the mitral apparatus; and 3) MV replacement with removal of the mitral apparatus. MV repair is considered when the valve is suitable for repair. This procedure preserves the patient's native valve without prosthesis and therefore avoids the risk of chronic anticoagulation. The reconstructive procedure consists of annuloplasty (use of rigid-Carpentier or a flexible prosthetic-Duran ring); resection and placcation of the mitral annulus; elongating, shortening or re implanting of chordae tendinae; splitting of papillary muscles and repairing the subvalvular apparatus.

Indications for MV Surgery²¹

- (1) symptomatic patient with acute severe MR
- (2) symptomatic patients with chronic severe MR in the absence of severe LV dysfunction (severe LV dysfunction is defined as ejection fraction less than 0.30) and/or end-systolic dimension greater than 55 mm.
- (3) Asymptomatic patients with chronic severe MR and mild to moderate LV dysfunction, ejection fraction 0.30 to 0.60, and/or end-systolic dimension greater than or equal to 40 mm
- (4) asymptomatic patients with chronic severe MR, preserved LV function, and new onset of atrial fibrillation
- (5) asymptomatic patients with chronic severe MR, preserved LV function, and pulmonary hypertension

(pulmonary artery systolic pressure greater than 50 mm Hg at rest or greater than 60 mm Hg with exercise)

- (6) chronic severe MR due to a primary abnormality of the mitral apparatus and NYHA functional class III-IV symptoms and severe LV dysfunction (ejection fraction less than 0.30 and/or end-systolic dimension greater than 55 mm) in whom MV repair is highly likely.

Aortic stenosis

The most common cause of AS in adults is calcification of a normal trileaflet or congenital bicuspid valve.^{51,52} Rheumatic AS due to fusion of the commissures with scarring and eventual calcification of the cusps is less common and is invariably accompanied by MV disease.

Aortic stenosis is said to be severe if the valve area is less than 1.0 cm², mean gradient greater than 40 mm Hg, or jet velocity greater than 4.0 m per second. Once even moderate stenosis is present (jet velocity greater than 3.0 m per second), the average rate of progression is an increase in jet velocity of 0.3 m per second per year, an increase in mean pressure gradient of 7 mm Hg per year, and a decrease in valve area of 0.1 cm² per year.⁵³⁻⁵⁵ However, there is marked individual variability in the rate of hemodynamic progression. Eventually, symptoms of angina, syncope, or heart failure develop after a long latent period, and after the onset of symptoms, average survival is approximately 2 years in patients with heart failure, 3 years in those with syncope, and 5 years in those with angina,⁵⁶⁻⁵⁸ with a high risk of sudden death.

Asymptomatic patients with severe AS behave like normal adults of the same age group. In a prospective study of 123 asymptomatic adults with an initial jet velocity of at least 2.6 m per second, the rate of symptom development was 38% at 3 years for the total group.⁵⁹ A study of 622 asymptomatic hemodynamically significant patients on follow up showed the event free survival at 1,3 and 5 years were 82%, 67% and 33% respectively.⁶⁰

In patients with severe AS and low cardiac output may often present with a low mean pressure gradient of less than 30 mm Hg. Such patients can be difficult to distinguish from those with low cardiac output and only mild to moderate AS. Severe AS contributes to an elevated afterload, decreased ejection fraction, and low stroke volume, while primary contractile dysfunction is responsible for the decreased ejection fraction and low stroke volume. Low dose Dobutamine stress echo is indicated in these patients to see if the dobutamine infusion produces an increment in stroke volume and an increase in valve area greater than 0.2 cm² and little change in gradient. If so, it is likely that baseline evaluation overestimated the severity of stenosis.

Indications of Aortic valve replacement (AVR)

AVR is indicated in patients with

- (1) Symptomatic severe AS
- (2) asymptomatic severe AS undergoing coronary artery bypass surgery (CABG) or surgery on aorta or other heart valves
- (3) asymptomatic severe AS with LV systolic dysfunction
- (4) asymptomatic moderate AS if they are undergoing CABG or surgery on aorta or other valves.

Balloon aortic valvuloplasty (BAV)

BAV represents an attractive alternative to aortic valvotomy in children, adolescents, and young adults with congenital noncalcific AS,⁶¹ but its value is limited in adults with calcific AS. The major disadvantages of BAV in adults are due to serious acute complications occur with a frequency greater than 10%⁶²⁻⁶⁴ restenosis which occurs in about 50% of patients within 6 months^{65,66} and clinical deterioration occur within 6 to 12 months in most patients.^{67,68} Therefore, in adults with AS, balloon valvotomy is not a substitute for AVR. In spite of its disappointing intermediate term results, the procedure does have a role in patients who are not candidates for surgery.

Indications of BAV

Includes critical AS patients with

- (1) Cardiogenic shock⁶⁹
- (2) Who require a non surgical operation
- (3) Severe heart failure who are at extremely high operative risk as a "bridge" to AVR
- (4) Pregnant women⁷⁰
- (5) Severe comorbid conditions that preclude surgery
- (6) Who refuse surgical treatment.

AORTIC REGURGITATION

The causes of AR include idiopathic dilatation of the aorta, congenital abnormalities of the aortic valve (most notably bicuspid valves), calcific degeneration, rheumatic disease, infective endocarditis, systemic hypertension, myxomatous degeneration, dissection of the ascending aorta, and Marfan syndrome. Less common causes include traumatic injuries to the aortic valve, ankylosing spondylitis, syphilitic aortitis, rheumatoid arthritis, osteogenesis imperfecta, giant cell aortitis, Ehlers-Danlos syndrome, Reiter's syndrome, discrete subaortic stenosis, and ventricular septal defects with prolapse of an aortic cusp. Acute AR is produced by infective endocarditis, aortic dissection, and trauma.

In acute AR, as there is no time to accommodate the increase in regurgitant volume by the normal sized LV, the patient presents with pulmonary oedema and cardiogenic shock. LV end-diastolic pressure is increased and as it approaches the diastolic aortic and coronary artery pressures, myocardial perfusion pressure in the subendocardium is diminished. The

afterload is increased due to LV dilation and thinning of the LV wall and this combines with tachycardia to increase myocardial oxygen demand resulting in ischemia. Death due to pulmonary edema, ventricular arrhythmias, electromechanical dissociation, or circulatory collapse is common in acute severe AR. Urgent AVR is recommended in these patients.

In contrast to this, in chronic AR the left ventricle responds to the volume load with a series of compensatory mechanisms, including an increase in end-diastolic volume, an increase in chamber compliance that accommodates the increased volume without an increase in filling pressures, and a combination of eccentric and concentric hypertrophy. AR represents a condition of combined volume overload and pressure overload.⁷¹ LV ejection fraction is maintained despite the elevated afterload.^{72,73} The majority of patients remain asymptomatic throughout this compensated phase, which may last for decades. Vasodilator therapy has the potential to reduce the hemodynamic burden in such patients.

Natural history of AR

Asymptomatic patients with normal LV systolic function⁷⁴⁻⁸³

Progression to symptoms and/ or LV dysfunction	less than 6% per year
Progression to asymptomatic LV dysfunction	less than 3.5% per year
Sudden death	less than 0.2% per year

Asymptomatic patients with LV dysfunction⁸⁴⁻⁸⁶

Progression to cardiac symptoms	more than 25% per year
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Symptomatic patients⁸⁷⁻⁹¹

Mortality	more than 10% per year
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MANAGEMENT STRATEGY FOR PATIENTS WITH CHRONIC AORTIC REGURGITATION²¹

Medical therapy

There are 3 potential uses of vasodilating agents (ACE inhibitors) in chronic severe AR. (1) long-term treatment of patients with severe AR who have symptoms and/or LV dysfunction who are considered poor candidates for surgery because of additional cardiac or noncardiac factors. (2) Improvement in the hemodynamic profile of patients with severe heart failure symptoms and severe LV dysfunction with short-term vasodilator therapy before proceeding with AVR. (3) Prolongation of the compensated phase of asymptomatic patients who have volume-loaded left ventricles but normal systolic function.

In a recent study of 95 patients who were compared with placebo, long acting nifedipine and enalapril and followed up for seven years showed neither nifedipine nor enalapril reduced the development of symptoms

or LV dysfunction warranting AVR compared with placebo. Hence, the role of vasodilators is not clear at this stage.⁹²

Indications of AVR

AVR is indicated in patients with severe AR who are

- (1) symptomatic
- (2) asymptomatic patients with LV systolic dysfunction
- (3) asymptomatic patients who are undergoing CABG, or other surgery on the aorta, or other heart valves and
- (4) in asymptomatic patients who are having severe LV dilatation (end-diastolic dimension greater than 75 mm or end-systolic dimension greater than 55 mm).

Mixed valvular lesions

Usually a combination of valvular lesions is seen in a patient and the management of each of these patients differs. Hence, a proper assessment of the severity of the lesions by a 2D-echo and sometimes even a cardiac catheterization may have to be done in assessing the dominant lesion.

Mixed single valve disease

In mixed mitral or aortic valve disease, one lesion usually predominates over the other, and the pathophysiology resembles that of the pure dominant lesion. In patients with severe AS and mild AR the management resembles that of pure AS. In mixed mitral disease, predominant MS produces a left ventricle of normal volume, whereas in predominant MR, chamber dilatation occurs. A 2D echo can establish the diagnosis, where the chamber size has to be assessed in determining the dominant lesion. Doppler interrogation of the aortic valve and MVs with mixed disease should provide a reliable estimate of the transvalvular mean gradient. Sometimes cardiac catheterization is helpful in assessing the hemodynamics of a mixed lesion as well as to determine the dominant lesion.

Management²¹

It is difficult to establish perfect guidelines in mixed valve diseases as it is done in the case of a severe pure valve lesion. The most logical approach is to surgically correct disease that produces more than mild symptoms or, in the case of -dominant aortic valve disease, to operate in the presence of even mild symptoms. In regurgitant dominant lesions, surgery can be delayed until symptoms develop or asymptomatic LV dysfunction (as gauged by markers used in pure regurgitant disease) becomes apparent. The use of vasodilators to forestall surgery in patients with asymptomatic mixed disease is untested. Anticoagulants should be used in mixed mitral disease if atrial fibrillation is present. In mixed mitral disease with moderate or severe (3+ to 4+)

regurgitation, percutaneous mitral balloon valvotomy is contraindicated because regurgitation may worsen.

Combined mitral stenosis and aortic regurgitation

When both AR and MS coexist, severe MS usually coexists with mild AR with pathophysiology similar to that of isolated MS. However, the coexistent AR is occasionally severe. MS restricts LV filling, blunting the impact of AR on LV volume. Mechanical correction of both lesions is eventually necessary in most patients. Development of symptoms or pulmonary hypertension is the usual indication for intervention. Combined aortic valve and MV replacement is a reasonable approach, but when correction is anticipated in patients with predominant MS, balloon mitral valvotomy followed by AVR may be performed. In most cases, it is advisable to perform mitral valvotomy first and then monitor the patient for symptomatic improvement. If symptoms disappear, correction of AR can be delayed.

Combined mitral stenosis and tricuspid regurgitation

It is important to establish whether tricuspid regurgitation is primary or secondary to pulmonary hypertension. In general, if pulmonary hypertension is severe and the tricuspid valve anatomy is not grossly distorted, improvement in TR can be expected after correction of MS. Unfortunately, the status of the tricuspid valve after correction of MS is difficult to predict. On the other hand, if there is severe rheumatic deformity of the tricuspid valve, dilatation of the tricuspid annulus, or severe TR, competence is likely to be restored only by surgery. If the MV anatomy is favorable for percutaneous balloon valvotomy and there is concomitant pulmonary hypertension, valvotomy should be performed regardless of symptom status. After successful mitral valvotomy, pulmonary hypertension and TR almost always diminish.⁹³ If MV surgery is performed, concomitant tricuspid annuloplasty should be considered, especially if there are preoperative signs or symptoms of right-sided heart failure, rather than risking severe persistent TR, which may necessitate a second operation.⁹⁴

Combined mitral regurgitation and aortic regurgitation

It is important to determine the dominant lesion and to treat primarily according to that lesion as both these lesions have different pathophysiological effects and different guidelines for the timing of surgery. Although both lesions produce LV dilatation, AR will produce modest systemic systolic hypertension and a mild increase in LV wall thickness. 2D echocardiography is usually performed to assess the severity of AR and MR, LV size and function, left atrial size, pulmonary artery pressure, and feasibility of MV repair. When surgery is required, AVR plus MV repair is the preferred strategy when MV repair is possible.⁹⁵

Combined mitral stenosis and aortic stenosis

In patients with significant AS and MS, the physical findings of AS generally dominate, and those of MS may be overlooked, whereas the symptoms are usually those of MS. Noninvasive evaluation should be performed with 2D and Doppler echocardiographic studies to evaluate the severity of AS and MS, paying special attention to suitability for mitral balloon valvotomy in symptomatic patients, and to assess ventricular size and function. If the degree of AS appears to be mild and the MV is acceptable for balloon valvotomy, this should be attempted first. If mitral balloon valvotomy is successful, the aortic valve should then be re-evaluated.

Combined aortic stenosis and mitral regurgitation

Severe AS will worsen the degree of MR. In addition, MR may cause difficulty in assessing the severity of AS because of reduced forward flow. MR will also enhance LV ejection performance, thereby masking the early development of LV systolic dysfunction caused by AS. Development of atrial fibrillation and loss of atrial systole may further reduce forward output because of impaired filling of the hypertrophied left ventricle. Patients with severe AS and severe MR (with abnormal MV morphology) with symptoms, LV dysfunction, or pulmonary hypertension should undergo combined AVR and MV replacement or MV repair. In patients with mild to moderate AS and severe MR in whom surgery on the MV is indicated because of symptoms, LV dysfunction, or pulmonary hypertension, preoperative assessment of the severity of AS may be difficult because of reduced forward stroke volume. If the mean aortic valve gradient is greater than 30 mm Hg, AVR should be performed.

TRICUSPID VALVE DISEASE

Tricuspid regurgitation

The commonest cause of TR is due to the dilatation of the right ventricle and tricuspid annulus causing secondary (functional) TR rather than primary TR. A systolic right ventricular systolic pressure of greater than 55 mm Hg will cause functional TR, whereas TR occurring with systolic pulmonary artery pressures less than 40 mm Hg is likely to reflect a structural abnormality of the valve apparatus.²¹ Secondary TR can be found in right ventricular hypertension secondary to any form of cardiac or pulmonary vascular disease, most commonly mitral valve disease. Pulmonic stenosis, RVMI, Eisenmenger syndrome, primary pulmonary hypertension and rarely cor pulmonale gives rise to functional TR.

Non rheumatic causes of primary TR includes infective endocarditis, Ebstein anomaly, Tricuspid valve prolapse, L-TGA, Carcinoid, papillary muscle dysfunction due to right ventricular myocardial infarction, trauma, marfan's syndrome, rarely due to rheumatoid arthritis and radiation injury, cleft tricuspid

valve as part of atrioventricular canal malformations. Anorectic drugs may also cause TR.

Patients with severe TR of any cause have a poor long-term outcome because of RV dysfunction and/or systemic venous congestion.⁹⁶ When the valve leaflets themselves are diseased, abnormal, or destroyed, valve replacement with a low-profile mechanical valve or bioprosthesis is often necessary.⁹⁷ A biological prosthesis is preferred because of the high rate of thromboembolic complications with mechanical prostheses in the tricuspid position. Surgery on the tricuspid valve is commonly done at the time of MV surgery. TR associated with dilatation of the tricuspid annulus should be repaired,^{98,99} because tricuspid dilatation is an ongoing process that may progress to severe TR if left untreated.

Tricuspid stenosis

Rheumatic etiology is the commonest cause of tricuspid stenosis. At autopsy, 15% of patients with rheumatic heart disease TS is found but is of clinical significance in only about 5 percent.¹⁰⁰ Rarely, infective endocarditis (with large bulky vegetations), congenital abnormalities, carcinoid, Fabry's disease, Whipple's disease, or previous methysergide therapy may be implicated.¹⁰¹ Right atrial mass lesions represent a nonvalvular cause of obstruction to the tricuspid orifice and may also over time destroy the leaflets and cause regurgitation. Rheumatic tricuspid involvement usually results in both stenosis and regurgitation.

A mean diastolic pressure gradient across the tricuspid valve as low as 2 mm Hg is sufficient to establish the diagnosis of TS. Invasively, right atrial and ventricular pressures should be recorded simultaneously, using two catheters or a single catheter with a double lumen, with one lumen opening on either side of the tricuspid valve.

Tricuspid valve balloon valvotomy has been advocated for tricuspid stenosis of various causes.¹⁰²⁻¹⁰⁴ However, severe TR is a common consequence of this procedure, and results are poor when severe TR develops.

PULMONARY VALVE LESIONS

Pulmonic stenosis

Almost all cases of pulmonary valve stenosis are congenital in origin and only rarely do acquired disorders such as carcinoid and rheumatic fever affect the pulmonary valve. In patients with Noonan's syndrome, the valve may be thickened and dysplastic, with the stenosis caused by inability of the valve leaflets to separate sufficiently during ventricular systole.¹⁰⁵

Children and adolescents are usually asymptomatic even when the pulmonary stenosis is severe, whereas the adults may develop exertional dyspnoea and easy fatigability due to the inability to increase cardiac

output adequately with exercise. Exertional syncope or light-headedness may occur in the presence of severe pulmonic stenosis with systemic or suprasystemic RV pressures, with decreased preload or dehydration, or with a low systemic vascular resistance state (such as pregnancy). Long-standing untreated severe obstruction may lead on to TR and RV failure.

Indications for balloon valvotomy in pulmonic Stenosis

1. In adolescent and young adult patients with pulmonic stenosis who have exertional dyspnea, angina, syncope, or presyncope and an RV-to-pulmonary artery peak-to-peak gradient greater than 30 mm Hg at catheterization.
2. In asymptomatic adolescent and young adult patients with pulmonic stenosis and RV-to-pulmonary artery peak-to-peak gradient greater than 40 mm Hg at catheterization.

Pulmonary regurgitation

It can be caused a variety of conditions like absent or rudimentary pulmonary valve leaflets, bicuspid or quadricuspid pulmonary valves, prolapse of valve leaflets during occlusion of ventricular septal defect, idiopathic dilatation of the pulmonary artery, involvement in carcinoid, rheumatic, or syphilitic disease, or endocarditis, severe pulmonary hypertension and most commonly, iatrogenic, surgical or balloon pulmonary valvotomy/valvuloplasty for pulmonary stenosis or pulmonary atresia or transannular patch to relieve pulmonary valve ring hypoplasia in patients with tetralogy of Fallot.

Symptomatic patients with severe pulmonary regurgitation needs valve replacement, but for asymptomatic patients, the indications based on regurgitant fraction, RV end-diastolic or end systolic volume, and RV ejection fraction remain unclear. It is better to replace the valve before RV function deteriorates, and irreversible damage to ventricular performance occurs.¹⁰⁶

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