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Review Article

Maintenance of sinus rhythm and treatment of atrial fibrillation in mitral stenosis

Musa Sahin[a], Hakki Simsek[b], Berkay Ekici[a], Sekan Akdag[c], Mustafa Tuncer[c]

[a]*Department of , Cardiology Bitlis State Hospital, Bitlis, Turkey*
[b]*Department of Cardiology Osmaniye State Hospital, Osmaniye, Turkey*
[c]*Department of Cardiology Yuzunci Yil University, Faculty of Medicine, Van, Turkey*

Abstract. Rheumatic fever is the most common cause of mitral stenosis. The most common complication of mitral stenosis is atrial fibrillation (AF). AF precipitates symptoms, greatly increases the risk of systemic embolisation, and reduces cardiac output and exercise capacity. Systemic embolization most often occurs in patients with both AF and mitral stenosis. Maintenance of the sinus rhythm in patients with mitral stenosis is very important because of reduce the risk of cerebral embolism, conservation of cardiac output and exercise capacity, and reduction of symptoms.

Key words: mitral stenosis, atrial fibrillation, cerebral embolism

1. Introduction

The predominant cause of mitral stenosis (MS) is rheumatic fever (1). The most common complication of MS is atrial fibrillation (AF) (2). Fundamental moment in MS is beginning of AF which is often caused by atrial inflammation and remodelling. AF occurs in 40–75% of patients who are symptomatic for MS, precipitates such symptoms, greatly increases the risk of systemic embolisation, and reduces cardiac output and exercise capacity (3). Systemic embolization most often occurs in patients with AF and mitral stenosis. Protection of the sinus rhythm in patients with MS is very important because of reduce the risk of cerebral embolism, conservation of cardiac output and exercise capacity, and reduction of symptoms.

The aim of this paper is to emphasize the importance of the protection of sinus rhythm in patients with mitral stenosis.

*Correspondence: Hakki Simsek,
Osmaniye State Hospital, Cardiology Department, Osmaniye, Turkey
Tel: 05052178039
dr.hsimsek@hotmail.com
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2. Mitral Stenosis

Mitral stenosis most frequently follows rheumatic fever (4). Two-thirds of the world's population live in developing countries with a high prevalence of rheumatic fever or rheumatic heart disease, resulting in a large population with mitral stenosis. Progression of MS in developing countries is malignant and intervention is often necessary during teenage years (5). In this countries MS remains an important cause of morbidity. Isolated MS occurs in 40% of all patients presenting with rheumatic heart disease, and a history of rheumatic fever can be elicited from approximately 60% of patients presenting with pure MS (6,7). The ratio of women to men presenting with isolated MS is 2:1 (6-8).

Rheumatic heart disease (RHD) is the most serious manifestation of acute rheumatic fever and is the end result of carditis, which affects 30% to 45% of patients with acute rheumatic fever (9). With acute rheumatic fever, there is inflammation and edema of the leaflets, with small fibrin-platelet thrombi along the leaflet contact zones. Subsequent scarring leads to the characteristic valve deformity with obliteration of the normal leaflet architecture by fibrosis, neovascularization and increased collagen and tissue cellularity. Superimposed calcification results in further dysfunction. This injury to the

cardiac valves, which is the hallmark of rheumatic heart disease, may be chronic and progressive. Rheumatic fever results in characteristic changes of the mitral valve (MV) with the diagnostic features being thickening at the leaflet edges, fusion of the commissures, and chordal shortening and fusion (10). The end is a funnel-shaped mitral apparatus in which the orifice of the mitral opening is decreased in size. Interchordal fusion obliterates the secondary orifices, and commissural fusion narrows the principal orifice (11)

The normal MV area is 4.0 to 5.0 cm^2. Narrowing of the valve area to less than 2.5 cm^2 usually occurs before the development of symptoms (12). With a reduction in valve area by the rheumatic process, blood can flow from the left atrium to the left ventricle just propelled by a pressure gradient. Transvalvular pressure gradient for any given valve area is a function of the square of the transvalvular flow rate (13). Hence a doubling of flow rate quadruples the pressure gradient. The elevated left atrial pressure, in turn, raises pulmonary venous and capillary pressures, resulting in exertional dyspnea. The first bouts of dyspnea in patients with MS are usually precipitated by tachycardia resulting from exercise, pregnancy, hyperthyroidism, anemia, infection, or AF, all of which both increase the rate of blood flow across the mitral orifice resulting in further elevation of the left atrial pressure and decrease the diastolic filling time resulting in a reduction in forward cardiac output (1,14). Because diastole shortens proportionately more than systole as heart rate increases, the time available for flow across the mitral valve is reduced at higher heart rates. Therefore at any given stroke volume, tachycardia results in a higher instantaneous volume flow rate and a higher transmitral pressure gradient, which elevates left atrial pressures further (15). This higher transmitral gradient, often in combination with inadequate ventricular filling (due to the shortened diastolic filling time), explains the sudden occurrence of dyspnea and pulmonary edema in previously asymptomatic patients with MS who develop AF with a rapid ventricular rate.

An MV area greater than 1.5 cm^2 usually does not produce symptoms at rest (16). As the obstruction degree across the MV increases, decreasing effort tolerance occurs. Severity of mitral valve obstruction is best described by using mean gradient, pulmonary artery systolic pressure, and valve area as follows: mild (area greater than 1.5 cm^2, mean gradient less than 5 mmHg, or pulmonary artery systolic pressure less than 30 mmHg), moderate (area 1.0 to 1.5 cm^2, mean gradient 5 to 10 mmHg, or pulmonary artery systolic pressure 30 to 50 mmHg), and severe (area less than 1.0 cm^2, mean gradient greater than 10 mmHg, or pulmonary artery systolic pressure greater than 50 mmHg) (17).

3. Atrial fibrillation

Patients with MS are prone to developing atrial arrhythmias, particularly atrial fibrillation and atrial flutter. Thirty to forty percent of patients with symptomatic MS develop atrial fibrillation (6,7). MS and atrial inflammation secondary to rheumatic carditis causes left atrial dilation, fibrosis of the atrial wall, and disorganization of the atrial muscle bundles. These changes lead to disparate conduction velocities and inhomogeneous refractory periods. Premature atrial activation, caused either by an automatic focus or reentry, may stimulate the left atrium during the vulnerable period and thereby precipitate AF. The development of this arrhythmia correlates independently with the severity of the MS, the degree of left atrial dilation, and the height of the left atrial pressure (2). However, in many series of patients with severe MS undergoing percutaneous balloon mitral valvotomy (PBV), the strongest predictor of AF is older age (18). AF occurs more commonly in older patients (6) with severe MS 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 (8). AF per se causes diffuse atrophy of atrial muscle, further atrial enlargement, and further inhomogeneity of refractoriness and conduction. These changes, in turn, lead to irreversible AF (2).

Atrial contraction augments the presystolic transmitral valvular gradient by approximately 30 percent in patients with MS. Withdrawal of atrial transport when AF develops reduces cardiac output by approximately 20%, and may precipitate or worsen symptoms caused by loss of the atrial contribution to filling and to a short diastolic filling period when the ventricular rate is not well controlled. In addition, risk of arterial embolization, especially stroke, is significantly increased in patients with MS and AF (6,7,19) caused by left atrial thrombus formation. Although systemic embolization most often occurs in patients with AF, 20% of patients with MS and a systemic embolic event are in sinus rhythm (2). When embolization occurs in patients in sinus rhythm, the possibility of transient AF. The risk of embolism correlates directly with patient age and left atrial size and inversely with the cardiac output (2). Approximately half of all

clinically apparent emboli are found in the cerebral vessels (2). Coronary embolism may lead to myocardial infarction and/or angina pectoris, and renal emboli may be responsible for the development of systemic hypertension.

4. Maintenance of sinus rhythm and treatment of atrial fibrillation

Protection of the sinus rhythm in patients with MS is very important because of reduce the risk of cerebral embolism, conservation of cardiac output and exercise capacity, and reduction of symptoms. For the first time that long-term beta blocker therapy causes a significant decrease in P wave duration and dispersion, which are indicating increased risk for AF, in patients with rheumatic mitral Stenosis (20). In the second, early intervention with percutaneous valvotomy may prevent development of AF (18). Sinus rhythm is difficult to achieve and maintain in patients with rheumatic mitral valvular stenosis and AF. Also PBV could allow conversion to normal sinus rhythm in suitable patients (left-atrial diameter <45 mm, duration of atrial fibrillation <1 year) but does not seem to affect persistence of atrial fibrillation (21). Frequency of embolism can be reduced by PBV (22)

Repeated paroxysmal AF may be treated for maintenance of sinus rhythm in selected patients with Class IC antiarrhythmic drugs or Class III antiarrhythmic drugs; however, eventually, the AF becomes resistant to prevention or cardioversion (23), and control of ventricular response becomes the mainstay of therapy. Digoxin slows the heart rate response in patients with atrial fibrillation and MS (24). Nevertheless, heart rate–regulating calcium channel blockers or beta blockers are more effective for preventing exercise induced increases in heart rate. Patients with either paroxysmal or sustained AF should be treated with long-term anticoagulation with warfarin to prevent embolic events if they do not have a strong contraindication to anticoagulation (25,26).

Patients with chronic AF who undergo surgical mitral valve repair or mitral valve replacement may undergo the maze procedure (27). The maze prosedure involves the creation of linear lesions in the atria at cardiac surgery. The decision to performthe maze procedure should be based on surgical expertise as well as patient age and comorbidities, as this procedure may add to the length and complexity of the operation. More than 80% of patients undergoing this procedure can be maintained in sinus rhythm postoperatively and can regain normal atrial function, which may prevent future thromboembolic events by restoring normal sinus rhythm (28,29), including a satisfactory success rate in those with significant left atrial enlargement (27). But the decision to proceed with a Maze procedure should be based on the age and health of the patient, as well as the surgical expertise, because this procedure may add to the morbidity of the operation (17).

Management of AF in patients with MS is similar to management in patients with AF of any cause. However, it typically is more difficult to restore and maintain sinus rhythm because of pressure overload of the left atrium in conjunction with effects of the rheumatic process on atrial tissue and the conducting system (2). Treatment of an acute episode of rapid AF be composed of anticoagulation with heparin and control of the heart rate response. Intravenous digoxin, nondihydropyridine calcium channel antagonist, or beta blockers should be used to control ventricular response by slowing conduction through the atrioventricular node. Also intravenous or oral amiodarone can be used when beta blockers or nondihydropyridine calcium channel antagonist cannot be used. If there is hemodynamic instability, electrical cardioversion should be undertaken urgently, with intravenous heparin before, during, and after the procedure. Patients who have been in AF longer than 24 to 48 hour without anticoagulation are at an increased risk for embolic events after cardioversion, but embolization may occur with less than 24 hour of atrial fibrillation. If the decision has been made to proceed with elective cardioversion in a patient who has had documented AF for longer than 24 to 48 h and who has not been on long-term anticoagulation, One of two approaches is recommended based on data from patients with nonrheumatic AF. The first is anticoagulation with warfarin for more than 3 weeks, followed by elective cardioversion (30). Alternatively, if a transesophageal echocardiogram shows no atrial thrombus, immediate cardioversion can be carried out provided the patient is effectively anticoagulated with intravenous heparin before and during the procedure and with warfarin for at least 1 month after cardioversion (17). Paroxysmal AF and repeated conversions, spontaneous or induced, carry the risk of embolization. In patients who cannot be converted or maintained in sinus rhythm, digitalis should be used to maintain the ventricular rate at rest at approximately 60 beats/min. If this is not possible, small doses of a beta-blocking agent, such as atenolol (25 mg

daily) or metoprolol (50 to 100 mg daily), may be added. Beta blockers are particularly helpful in preventing rapid ventricular responses that develop during exertion. Multiple repeat cardioversions are not indicated if the patient fails to sustain sinus rhythm while on adequate doses of an antiarrhythmic (2).

In conclusion maintenance of the sinus rhythm in patients with mitral stenosis is very important because of reduce the risk of cerebral embolism, conservation of cardiac output and exercise capacity, and reduction of symptoms.

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