# Comparison of two patients with mitral stenosis and importance of sinus rhythm: case report

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**Abstract.** We present two woman patients have rheumatic mitral stenosis (MS). They complained of about shortness of breath and palpitation. First patient, she had had two stroke without any sequela (respectively 7 years and 4 years ago). And she has atrial fibrillation (AF) in electrocardiography (ECG). Another woman patient didn't have any complaint before. Latter patient has sinus tachycardia in ECG. Transthoracic echocardiography (TTE) disclosed severe mitral valve stenosis and left atrial dilatation in both patients. But first patient has multiple thrombus in left atrium. Mitral valve surgery was suggested for both patients. In conclusion maintenance of sinus rhythm, in the patients who had MS, is very important for both the protection of left atrial thrombus and the reduction of the development of systemic embolus related to it.

Key words: mitral stenosis, sinus rhythm, atrial fibrillation, left atrial thrombus

#### 1. Introduction

The predominant cause of mitral stenosis (MS) is rheumatic fever (1). The interval between the initial episode of rheumatic fever and clinical evidence of mitral valve obstruction is variable, ranging from a few years to more than 20 years. The most common complication of MS is atrial fibrillation (AF) (1). Beginning of AF, which is often caused by atrial inflammation and remodelling, is a fundamental moment in MS. AF occurs in 40-75% of patients who are symptomatic for MS, precipitates such symptoms, greatly increases the risk of systemic embolism, and reduces cardiac output and exercise capacity (2). Although systemic embolism mostly occurs in patients with AF, 20 percent of patients with MS and a systemic embolic event are in sinus rhythm (1).

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#### 2. Case reports

First case: A 66-year-old women admitted to our outpatient clinic with shortness of breath and palpitation. She had had two stroke without any sequela (respectively 7 years and 4 years ago). But the patient did not have any cardiac examination carefully before. In cardiac examination, a diastolic rumbling was heard over the apex. Electrocardiography (ECG) revealed atrial fibrillation with rapid ventricular response. The blood pressure was 110/70 mmHg with irregular pulse and the rate was 110/min. TTE disclosed severe mitral valve stenosis (1 cm<sup>2</sup>) with a peak gradient of 19 mmHg and a mean gradient of 10 mmHg across the valve, left atrial dilatation (7.4 cm), multiple thrombus in the left atrium, severe tricuspid valve regurgitation and a artery pressure of 70 mmHg pulmonary (Fig. 1).

Second case: A 67-year-old woman admitted to our outpatient with shortness of breath. The shortness of breath has started a week ago. In cardiac examination, a diastolic rumbling was heard over the apex. ECG revealed sinus tachycardia. The blood pressure was 150/90 mmHg with regular pulse rate of 105/min. TTE

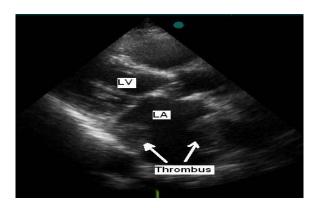


Fig. 1. First patient with severe MS, AF and left atrial thrombus.

disclosed severe mitral valve stenosis (0.9 cm<sup>2</sup>) with a peak gradient of 16 mmHg and a mean gradient of 10 mmHg across the valve, left atrial dilatation (4.9cm), mild/moderate mitral regurgitation, mild aortic regurgitation, moderate tricuspid valve regurgitation and a pulmonary artery pressure of 60 mmHg (Fig. 2). Mitral valve surgery was suggested for both patients.



Fig. 2. Second patient with severe MS and sinus tachycardia

## 3. Discussion

Rheumatic heart disease 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 (3). Injury to the cardiac valves, which is the hallmark of rheumatic heart disease, may be chronic and progressive and, in conjunction with left ventricular dysfunction, can lead to congestive heart failure and death. Both acute rheumatic fever and rheumatic heart disease are rare in developed countries, but these continue essentially unchecked in many developing countries (4)

Rheumatic carditis is major cause of mitral stenosis. Isolated MS 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 (5). MS prevents suitable opening during diastolic filling of the left ventricle because of which is an obstruction to left ventricle inflow at the level of the mitral valve as a result of a structural abnormality of the mitral valve apparatus. Hence blood can flow from the left atrium to the left ventricle only if propelled by a pressure gradient. This diastolic transmitral gradient is the essential expression of MS (6) and results in elevation of left atrial pressure, which is reflected back into the pulmonary venous circulation.

Mitral stenosis and atrial inflammation secondary to rheumatic carditis causes left atrial dilatation, 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 (1). However, in many series of patients with severe MS undergoing percutaneous balloon valvuloplasty (PBV), the strongest mitral predictor of AF is older age (7). 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 (1). AF is the most common arrhythmia in patients with MS in clinical practice. Risk of arterial embolization, especially stroke, is significantly increased in patients with AF (8). Also AF reduces cardiac output, exercise capacity (2) and increases symptoms such as palpitations and shortness of breath. Likewise, our first patient with severe MS and AF had higher palpitation and shortness of breath than latter. Also this patient had two stroke without any sequela (respectively 7 years and 4 years ago) and did not have any cardiac examination carefully before. Therefore MS has not been detected and anticoagulant therapy has not been begun. So we detected multiple thrombus in left atrium by TTE.

The protection of the sinus rhythm in patients with MS is very important to reduce the risk of cerebral embolism, conservation of cardiac output and exercise capacity, and reduction of symptoms such as palpitations and shortness of breath. In parallel the symptoms of our second patient who has severe MS and sinus rhythm, were less than the first patient. Also, there was no thrombus in left atrium of the second patient and left atrial dilatation was less than first patient. Also the presence of mild/moderate mitral regurgitation may contribute to do not formation of thrombus in left atrium in second patient. Maintenance of sinus rhythm is changeable across patients, with most (9) but not all studies (10) showing substantial difficulty. For the first time that longterm beta blocker therapy causes a significant decrease in P wave duration and dispersion, which are indicating increased risk for AF, in patients with rheumatic MS (11). Treatment of an acute episode of rapid AF consists of anticoagulation with heparin and control of the heart rate response by intravenous digoxin, heart rate-regulating calcium channel blockers, or beta blockers, or amiodarone. If there is hemodynamic instability, electrical cardioversion should be undertaken urgently. In selected patients, chemical cardioversion may also be attempted (12). Early intervention with percutaneous valvuloplasty may prevent development of AF (7). 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 AF (4). Frequency of embolism can be reduced by PBV (13). Repeated paroxysmal AF may be treated for maintenance of sinus rhythm in selected patients with Class IC antiarrhythmic drugs or Class III antiarrhythmic drugs (12).

Patients with chronic AF who undergo surgical mitral valve repair or mitral valve replacement may undergo the maze procedure (atrial compartment operation) (14). More than 80 percent of patients undergoing this procedure can be maintained in sinus rhythm postoperatively and can regain normal atrial function, including a satisfactory success rate in those with significant left atrial enlargement (14). As a result, each patient, experienced cerebral emboli and had AF should be necessarily evaluated for the rheumatic heart disease. And maintenance of sinus rhythm, in the patients who had MS, is very important for both the protection of left ventricular ejection fraction, and left atrial thrombus and the reduction of the development of systemic embolism related to it.

### Referances

 Otto CM, Bonow RO. Valvular heart disease. In: Braunwald E. (ed) Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine, 8th ed. Philadelphia, Saunders 2008, pp1646-1656.

- 2. Vora A, Karnad D, Goyal V, et al. Control of rate versus rhythm in rheumatic atrial fibrillation: a randomized study. Indian Heart J 2004; 56: 110-116.
- Moore AG. Rheumatic Heart Disease. In: Murphy JG, Lloyd MA. (eds) Mayo Clinic Cardiology, (3rd ed). Rochester, Mayo Clinic Scientific Pres 2007, pp 549-554.
- 4. Chandrashekhar Y, Westaby S, Narula J. Mitral stenosis. Lancet 2009; 374: 1271-1283.
- Rowe JC, Bland EF, Sprague HB, White PD. The course of mitral stenosis without surgery: ten-and twenty-year perspectives. Ann Intern Med 1960; 52: 741-749.
- 6. Braunwald E, Moscovitz HL, Mram SS, et al. The hemodynamics of the left side of the heart as studied by simultaneous left atrial, left ventricular, and aortic pressures; particular reference to mitral stenosis. Circulation 1955; 12: 69-81.
- Krasuski RA, Assar MD, Wang A, et al. Usefulness of percutaneous balloon mitral commissurotomy in preventing the development of atrial fibrillation in patients with mitral stenosis. Am J Cardiol 2004; 93: 936-939.
- Coulshed N, Epstein EJ, McKendrick CS, Galloway RW, Walker E. Systemic embolism in mitral valve disease. Br Heart J 1970; 32: 26-34.
- 9. Langerveld J, van Hemel NM, Kelder JC, et al. Long-term follow-up of cardiac rhythm after percutaneous mitral balloon valvotomy. Does atrial fibrillation persist? Europace 2003; 5: 47-53.
- 10. Hu CL, Jiang H, Tang QZ, et al. Comparison of rate control and rhythm control in patients with atrial fi brillation after percutaneous mitral balloon valvotomy: a randomised controlled study. Heart 2006; 92: 1096–1101.
- 11. Erbay AR, Turhan H, Yasar AS, et al. Effects of long-term beta-blocker therapy on P-wave duration and dispersion in patients with rheumatic mitral stenosis. International Journal of Cardiology 2005; 102: 33-37.
- 12. Bonow RO, Carabello BA, Chatterjee K, et al. 2008 Focused Update Incorporated Into the ACC/AHA 2006 Guidelines for the Management of Patients With Valvular Heart Disease: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 1998 Guidelines for the Management of Patients With Valvular Heart Disease): Endorsed by the Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons Circulation 2008;118: e564.
- Chiang CW, Lo SK, Cheng NJ, et al. Predictors of systemic embolism in patients with mitral stenosis. A prospective study. Ann Intern Med 1988; 128: 885-889.
- Otto CM. Surgical and percutaneous intervention for mitral stenosis. In: Otto CM. (ed) Valvular Heart Disease, (2nd ed.) Philadelphia: Saunders 2004, pp 272-276.