

An Overview of the Mitral Regurgitation

Mitral Yetersizliğine Kısa Bir Bakış

Mitral regurgitation (MR) may be secondary to abnormality of the valve leaflets, mitral annulus, papillary muscles and their adjacent musculature. Common causes that affect these structures are rheumatic heart disease, annular dilatation and calcification, degenerative disease and myocardial ischaemia or infarction (1-3). Echocardiographic evaluation of MR (1-3) includes:

1. Two-dimensional color and continuous wave Doppler studies. Doppler echocardiography for detecting MR has nearly 100% sensitivity. Specificity is also high for significant regurgitation. Functional and anatomical relations of mitral valve and left ventricle can be evaluated.

2. Extension of the regurgitant jet into the left atrium. In these method, MR is graded by various parameters such as jet length, jet area, ratio of jet area to left atrium. Jet area $<4 \text{ cm}^2$ is accepted as mild, $4-8 \text{ cm}^2$ as moderate and $>8 \text{ cm}^2$ as severe MR. For the ratio of jet area to left atrium, $<20\%$ is accepted as mild, $20-40\%$ as moderate and $>40\%$ as severe MR. However, jet area is dependent on gain settings, pulse repetition frequency, field depth, direction of the jet and loading conditions.

3. Regurgitant volume and regurgitant fraction.

4. Signal intensity of the regurgitant jet. The strength of the continuous wave Doppler signal of the regurgitant jet relative to that of antegrade transmitral flow provides information about severity of MR. However this method has many limitations.

5. Transmitral early filling velocity. As a result of elevated left atrium pressure, peak early transmitral filling velocities are increased in severe MR ($>1.3 \text{ m/s}$).

6. Pattern of flow in the pulmonary veins. In severe MR, the marked systolic elevation in left atrium causes reversal of systolic pulmonary venous flow. For diagnosing of severe MR, this method has 96% specificity and 52% sensitivity. A blunted systolic flow and a systolic to diastolic velocity ratio of less than one may be seen in moderate MR. However, this is not reliable because systolic to diastolic velocity ratio of less than one can be seen in normal young adults and in patients with left ventricular dysfunction and elevated left ventricle filling pressure.

7. Pulmonary artery systolic pressure. Severe MR causes elevation in left atrial pressure and elevati-

on of pulmonary artery systolic pressure.

8. Impact on the left atrium and left ventricle. A left atrium with diameter more than $5,5 \text{ cm}$ and a dilated left ventricle with end-diastolic diameter over 7 cm supports the diagnosis of severe MR. A reduction in the left ventricular ejection fraction together with a progressive increase in end-systolic diameter and volume are important criteria for timing of mitral valve surgery.

9. Vena contracta width. Vena contracta is the width of regurgitant jet at the narrowest part proximally to regurgitant orifice. Width of $>0,5 \text{ cm}$ is related to severe MR. This method needs high resolution and zoom images technically or transesophageal echocardiographic recordings. Because of limited lateral resolution, it may cause overestimation.

10. Proximal isovelocity surface area (PISA) method. This method is not used routinely. It is based on the principles of conservation of flow and continuity equation. The acceleration of flow towards to the regurgitant orifice on the left ventricular side of mitral valve forms isovelocity hemispheres. Because of all flow that passes through regurgitant orifice must first pass through these isovelocity hemispheres, it is possible to calculate regurgitant velocity and effective regurgitant orifice. Effective regurgitant orifice of $1-10 \text{ mm}^2$ upper scrip is graded as mild, $10-25 \text{ mm}^2$ as moderate-severe and $>50 \text{ mm}^2$ as severe. There are technical and practical factors that limit use of this method such as presence of more than one jet, eccentricity of jet and losing hemispheric shape.

There are medical and surgical (valve repair and valve replacement) therapies in management of chronic MR.

Medical therapy of chronic MR includes diuretics and nitrates for pulmonary congestion, beta-blockers and digoxin for rate control and antiarrhythmics for rhythm control in presence of atrial fibrillation (1, 2).

Drugs that reduce afterload are beneficial in the management of both acute and chronic forms of MR. By reducing the impedance to ejection into the aorta, the volume of blood regurgitating into the left atrium is reduced. In addition, decreasing left ventricular volume reduces the diameter of the mitral annulus and thereby the regurgitant orifice. Angiotensin converting enzyme (ACE) inhibitors reduce regurgitant volume and increase antegrade flow. These drugs are effective especially in the management of symptomatic patients (1, 2).

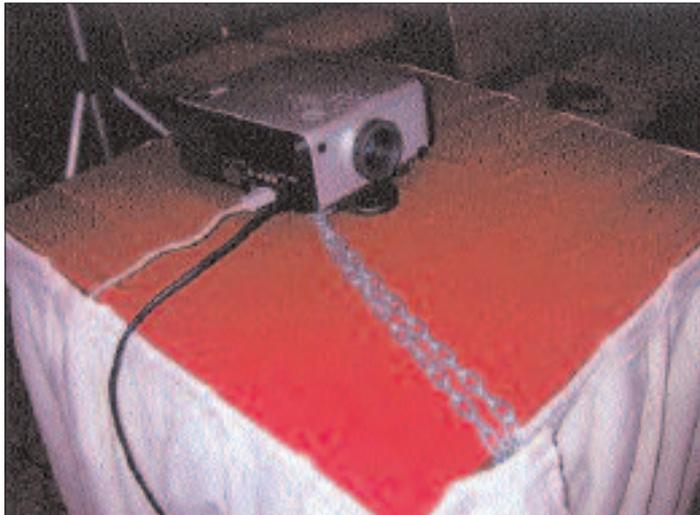
Effect of ACE inhibitors on asymptomatic MR has been evaluated in several studies (4). Calabro et al, have demonstrated that single dose of oral enalapril reduces degree of MR and improves left ventricle loading conditions and systolic performance (5). It has been reported that long-term quinapril treatment makes regression in left ventricle dilatation, hypertrophy and mass in regurgitant valves including MR and potentially may postpone the time of the mitral valve replacement (6). Marcotte et al. have suggested that lisinopril treatment reduces degree of MR in chronic moderate MR with normal left ventricular function but reported a significant rate of side effects (7). Shcon et al. have similarly reported that quinapril treatment improves functional capacity by reducing regurgitant fraction, left ventricular diameters and mass in patients with chronic moderate MR and delays the timing for surgery (8). Lanass et al. have also demonstrated that digoxin and enalapril together improve functional class in patients with heart failure due to MR (9). Losartan has been shown to have similar effects (10).

In the present study (11), published in the March issue of the Anadolu Kardiyoloji Dergisi the effect of the enalapril on the effective regurgitant orifice in patients with rheumatic mitral regurgitation was investigated. The authors have concluded that it would be appropriate to initiate ACE inhibitor therapy in early stage of disease in pediatric population. Although this study does not provide findings of the previous studies, the authors may be congratulated for their efforts and work-up.

Osman Bolca, MD
Clinic of Cardiology
Siyami Ersek Center of Thorax and
Cardiovascular Surgery, İstanbul

References

1. Braunwald E, Zipes DP, Libby P. Valvular heart disease. In: Braunwald E, Zipes DP, Libby P, editors. Heart Disease: A Textbook of Cardiovascular Medicine. 6th edition. Philadelphia, PA: WB Saunders Co; 2001. p.1653-60.
2. Crawford MH, DiMarco JP, Paulus WJ. Cardiology. 2nd edition. London: Mosby; 2004. p.1039-83.
3. Weyman A. Principles and Practice of Echocardiography. 2nd edition. London: Lea-Febriger; 1994. p.428-92.
4. Pisacane C, Pacileo G, Santoro G, et al. New insights in the pathophysiology of mitral and aortic regurgitation in pediatric age: role of angiotensin-converting enzyme inhibitor therapy. Ital Heart J 2001; 2: 100-6.
5. Calabro R, Pisacane C, Pacileo G, Russo MG. Hemodynamic effects of a single oral dose of enalapril among children with asymptomatic chronic mitral regurgitation. Am Heart J 1999; 138: 955-61.
6. Schon HR. Hemodynamic and morphologic changes after long-term angiotensin converting enzyme inhibition in patients with chronic valvular regurgitation. J Hypertens 1994 (Suppl);12: S95-S104.
7. Marcotte F, Honos GN, Walling AD, et al. Effect of angiotensin-converting enzyme inhibitor therapy in mitral regurgitation with normal left ventricular function. Can J Cardiol 1997; 13: 479-85.
8. Schon HR, Schroter G, Barthel P, Schomig A. Quinapril therapy in patients with chronic mitral regurgitation. J Heart Valve Dis 1994; 3: 303-12.
9. Lanass F, Garces E, Eggers G, et al. Comparison of the effects of digoxin or enalapril in the treatment of heart failure due to mitral insufficiency. Rev Med Chil 1998; 126: 251-7.
10. Dujardin KS, Enriquez-Sarano M, Bailey KR, Seward JB, Tajik AJ. Effect of losartan on degree of mitral regurgitation quantified by echocardiography. Am J Cardiol 2001; 87: 570-6.
11. Tunaoglu FS, Olgunturk R, Kula S, Oğuz D. Effective regurgitant orifice area of rheumatic mitral insufficiency: response to angiotensin converting enzyme inhibitor treatment. Anadolu Kardiyol Derg 2004; 4: 3-7.



Prof.Dr. Cahide Soydaş Çınar Prangalı Projektör, Arjantin 2003