Mitral stenosis-beyond valvular disease

Mitral stenosis-kapak hastalığı ötesinde

Left ventricular function in patients with mitral stenosis has been investigated during the last decades. First angiographic studies (1, 2) found higher left ventricular end-systolic volumes and lower ejection fraction in patients with mitral stenosis than in controls (1, 2). Ventriculography showed distorted contraction of the postero-basal segment (1-3) and occasionally anterior hypokinesis (2, 4), that was related to the rigidity and immobilization of mitral valve complex, proposed to be due to scarring of the mitral valve complex and fibrosis of the papillary muscle. In some angiographic studies, generalized rather than regional LV motion abnormalities due to immobility and rigidity of the mitral valve apparatus;

2. Subtle generalized LV dysfunction due to rheumatic myocarditis known as myocardial factor;

3. Altered loading condition of the left ventricle due to under filling of the left ventricle.

Based on angiographic data it was difficult to define, which of these mechanisms has a predominant impact on left ventricular dysfunction in patients with rheumatic mitral stenosis.

Echocardiography allowed better visualization of the mitral valve, cardiac structure and function. Low-normal ejection fraction in patients with mitral stenosis was reproduced in first echocardiographic studies (7). Indexes of myocardial performance-stroke volume, ejection fraction, mean rate of circumferential fibers shortening, posterior wall and septal velocities were reduced in patients with mitral stenosis and correlated with clinical functional disability (8). Geometric changes in the left ventricular shape were observed in patients with rheumatic mitral stenosis. The left ventricle tended show a spherical rather than ellipsoidal shape, especially in the apical segments, due to architectural remodeling along with increased wall stress that was related to myocardial factor (9).

In the heart with mitral stenosis the inflow tract of the left ventricle is shorter than the outflow tract, the mitral ring is tilted and is oblique to the direction of the inflow tract; the circumference and the antero-posterior diameter of the mitral ring are greatly increased and the posterior wall of the left ventricle is shortened due to selective atrophy of the portion of the papillary muscle which is inserted into the posterior rim of the mitral-aortic ring. The thickening of the valve leaflets and fibrosis of the chordae tendineae convert the valve into a rigid cylinder of dense scar tissue, immobilizing the posterior wall of the left ventricle with atrophy of this immobilized posterior papillary muscle (10).

Quantitative echocardiographic techniques have been important in the accurate assessment of systolic and diastolic deformation parameters in mitral stenosis. Doppler Tissue Imaging studies showed altered longitudinal deformation in patients with mitral stenosis: reduced myocardial mitral annular velocities (11), reduced annular velocities that correlated with M-mode mitral annular displacement and with left atrial ejection fraction (12).

Increase of systolic and diastolic myocardial annular velocities was observed in patients after percutaneous balloon mitral valvuloplasty as evidence of reversibility of left ventricular performance in mitral stenosis after the relief of obstruction (13). Another evidence of longitudinal LV systolic dysfunction in patients with mild to moderate mitral stenosis was obtained with Doppler tissue imaging - derived annular peak systolic strain rate and end systolic strain, that both were significantly lower in patients with mitral stenosis (14). 2D strain demonstrated that global strain and global strain rate are reduced in patients with mitral stenosis compared to normal subjects (15) as evidence of subclinical left ventricular dysfunction, which was interpreted as a rheumatic myocardial factor.

The impact of myocardial factor in subtle LV dysfunction in isolated rheumatic mitral stenosis was supported by biopsy-study performed in 15 patients from the left ventricle. Various degrees of pathological alterations of myocardial structures were observed in all patients: loss of myofilaments, degenerative changes in myofibrils, disarray and loss of myofibrils with changes in the shape and size of muscle cells, ultrastructural changes of the mitochondria, changes in nuclei and Golgi appa-
consideration when patients with mild-moderate mitral valve stenosis is important, and this should be taken into the consideration of rheumatic myocarditis. Subclinical myocardial dysfunction due to rheumatic myocarditis can be responsible for part of the symptoms in symptomatic patients with mild-moderate mitral stenosis.

The reversibility of subclinical LV dysfunction in mitral stenosis after the relief of obstruction was investigated. Posterior wall systolic and diastolic velocities measured by M-mode echocardiography improved in the patients with mitral stenosis after open mitral commissurotomy (19). Improvement of annular myocardial velocities immediately after the percutaneous mitral valvuloplasty (13) indicates at least partial reversibility of left ventricular dysfunction after the relief of obstruction.

The current study indicates that the impact of rheumatic myocarditis (myocardial factor) on subclinical left ventricular dysfunction is important, and this should be taken into the consideration when patients with mild-moderate mitral valve stenosis are referred for balloon mitral valvuloplasty.

Marina Leitman, Zvi Vered
Department of Cardiology, Assaf Harofeh Medical Center, Zerifin; Sackler School of Medicine, Tel Aviv University, Israel

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