Speckle-tracking imaging for the progression of mitral stenosis

In the pre-echocardiographic era of diagnosis, follow-up and prognosis of patients with mitral stenosis were assessed clinically and based on the cardiothoracic ratio on chest X-ray (1). Patients with small or only slightly enlarged hearts had better prognosis (1). Diagnosis was validated on auscultation, during surgery, or on autopsy (2). In the 1970s, pressure gradient over the mitral valve was measured directly during cardiac catheterization, and mitral valve area was calculated according to the Gorlin formula (3). The “myocardial factor” was thought to be responsible for the disability in patients with chronic atrial fibrillation, low cardiac output, and only mild mitral valve obstruction, and these patients did not improve following surgery (3). With the advance of modern echocardiography, accurate visualization and calculation of mitral valve gradient and area became available. Recently, the definition of mitral stenosis severity has changed (4). Most patients with mitral stenosis develop symptoms during the fourth to fifth decade of life—similar to the population described in the study of Gerede et al in the issue of the Journal (5). Follow-up of patients with mitral stenosis is usually based on clinical and echocardiographic parameters; however, additional predictors for possible progression could be valuable. Longitudinal and circumferential strain in patients with mitral stenosis and apparently normal myocardial function were reduced compared to normal subjects (6). Several factors may be responsible for myocardial dysfunction in mitral stenosis, including reduced preload of the left ventricle, rheumatic myocarditis (myocardial factor), and fibrotic changes of the basal left ventricular segments close to the rigid rheumatic mitral valve (7, 8). There is controversial data regarding the deformation parameters of the left ventricle in mitral stenosis. Longitudinal strain and strain rate were reduced in patients with mitral stenosis compared to the control subjects, but no correlation was found with severity of mitral stenosis (9). This may suggest that unloading of the left ventricle is not a major determinant of reduced strain. In another work, global longitudinal and circumferential strain improved significantly shortly after percutaneous balloon mitral valvuloplasty (10), which may support an opposite concept. In the work of Gerede et al (5), reduced global strain and strain rate was predictive for progression of mitral stenosis. These observations are of potential interest. Worse myocardial mechanics could be related to the older age of these patients with rheumatic valve disease and more prominent changes in the myocardium. Patients with mitral stenosis had likely been infected with group B beta-hemolytic streptococcus, but not all are affected with rheumatic fever and rheumatic heart disease in a similar fashion. Aschoff bodies develop in individuals who are sensitive to the antigens. Probably, more sensitive patients will develop more significant valvular heart disease and rheumatic myocarditis, similar to the patients observed in this study. Therefore, patients with mitral stenosis and lower strain and strain rate despite normal ejection fraction should be followed up more closely. Additional larger studies are required to assess the true significance of this work.

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