

How to deal with calcified mitral stenosis and diastolic dysfunction

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Introduction: As the population ages, there is an increase in mitral apparatus degenerative disease and a decrease in rheumatic disease. Diagnosis and treatment are quite different due to the different distribution of calcifications and the heart diastolic conditions in which the diseases occur.¹⁻³

Case report: 74-year-old male patient with a history of long-term arterial hypertension, diabetes, permanent atrial fibrillation, and mechanical aortic valve implantation in 2006 was admitted to the hospital due to exercise intolerance, NYHA (New York Heart Association) III class, with clinical signs of dominantly right-sided decompensation. Echo revealed preserved systolic function of the non-dilated left ventricle, but diastolic dysfunction in the restriction phase. There was a slightly dilated right ventricle with decreased longitudinal function, TAPSE (tricuspid annular plane systolic excursion) 12mm, bounce and a D-shape of ventricular septum in diastole. The mechanical aortic valve was functioning well. Mitral valve area (MVA) planimetry was not done due to poor window in parasternal short axis (PSAX). A mean gradient of 7mmHg was measured by continuous wave (CW) Doppler. MVA of 2.7cm² was obtained by pressure half time (PHT). With longer heart cycles diastasis was visible at the end of diastole. The continuity equation was not used because of atrial fibrillation. By PISA (proximal isovelocity surface area) method, which is an only echo method independent from flow conditions in mitral stenosis, calculate area was 1.0 cm². Pericardial calcification deposits were shown by ultrasound and then by CT scan. Considering the inconsistent findings of mitral stenosis severity with a possible diagnosis of constrictive pericarditis, right heart catheterization (RHC) was performed. It revealed very high left ventricular end diastolic pressure (LVEDP) (40 mmHg), severe pulmonary hypertension (79/34 mmHg, mean 54 mmHg) as a combination of high pulmonary capillary wedge pressure (PCWP) (25 mmHg) and pulmonary vascular resistance (PVR) (6.55 Wood). The diastolic pressures of the left and right side of the heart differed significantly (right ventricular end diastolic pressure (RVEDP) of 15mmHg and LVEDP of 40 mmHg, so constrictive pericarditis was ruled out.

Conclusion: After all, different results were obtained measuring the severity of mitral valve stenosis, but with certain severe diastolic dysfunction of the left ventricle and severe pulmonary hypertension. The patient was further presented to the heart team and a pulmonary hypertension reactivity test was performed. Since the result was positive, sildenafil therapy was introduced. The RHC will be repeated in three months when the heart team will decide on further treatment modalities.

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LITERATURE

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