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Regional left ventricular longitudinal myocardial dysfunction in mitral valve prolapse could be primary

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Background. Regional left ventricular dysfunction in patients with mitral valve prolapse (MVP) and normal ejection fraction has been described by different Authors, and recent data point to a dysfunction (prevalently longitudinal strain) of the myocardium of the LV base secondary to dilatation of the mitral annulus.

Purpose. To investigate degree and extent of regional LV dysfunction and its mechanisms in patients with MVP, severe regurgitation and normal global systolic function, compared to patients with equivalent degree of regurgitation but functional etiology (FMR).

Methods. Speckle-tracking echocardiography was performed in 30 controls (N), and in severe primary (MVP, n= 50) or functional (FMR, n= 20) mitral regurgitation, to measure global, regional and segmental longitudinal peak systolic strain (LPSS, %), and time delay of peak maximum strain (TTPd, ms, calculated as time to peak maximum strain - time of aortic valve closure). Maximum and minimum mitral annulus diameters and area were measured with 3D echo. We also evaluated as recommended: LV end-diastolic volume index (EDVi, ml/m2), ejection fraction (EF, %), and left atrial end-systolic volume index (LAESVi, ml/m2) with 2D echo; LV stroke volume index, and non-invasive pulmonary systolic (PSP, mmHg) and diastolic pressures (PDP), mmHg) with Doppler echo.

Results. Age, heart rate, BSA and systolic blood pressure were similar between groups. Atrial fibrillation was present in 34% of MVP and 71% of FMR patients. LV EF was normal in MVP and reduced in FMR (43 ± 14 % vs N, p<.001). LV EDVi (MVP: 77 ± 20 ml/m2; FMR: 107 ± 35, both p<.001 vs N) and LAESVi (MVP: 91 ± 26 ml/m2; FMR: 80 ± 30, both p<.001 vs N) were similarly increased (volume overload) in MVP and FMR, as were PSP (MVP: 42 ± 23 ml/m2; FMR: 52 ± 25, both p<.001 vs N) and PDP (MVP: 16 ± 6 ml/m2; MVP: 15 ± 5, both p<.001 vs N). In FMR, LPSS was reduced globally (-12.8 ± 3.3, p<.001 vs N and MVP) and similarly at LV base, papillary and apical levels. In contrast, in MVP global (-19.4 ± 3.7%) and apical (-23.4 ± 4.5%) LPSS were normal, whereas LV base (-12.3 ± 5.8%, p=.003 vs N) and papillary (-17.1 ± 4%, p=.024 vs N) LPSS were reduced; further, LPSS reduction was localized to the anterior (-16 ± 4, p=.028 vs N), lateral (-17 ± 5, p=.006 vs N) and posterior (-16 ± 6, p=.007 vs N) segments, and was associated with an increased TTPd in the same segments in MVP but not in FMR patients. At multivariate analysis, degree and localisation of regional myocardial dysfunction in patients with MVP was not related to the prolapsing scallop, dimension of the mitral annulus, degree of volume overload or pulmonary pressures, or stroke volume index.

Conclusions. In patients with MVP, severe regurgitation and normal EF, there is a specific dysfunction pattern of regional LV longitudinal function which appears to be primary and not dependent on the degree of preload increase, mitral annulus dilatation, or localization of the prolapsing scallop.