




Prognostic value of left ventricular longitudinal strain in patients with aortic stenosis

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Left ventricular (LV) hypertrophy in patients with aortic stenosis (AS) is an adaptive mechanism that tries to maintain left ventricular systolic function despite obstruction.¹ The appearance of myocardial fibrosis (MF) is a turning point from adaptive hypertrophy to maladaptation, that changes ventricular function and represents the basis for development of ventricular arrhythmias. There are two types of MF: reversible interstitial MF in early stage, and irreversible replacement MF in the later stage of disease. MF occupies LV midwall, usually in LV posterobasal part.

Cardiac magnetic resonance (CMR) by late gadolinium enhancement (LGE) is the „gold standard“ in diagnosis of replacement MF. Positive LGE results indicate significantly greater risk of adverse outcome than it is in patients without MF. Echocardiography suggests the presence of MF with significant reliability, because global longitudinal strain (GLS) shows good correlation with results of CMR. Consequently, GLS can serve as a surrogate marker of MF and an indicator of adverse events in AS.²⁻⁴

MF mostly influences LV longitudinal function, while circumferential and radial deformation are less modified. In severe compensated AS circumferential deformation and apical rotation (ApR) are increased, what is likely a compensative mechanism for keeping cardiac output. The increased ApR is also associated with worse survival. With disease progression these compensative changes disappear.

Mechanical dispersion (MD) is an indicator of contractile heterogeneity caused by fibrosis. Increased MD demonstrates significant association with mortality and can serve as an additional prognostic parameter. Along with more extensive MF the risk for sudden cardiac death increases what raises the question of the benefit of cardioverter-defibrillator.

In conclusion, impaired GLS suggests MF, what must be confirmed by CMR. MF classifies the patients with severe asymptomatic AS into the group with increased risk of adverse outcome. Therefore, it is to be expected that these new facts will result in new guidelines for treatment of severe asymptomatic AS.

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LITERATURE

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