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The Incremental Effect of Obesity on Myocardial Fibrosis In Patients with Aortic Stenosis

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There are many studies that show the myocardial fibrosis resulting from hypertension, aortic stenosis or hypertrophic cardiomyopathy, which are under the same condition with chronic pressure overloading.[1-4] Multiple factors are recommended for impaired cardiac function in patients with hypertension, such as inflammation, adaptive ventricular remodeling, increased mechanical stress inducing subsequent ventricular hypertrophy, interstitial and perivascular fibrosis, endothelial dysfunction and neurohormonal factors.[5-10]

The development of left ventricular (LV) hypertrophy is actually a combined consequence of chronic pressure or volume overload in hypertension or aortic stenosis. To compensate for chronic pressure overload in these subjects, LV wall thickness gradually increases in order to normalize wall stress, leading to concentric LV remodeling and hypertrophy.[11-13] Activation of several biological processes including various hormones, growth factors and cytokines also contribute to protein genesis by promoting muscle cell growth, leading to structural alterations and remodeling.[14]

We can inference the fatigue and essential compensatory mechanism of myocardium. The similar process can occur in obese patients. Moderate to severe cases of obesity was presented as leading to increased LV wall stress, compensatory LV hypertrophy and LV dysfunction. Alpert[15] used a term of 'obesity cardiomyopathy' expressing the series of myocardial dysfunction. Compared with healthy lean individuals, increased epicardial adipose tissue in obese group is expected to result in more extensive fatty infiltration in the myocardium.[16] They showed the correlation between incidence of atrial fibrillation and an excess adiposity and fibrosis in obesity with histologic demonstration.[17]

In this study, Ávila-Vanzzini et al.[18] presented concomitant overweight and obesity (OW/O) leading to pathological LV remodeling in patients with aortic stenosis. The most interesting point is that the authors revealed histologic patterns correlated well with body mass index or global longitudinal strain (GLS). Although not all tissues were obtained due to safety implications for patients, they showed and definitized the myocardial change in the concrete. However, the assessment of the area of fibrosis or lipid vacuoles in 5 fields leaves an inaccurate possibility for objective calculation.

The other attractive point of this study is incremental effect of obesity on this histologic change. The authors tried to maximize the setting of myocardial dysfunction both with obesity and chronic pressure-overloading condition (aortic stenosis). GLS imaging is being accepted for objective tool for systolic cardiac function.[19][20] In general, they reasonably placed the proper characters with compatible assessment tool to show the correlation of imaging and histologic pattern.

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