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Evidence that obesity is an aggravating condition in hypertrophic cardiomyopathy (HCM) has been well established in several past studies. The risk of developing heart failure was shown to be clearly higher (1-3) in obese HCM patients, although no increased risk of death was found due to higher body-mass-index (BMI). Given that the mechanisms leading to HCM are manifold and still not fully understood, a better awareness of the conditions exacerbating HCM is therefore of great interest for risk stratification, prevention and therapy, but it may also contribute to a better fundamental understanding of the disease progression.

Cardiovascular MRI (CMR) studies evaluating global cardiac parameters have shown a strong correlation of BMI classification with left-ventricular (LV) mass (1, 4). However, beyond LV mass as a morphologic parameter, systolic function and morphology in HCM patients, once indexed to body size, were independent of BMI (1) or showed no stronger association with it than in normal subjects (4). CMR techniques have evolved considerably in the last decade, now allowing a much more detailed analysis of the myocardium in its function and tissue characteristics. Dynamic strain analyses of standard cine-MRI series have become possible via feature tracking, a tool that is now available as part of many commercial post-processing tools and that allows evaluation of larger cohorts including retrospective time spans.

In this JMRI issue, Yang et al. (5) report a CMR strain analysis aiming at potential correlations in more subtle functional impairments in 247 HCM patients divided into three BMI categories. The authors evaluated classic global left-ventricular (LV) function and morphology, but also strain and percentage of late-gadolinium enhancement (LGE) in 247 Chinese HCM patients. They were classified in 3 groups: obese, overweight and normal, with a specific BMI categorization used for Asian subjects. Interestingly, the proportions of these group assignments turned out to be about equal. As in the earlier studies, LV mass was associated with BMI, and in addition, the ratio of LV mass and end-diastolic volume was also correlated with BMI, indicating a change in LV geometry. The prevalence of LGE presence was not different across the three groups nor was the percentage of LGE tissue, although the latter was globally associated with LV functional deficits, independent of body size. It should be noted that presence of LGE was one of the criteria of HCM diagnosis and that it was found in an earlier study to correlate with occurrence of sudden cardiac death at 5 years in HCM patients (6).

Strain analysis, however, revealed relatively clear impairments of peak strains in all directions in obese compared with non-obese HCM patients. Although these peak strains were only slightly (but significantly) lower in the obese group, the authors hypothesize that they may indicate an insufficiency of the remodeling LV wall to deliver adequate systolic contraction, and that they may therefore be an early indication of the process eventually leading to heart failure. It remains unclear whether vascular alterations that may occur simultaneously played a role in this finding, since no analysis of coronary circulation was performed upon inclusion of HCM patients. Also, increased amounts of epicardial adipose tissue and myocardial triglyceride content have been shown to be associated with obesity and cardiac dysfunction (7) and may therefore play a role as well as myocardial energetics. The study by XX et al. also did not account for potentially higher prevalence of other conditions such as diabetes in the obese group, which precludes it from reporting a strict association of obesity alone with their findings of subtle functional impairments at this time. As an outlook, the impact of cardiac ectopic fat on these subtle functional impairments would be a very interesting complement. Future studies including a rest/stress myocardial perfusion MRI assessment, MR spectroscopy and a fat/water compartment analysis may answer these questions left unaddressed by this study in the future.

Importantly, the authors open towards potential therapeutic approaches, for which the diagnostic information on the relatively early subtle functional changes they found is important. They may indeed trigger therapy such as (adequately controlled) exercise sessions that could retard the onset of heart failure. New therapeutic approaches of obesity with GLP1 analogs and bi-agonist GIP-GLP1 are promising pharmacological approaches as well as the very recently proposed tirzepatide (8).

MRI strain measurements might then even be used for monitoring the effects of these therapeutic strategies.

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