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Perspective

Analysis of Obesity and Cardiovascular Disease

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DESCRIPTION

Obesity is on the rise globally. Obesity's significant link to cardiovascular illness has prompted a slew of research projects aimed at better understanding the relationship between obesity and the circulatory system and its repercussions. Obesity is linked to a higher risk of heart failure and mortality in general. This link is mostly related to obesity's impacts on cardiac structure and function, but it's also owing to the high prevalence of comorbid conditions including coronary artery disease, hypertension, sleep apnea, and diabetes mellitus. Cardiac Magnetic Resonance (MR) imaging is currently a well-recognized cardiac imaging tool that can aid either by precisely assessing the cardiomyopathy caused by chronic volume overload or by identifying the cause of the cardiomyopathy.

Concerning Left Ventricular (LV) remodeling, their study found, as have many previous studies using echocardiography, that LV mass was increased in obese subjects compared to controls, even though the values they found in obese subjects were within the range of normal values previously obtained in large series of asymptomatic subjects.

This rise in mass in obese participants was most likely caused by ventricular adaptation to a persistent increase in stroke volume, which was also found to be substantial. The decrease in LV mass following weight loss was related with a decrease in visceral fat mass and hyperleptinemia. However, because body mass index and End-Diastolic Volume (EDV) were independent predictors of LV mass regression in multivariate analysis. The second finding of the study is that peak filling rate (PFR [ml/s]) normalized by EDV (PFR/EDV) was shown to be lower and linked with a longer Time to Peak Filling Rate (TPFR) in obese patients compared to controls, indicating a substantial loss in diastolic function. However, analyzing this conclusion remains tricky, and it must be handled with caution. To investigate diastolic LV function, selected a quantitative method to the wall kinesis during fast diastolic filling after manually contouring endocardial margins of small axis. This method was first reported in radionuclide angiography before being used in a small number of cardiac MR imaging and electron beam computed tomography investigations. Variations seen between obese people and controls, or changes following weight loss, are more likely to be related to differences in preload condition than to true diastolic function alteration. Furthermore, when manual tracing is utilized to estimate the slope during fast filling, slight mistakes or changes in tracing can lead to big variations in slope determination and TPFR determinates, which may explain some of the large variability in normal values with high SD.

Totally automated boundary identification, which is now possible in cardiac MR, should be used at greater temporal resolution. However, after removing people with coronary artery disease, hypertension, and diabetes, researchers evaluated both the systolic and diastolic performance of 142 obese adults utilizing direct evaluation of cardiac characteristics and less load-dependent measures using tissue. They discovered that even after controlling for mean arterial pressure and LV mass, overweight people with no overt cardiac disease have subclinical abnormalities in LV function. Strain imaging in cardiac MR has also been extensively explored during systole in a large number of asymptomatic people.

Two cardiac MR imaging methods, which were just disclosed in the Journal, might also be highly beneficial in future obesity investigations. The first relates to the measurement of intramyocellular triglyceride build-up in the heart using a new spectroscopic approach. The second is a highly fascinating and new approach of assessing myocardial T1 mapping following gadolinium injection to estimate interstitial myocardial fibrosis. Both approaches should be highly useful in the study of obesity, because recent animal studies have revealed that excess fatty acids aid to damage cardiac myocytes by raising triglyceride content and the rate of apoptosis.

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