Letter to the Editor

Heart Failure With Preserved Ejection Fraction: Is it Due to Contractile Dysfunction?

To the Editor:

I read with interest the recent article on heart failure with preserved ejection fraction (HFPEF). The authors should be congratulated for their discussion of this interesting condition. They correctly emphasize that HFPEF is usually associated with concentric left ventricular hypertrophy in human and experimental studies. In clinical observational studies there is a 26–40% increase in left ventricular mass, but with a relatively normal end-diastolic volume compared with controls. Furthermore, it has been demonstrated repeatedly that there are significant systolic function abnormalities of strain, strain rate and tissue Doppler and that these correlate (r=0.81) with the severity of the diastolic dysfunction in both HFPEF and in heart failure with a reduced EF. Further, sarcomeric hypertrophic cardiomyopathies are disorders of the contractile proteins and display myocardial disarray; both these abnormalities would be expected to cause contractile dysfunction and yet the EF is usually normal or increased.

How can this apparent paradox of a preserved EF with widespread (ie, global) and significant contractile abnormalities be reconciled? One possibility is that an increase in end-diastolic left ventricular wall thickening would lead to augmented systolic thickening. In the presence of contractile dysfunction and concentric left ventricular hypertrophy, radial wall thickening (end-systolic thickness minus end-diastolic thickness) may be nearly normal. As the external volume of the heart changes little during the cardiac cycle, the endocardial displacement and EF will be normal. The pathogenesis of HFPEF may be explained by the combination of concentric LVH and contractile dysfunction even though EF is preserved. The assumption that a preserved EF means that systolic function is normal is flawed.

References


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