Letter by Di Bella et al Regarding Article, “Effect of Combined Systolic and Diastolic Functional Parameter Assessment for Differentiation of Cardiac Amyloidosis From Other Causes of Concentric Left Ventricular Hypertrophy”

To the Editor:

We read with great interest the article by Liu et al about the combined role of systolic septal longitudinal base-to-apex strain gradient and diastolic deceleration time of early filling in differentiating cardiac amyloidosis (CA) from other causes of concentric left ventricular hypertrophy (arterial hypertension, Fabry disease, and Friedreich’s ataxia).

Myocardial function is a complex physiological event because of radial, circumferential, and longitudinal deformation that can be explored by 2-dimensional (2D)-strain echocardiography, tagging MRI, and sonomicrometry. Recently, we have analyzed the epicardial and endocardial deformation by 2D-strain echocardiography in patients with transthyretin-related CA and hypertrophic cardiomyopathy. The main results of our study were (1) longitudinal, radial, and circumferential deformations are impaired both in patients with transthyretin-CA and hypertrophic cardiomyopathy with a preserved ejection fraction; and (2) impairment of epicardial circumferential strain is greater in transthyretin-CA than in hypertrophic cardiomyopathy.

There are several differences between our work and the work by Liu et al. Namely, different patient populations (transthyretin-CA versus hypertrophic cardiomyopathy in our work; light-chain amyloidosis versus other causes of symmetrical left ventricle hypertrophy in the work by Liu et al) have been compared, and different echocardiographic methodologies (evaluation of endocardial and epicardial longitudinal and circumferential and radial strain in our study; evaluation of longitudinal strain only in the work by Liu et al) have been used. Nevertheless, we think that, in addition to the systolic septal longitudinal base-to-apex strain gradient and diastolic deceleration time of early filling, also epicardial circumferential strain could aid in differentiating CA from other causes of left ventricle hypertrophy. Moreover, although CA is a heterogeneous disease caused by myocardial deposition of different proteins (eg, immunoglobulin light chain in amyloidosis, transthyretin in familial or senile amyloidosis, and serum amyloid A proteins in chronic inflammatory diseases), the different forms of systemic amyloidosis have in common some additional echocardiographic (eg, homogenous atioventricular valve thickening, atrial septum thickening, and sparkling/granular appearance of ventricular septum) and nonechocardiographic diagnostic features, such as low voltage of the QRS complexes at ECG, increase of N-terminal probrain natriuretic peptide, and even of troponin I or T.

We strongly appreciate the approach of Liu et al who use a combination of systolic and diastolic function parameters to diagnose CA. We think that several strain parameters, including epicardial circumferential strain, together with other routinely used laboratory and instrumental parameters should be considered, in clinical practice, to differentiate CA from other causes of left ventricle hypertrophy, thus reducing the need for magnetic resonance or nuclear medicine examinations.

Disclosures

None.

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Circ Cardiovasc Imaging, 2014;7:215
doi: 10.1161/CIRCIMAGING.113.001478
Circulation: Cardiovascular Imaging is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 1941-9651. Online ISSN: 1942-0080

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