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Editorial

Emerging Indicators of Left Atrial Function Evaluation Considering the Unique Characteristics of Hypertrophic Cardiomyopathy

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▸ See the article "Left Atrial Velocity Vector Imaging Can Assess Early Diastolic Dysfunction in Left Ventricular Hypertrophy and Hypertrophic Cardiomyopathy" in volume 31 on page 41.

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Left ventricular (LV) diastolic dysfunction is a common pathophysiology in hypertrophic cardiomyopathy (HCM). According to the 2016 guidelines, one should evaluate 4 variables to assess LV diastolic function: annular e′ velocity, average E/e′ ratio, left atrial (LA) maximal volume index, and peak tricuspid regurgitation (TR) velocity.[1] However, the suggested algorithm is sometimes limited in some of the disorders such as myocardial diseases including HCM, valve diseases, and atrial arrhythmias.

Yoon et al.[2] investigated the characteristics of LA function in patients with HCM compared to those with LV hypertrophy (LVH) and normal controls and whether strain rate (SR) was useful to differentiate their LA functional difference. The authors demonstrated that e′ velocity, E/e′ ratio, and late diastolic SR at the mid-left atrium were significantly different among the 3 groups (normal vs. LVH vs. HCM). Previous studies reported that LA function was significantly reduced in patients with HCM compared with controls.[3] Although current guidelines recommend evaluating similar parameters such as E/e′ ratio, LA volume index, peak TR rate, and pulmonary vein atrial inversion rate in patients with HCM, as in the general population,[1] these conventional parameters could provide information to underestimate or overestimate LV diastolic function in HCM. Also, we must consider the characteristics of HCM.

LA dysfunction is usually related to chronic structural remodeling owing to long-standing pressure and volume overload. Although elevated LV filling pressure causes LA dysfunction in HCM, LA dysfunction may occur as a myopathic process irrespective of loading conditions.[4] Additionally, one unique characteristic of HCM is that it is a genetic disorder. Although genetic contribution to LA function has not been well established, we previously reported that LA function (measured by a′ in echocardiography and LA total emptying fraction by cardiovascular magnetic resonance [CMR]) was significantly reduced in HCM patients with pathogenic or likely-pathogenic sarcomere gene mutation.[3] In this study, the LA total emptying fraction was significantly correlated to sarcomere gene mutation independent of amount of late gadolinium enhancement (LGE) in the LV, which suggests that LA dysfunction is a unique finding of HCM as an LA myopathy related to sarcomere gene mutations independent of LA loading conditions.[3] Another previous study also demonstrated that LA total emptying fraction was impaired in sarcomere mutation carriers associated with HCM without overt LVH compared with a control group.[5] Although LA myopathy is considered a

unique characteristic in HCM, LV fibrosis cannot be omitted as an important factor contributing to LA dysfunction in HCM, and a number of studies has identified a correlation between LGE and LA function.[6]

LA size has been an established surrogate marker for the severity of LV diastolic dysfunction. However, LA volume is not sensitive in the early phase of LV diastolic dysfunction because LA remodeling is a time-dependent process. Accordingly, LA function may provide additional value in assessing diastolic function in the early stage of LV diastolic dysfunction.[7] LA phasic function assessed by speckle tracking echocardiography (STE) is less affected by loading conditions, and measurement of the LA strain and SR using STE has been established to be a feasible technique to evaluate LA function.[7] Previously, Her et al.[8] demonstrated that LA global strain by STE was significantly correlated with degree of histologic LA fibrosis but not LA volume index in patients with mitral valve disease. Peak LA strain and SRs using STE have been used to quantitate global and regional LA contractility.[9] Furthermore, Lee et al.[6] presented LA reservoir strain as a useful index to categorize LV diastolic dysfunction and predict heart failure events in HCM. Recently, CMR-feature tracking analysis for quantification of global and regional LA functions has been validated as a feasible and reproducible tool for assessment of LA function in patients with HCM.[3][10]

In conclusion, echo-Doppler assessment of LV diastolic function is limited in many ways in patients with HCM, and more promising parameters considering the unique characteristics of HCM are needed. Therefore, the report by Yoon et al.[2] can provide an insight for assessment of LV diastolic function or LA myopathic process by phasic LA function by tissue tracking echocardiography in HCM.

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Conflict of Interest

The author has no financial conflicts of interest.

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