Editorial Comment

Basal septal hypertrophy in hypertension; about time to introduce an objective and reproducible quantification

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ubclinical left ventricular (LV) dysfunction, abnormal LV geometry, such as LV hypertrophy (LVH) and concentric remodeling, and left atrial remodeling are the key components of hypertensive heart disease by increased arterial load [1]. Although the association between the duration and severity of hypertension and abnormal LV geometry is well known, other factors, such as genetic, metabolic and environmental factors may be also equally important [2]. LVH may be eccentric or concentric, or only involve interventricular septum either the whole septum (isolated septal hypertrophy) or the basal/upper part (Fig. 1). The regional myocardial function assessed by strain is clearly decreased in LV segments with basal septal hypertrophy (BSH). Traditionally, BSH, also termed sigmoidal septum, is visually assessed by echocardiography and is not included in the conventional classification of LVH types. International guidelines on cardiac chamber quantification by echocardiography in adults, recommend measurement of interventricular septum immediately below the septal bulge assessed by linear method because of the risk of overestimation of the true LV mass [3,4], or even in mid-septal level, which has best agreement with cardiac magnetic resonance (CMR) in patients with aortic stenosis and BSH [5]. Of note, three-dimensional CMR incorporates regional differences in LV wall thickness, and therefore provides more accurate measurements.

In some patients, particularly in elderly women, patients with aortic stenosis, systemic hypertension and increased arterial stiffness, or individuals with smaller LV cavity dimensions and high-normal LV ejection fraction, BSH may increase the risk of dynamic LV obstruction, even in the absence of underlying hypertrophic obstructive cardiomyopathy (Saeed et al. unpublished observation).

Apart from the traditional visual assessment, BSH can be also defined by a somehow more quantitative method of basal-to-mid-septal thickness ratio of more than 1.4 in either parasternal long-axis view or apical four-chamber view. This method, however, has limited reproducibility. The use of a single echocardiographic view to measure basal-to-mid septal ratio may not be appropriate as some patients with hypertension or metabolic syndrome may have a proportionate septal hypertrophy without a typical bulging on parasternal long-axis view but show a prominent mid-septal bulging on apical four-chamber view (Fig. 2).

The clinical significance and prognostic value of BSH were not known for a long time, probably because of lack of a more robust, objective and reproducible method for characterization and quantification of BSH. However, the data on the clinical significance of BSH is now emerging. Most recently, an association between BSH and impaired LV function, and LV and left atrial remodeling was documented in hypertensive patients [6]. The authors postulated that BSH might be an early marker of hypertension-induced LV remodeling. Hence, this implies a more detailed assessment and quantification of BSH with more objective and reproducible methods.

In this issue of the Journal of Hypertension, Marciniak et al. [7] introduced a novel method for assessing BSH. The LV endocardium curvatures were used for characterization and quantification of BSH in a cohort of 220 individuals including 161 hypertensive patients and 59 healthy controls, with a 20% prevalence of BSH. The average septal curvature (ASC) is calculated from a well established and widely available endocardial segmentation tool, which is used to assess strain by echocardiography. Patients with BSH are more concave in the BSH segment and reports a more negative ASC compared with patients without BSH. The ASC method might look complicated at the first look but can be performed fully automatized and seemed to be a robust and objective echocardiographic measure of BSH with good reproducibility. Given its higher reproducibility compared with the traditional visual assessment of BSH or the quantitative method of basal-to-mid-septal thickness ratio, easy interpretation and the potential for implementation in 3D-imaging modalities, ASC seem to be a promising
FIGURE 1 Echocardiographic images from hypertensive patients: Panel (a) shows a parasternal long-axis view and panel (b) shows an apical four-chamber view at end-diastole showing normal left ventricular wall thicknesses and chamber dimensions. Panel (c) demonstrates a parasternal long-axis view at end-diastole and shows asymmetric septal hypertrophy but normal posterior wall thickness. Panel (d) displays a parasternal long-axis view and shows typical basal septal hypertrophy (BSH) with normal mid-septal thickness (1) but septal bulging (2) with a basal-to-mid septal thickness ratio of more than 1.5. Panel (e) is Bull’s eye plot from the same patient showing reduced longitudinal strain of the basal septal segments (−8 and −10%), with an average global longitudinal strain (GLS) of −16%. Panel (f) displays a parasternal long-axis view at end-diastole showing typical BSH (basal-to-mid-septal thickness ratio >1.5) and (g) is Bull’s eye plot from the patient in (f) showing normal global GLS, but reduced regional strain at the basal septal segment of −11%. BSH, basal septal hypertrophy.

FIGURE 2 Echocardiographic images from a patient with metabolic syndrome (hypertension, diabetes and obesity). Panel (a) shows a parasternal long-axis view with concentric left ventricular hypertrophy with a proportionate hypertrophy of the whole septum, but no basal septal hypertrophy (BSH) or septal bulge. Panel (b) demonstrates an apical four-chamber view from the same patient and shows localized thickening/bulging of mid-septum (arrows).
echocardiographic marker for assessment of BSH. Similarly, because of its closer association with structural and functional changes in hypertensive heart, ASC may be a superior marker for use in routine clinical care compared with the traditional ones. These interesting results are directly relevant for daily clinical practice to detect unrevealed hypertension, and now need to be confirmed in larger hypertensive populations with and without BSH. Whether BSH has a genetic background, and is partly related with hypertrophic obstructive cardiomyopathy [8], should also be focused in future research.

ACKNOWLEDGEMENTS

Conflicts of interest
There are no conflicts of interest.

REFERENCES


