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Research Article

The septal bulge—an early echocardiographic sign in hypertensive heart disease

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Abstract

Patients in the early stage of hypertensive heart disease tend to have normal echocardiographic findings. The aim of this study was to investigate whether pathology-specific echocardiographic morphologic and functional parameters can help to detect subclinical hypertensive heart disease. One hundred ten consecutive patients without a history and medication for arterial hypertension (AH) or other cardiac diseases were enrolled. Standard echocardiography and two-dimensional speckle-tracking-imaging analysis were performed. Resting blood pressure (BP) measurement, cycle ergometer test (CET), and 24-hour ambulatory BP monitoring (ABPM) were conducted. Patients were referred to ''septal bulge (SB)'' group (basal-septal wall thickness $\geq$2 mm thicker than mid-septal wall thickness) or ''no-SB'' group. Echocardiographic SB was found in 48 (43.6%) of 110 patients. In this SB group, 38 (79.2%) patients showed AH either by CET or ABPM. In contrast, in the no-SB group (n = 62), 59 (95.2%) patients had no positive test for AH by CET or ABPM. When AH was solely defined by resting BP, SB was a reasonable predictive sign for AH (sensitivity 73%, specificity 76%). However, when AH was confirmed by CET or ABPM the echocardiographic SB strongly predicted clinical AH (sensitivity 93%, specificity 86%). In addition, regional myocardial deformation of the basal-septum in SB group was significantly lower than in no-SB group (14 $\pm$ 4% vs. 17 $\pm$ 4%; $P < .001$). In conclusion, SB is a morphologic echocardiographic sign for early hypertensive heart disease. Sophisticated BP evaluation including resting BP, ABPM, and CET should be performed in all patients with an accidental finding of a SB in echocardiography. J Am Soc Hypertens 2016;10(1):70–80.

Keywords: Septal bulge; hypertension; blood pressure monitoring; echocardiography; heart disease.

Introduction

Arterial hypertension (AH) has been well recognized as a common risk factor for cardiovascular disease.[1–5] However, a great number of early hypertensive patients never experience any symptoms,[6] and the awareness rate of AH remains low in general. Thus, early diagnosis of AH remains a challenge, particularly in a subclinical population.

It is known that left ventricular (LV) hypertrophy with different remodeling patterns is one of the major cardiac manifestations of hypertensive heart disease, and echocardiographic LV hypertrophy could be detected in 20% to 40% of patients with AH.[7–10] However, there are often no specific echocardiographic features for hypertensive patients at the early stage of disease.[11] Previous echocardiographic studies have described asymmetric septal hypertrophy with a localized septal thickening at the basal-mid

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portion in patients with hypertrophic cardiomyopathy[12,13] or aortic valve stenosis.[14,15] Basal-septal hypertrophy may also occur in a subset of older normal subjects, with normal wall thickness (WT) elsewhere, and is considered to be an age-related anatomic variant.[16,17] This morphologic echocardiographic sign is termed as septal bulge (SB), sigmoid septum, or discrete upper septal thickening or knuckle.[18] A large community-based population study reported that SB was documented frequently in elderly individuals with higher systolic blood pressure (SBP). It was shown that the overall prevalence of SB was 1.5% and was markedly higher (18%) in the eighth decades of life.[18] Although pathologic and echocardiographic observations have indicated that SB is a structural response in hypertensive patients,[19–21] the nature and significance of the SB in subclinical AH was never investigated.

In addition, despite the fact that BP can be easily measured, AH sometimes cannot be diagnosed due to the underreported BP reading in the casual or self-measured BP measurement.[22] BP measurement with appropriate tools is essential to diagnosing AH early as well as to guiding AH management. It has been shown that, besides resting BP measurement in the office, AH could be clinically diagnosed by 24-hour ambulatory BP monitoring (ABPM) as well as exercise stress test in some resting normotensive individuals.[1,23–25]

In the present study, we prospectively screened a subset of population without history and medication of AH as well as other cardiac diseases. A sophisticated clinical assessment for AH was conducted in these patients, including resting BP measurement, cycle ergometer test (CET) and ABPM. We investigated the prevalence of echocardiographic SB and its relationship with AH. We speculate that SB might represent an earlier structural adaption in response to pressure overload before LV concentric hypertrophy in AH.

Methods

This study was initiated in February 2013. The study subjects were selected from a group of 8208 consecutive patients referred to the echocardiographic laboratory in the University Hospital of Würzburg between February 2013 and February 2014. The enrollment criteria included (1) no history of AH and any heart diseases and (2) no history of medications for AH. A total of 154 eligible patients were invited to take part in the AH screen study. Forty-four patients, who refused to receive 24-hour ABPM or were unable to perform CET, were excluded. Finally, 110 patients completed this study. The screening flowchart of the study is shown in Figure 1. Standard echocardiographic examination was performed in all patients. Resting electrocardiography (ECG), resting manual BP measurement, ABPM, and CET (ECG/BP) were performed on the same day in all patients. The study was approved by Local Ethics Committee at the University of Würzburg and conducted in accordance to the Declaration of Helsinki. Written informed consent was obtained from all patients or their guardians.

Brachial BP was measured at rest. Patients were seated quietly for at least 5 minutes in a chair before the measurement. At least 2 measurements were made in each patient. Afterward, CET was performed on an electrically braked cycle ergometer (Ergometrics 900, Ergoline, Bitz, Germany) with incremental loads at 50/75/100/150 Watts (each stage for 3 minutes), until exhaustion. Brachial BP was measured every 3 minutes interval during testing. The testing was stopped when the targeted age-adjusted heart rate was achieved, or when SBP increased to more than 250 mm Hg, or when chest pain or arrhythmia occurs.

After CET, 24-hour ABPM was performed using a digital oscillometric blood pressure device (Mobil-O-Graph NG version 20, I.E.M., Germany). BP was measured every 15 minutes during the waking period (8 AM–12 PM) and every 30 minutes during the sleeping period (12 PM–8 AM). The test was considered satisfactory when at least 70% of the BP readings were valid.

AH was suggested by resting BP: SBP $\geq$ 140 mm Hg or diastolic BP (DBP) $\geq$ 90 mm Hg based on the mean of 2 or more properly measured seated BP.[1,3] Patients were defined as AH with positive CET results (SBP $\geq$ 200 mm Hg at 50, 75, or 100 Watts[26]) or positive ABPM results (mean SBP $>$ 135 mm Hg or DBP $>$ 85 mm Hg during the waking period and SBP $>$ 120 mm Hg or DBP $>$ 70 mm Hg during the sleeping period[1]) or both.

The classification of high BP was defined as: normal, SBP $<$ 120, and DBP $<$ 80 mm Hg; high-normal, SBP = 120–139 or DBP = 90–99 mm Hg; stage 1 hypertension, SBP = 140–159 or DBP = 90–99 mm Hg; stage 2 hypertension, SBP $\geq$ 160 or DBP $\geq$ 100 mm Hg.[1,3]

A standard transthoracic echocardiographic examination was performed (GE, Vingmed, Horten, Norway). Standard two-dimensional (2D) images and Doppler recordings were obtained according to guidelines.[27] All offline measurements were performed in a remote workstation (EchoPAC version 112, GE, Horten, Norway). LV end-diastolic dimension (LVEDD), end-diastolic WT of the mid septum (IVSd) and the posterior wall (LVPWd), as well as left atrial end-systolic diameter were measured in the parasternal LV long axis view. The maximum basal- and mid-septal WT was measured in the LV parasternal long-axis view. Basal-septal to mid-septal WT ratio (WT_Ratio) was calculated as the basal-septal WT divided by the mid-septal WT. Relative WT = (IVSd + LVPWd)/LVEDD was calculated. LV mass indexed for height to allometric power of 2.7 was estimated by LV cavity dimension and WT at end diastole[27]: LV mass (g) = $0.8 \times [1.04 \times (\text{LVEDD} + \text{LVPWd} + \text{IVSd})^3 - (\text{LVEDD}^3)] + 0.6$. LV ejection fraction was measured with the biplane Simpson method in the apical 4- and 2-chamber views. Mitral annular plane systolic