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Research Article

Does masked hypertension impact left ventricular deformation?

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Abstract

Our aim was to compare left ventricular (LV) deformation in subjects with masked hypertension (MH) to normotensive and sustained hypertensive patients. This cross-sectional study included 185 untreated subjects who underwent 24-hour ambulatory blood pressure (BP) monitoring and complete two-dimensional echocardiographic (2DE) examination including multilayer strain analysis. MH was diagnosed if clinic BP was normal (<140/90 mm Hg), and 24-hour BP was increased (≥130/80 mm Hg). 2DE LV longitudinal and circumferential strains gradually and significantly decreased from normotensive controls across MH individuals to sustained hypertensive patients. 2DE radial strain was not different between groups. 2DE longitudinal and circumferential endocardial and midmyocardial layer strains progressively decreased from normotensive control to sustained hypertensive individuals. Longitudinal and circumferential epicardial layer strains were lower in sustained hypertensive patients than in normotensive controls. Clinic and 24-hour systolic BP were associated with 2DE LV longitudinal endocardial strain, midmyocardial strain, and 2DE circumferential endocardial strain in the whole-study population independent of LV structure and diastolic function. MH significantly affect LV deformation assessed by 2DE traditional strain and 2DE multilayer strain. Clinic and 24-hour systolic BP were associated with LV mechanics evaluated with comprehensive 2DE strain analysis independent of LV structure and diastolic function. J Am Soc Hypertens 2016;10(9):694–701.

Keywords: Arterial hypertension; function; left ventricular structure; mechanics.

Introduction

The term ''masked hypertension'' (MH), which refers to normal office blood pressure (BP) values and increased out-of-office BP, has been proposed by Pickering et al. in 2002.[1] The influence of MH and other BP patterns (normotension, white-coat hypertension, and sustained hypertension) on target organ damage and cardiovascular morbidity and mortality has been largely investigated.[2–5] The authors agree about the influence of MH on left ventricular (LV) hypertrophy.[2–5] Our recently published meta-analysis that included 4884 untreated subjects (2467 normotensive, 776 MH, and 1641 sustained hypertensive individuals) showed a progressive increase of LV mass from normotensive throughout MH to hypertensive subjects.[6] The majority of authors concur about the negative impact of MH on cardiovascular events and mortality.[7,8] The Finn-Home Study showed that MH had a significantly higher risk of cardiovascular events and all-cause mortality after adjustment for age, sex, and office BP than normotension.[9] However, MH lost its predictive significance after adjustment for home BP or concomitant cardiovascular risk factors.

Conflict of interest: None.

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The influence of MH on LV mechanics has not been investigated so far. However, previous studies demonstrated significant impairment of LV deformation in hypertensive patients.[10–12] The current guidelines[13] proposed the evaluation of LV longitudinal strain in hypertensive population because of its high predictive value that is even higher than for LV ejection fraction (EF).[14] Recent analysis showed significant difference in layer-specific longitudinal strain in hypertensive patients with LV hypertrophy in comparison with normal LV geometry subjects.[15] In addition, Lee et al[16] for the first time demonstrated significant independent prognostic value of epicardial LV longitudinal strain in regularly treated hypertensive patients.

Due to all aforementioned reasons, we decided to conduct this study in which we investigated LV deformation using traditional two-dimensional and multilayer strain in patients with normal BP, MH, and sustained hypertension.

Methodology

This cross-sectional study included 185 untreated subjects of similar age referred to our outpatient clinic due to ambulatory BP monitoring from January 2014 to October 2015. Patients included in this study are referred to our outpatient clinic from general practitioners, cardiologists, and as a part of primary prevention program. Subjects with heart failure, coronary artery disease, previous cerebrovascular insult, atrial fibrillation, congenital heart disease, valvular heart disease, neoplastic disease, cirrhosis of the liver, kidney failure or endocrine diseases including type II diabetes mellitus, and patients with unsatisfied echocardiographic images were excluded from the study. Stress test was performed in 15 patients due to suspect coronary artery disease based on symptoms or electrocardiographic changes. Three tests were positive for decreased coronary reserve, and these patients were excluded from the further study.

Anthropometric measures and laboratory analyses were taken from all the subjects included in the study. Body mass index (BMI) and body surface area (BSA) were calculated for each patient. The study was approved by the local Ethics Committee, and written informed consent was obtained from all the participants.

Clinic BP Measurement and 24-Hour Ambulatory BP Monitoring

Clinic arterial BP values were obtained by E-mega aneroid manometer (Riester, Jungingen, Germany) in the morning hours by measuring the average value of the two consecutive measurements in the sitting position. BP was obtained in at least two separate occasions.

All the participants underwent a 24-hour BP monitoring. The noninvasive 24-hour ambulatory BP monitoring was performed by Schiller BR-102 plus system (Schiller AG, Baar, Switzerland) according to the current guidelines.[17] The device was programmed to obtain BP readings at 20-minute intervals during the day (7:00 AM–11:00 PM) and at 30-minute intervals during the night (11:00 PM–07:00 AM). The recording was then analyzed to obtain a 24-hour average systolic and diastolic BP and heart rates.

MH was defined as an normal clinic BP (systolic BP $<$ 140 mm Hg and diastolic BP $<$ 90 mm Hg) measured in at least two separate occasions associated with a 24-hour ambulatory SBP $>$ 130 mm Hg and/or DBP $>$ 80 mm Hg, whereas those with sustained hypertension had an elevated clinic SBP of 140 mm Hg together with a 24-hour ambulatory SBP $\geq$ 130 mm Hg or DBP $\geq$ 80 mm Hg.

Echocardiography

Echocardiographic examination was performed by a Vivid 7 ultrasound machine (GE Healthcare, Horten, Norway). Echocardiographic examination was usually performed within 2–3 weeks after 24-hour BP monitoring, but not longer than 1 month.

The values of all two-dimensional echocardiographic (2DE) parameters were obtained as the average value of three consecutive cardiac cycles. LV diameters, interventricular septum, and relative wall thickness were determined according to the recommendations.[18] LV EF was assessed by the biplane method. LV mass was calculated by using the formula of the American Society of Echocardiography[18] and indexed for BSA. Left atrial volume was measured by the biplane method in four- and two-chamber views and indexed for BSA. Transmitral Doppler inflow and tissue Doppler velocities were obtained according to the guidelines.[19]

2DE LV Strain Analysis

2DE strain imaging was performed by using three consecutive cardiac cycles in apical (four-chamber, long axis, and two-chamber view) and parasternal short-axis view (at the level of papillary muscles).[20] The frame rate ranged between 50 and 70 Hz. The commercially available software 2DE Auto LVQ software (EchoPAC 112, GE Healthcare) was used for the 2DE strain analysis.

The 2DE strain rates were calculated by averaging all the values of the regional peak longitudinal strain and strain rate obtained in two-chamber, long axis, and four-chamber apical views. 2DE circumferential strain rates, as well as 2DE radial strain and strain rates, were assessed as the average of the LV six regional values measured in the parasternal short-axis view, at the level of papillary muscles. 2DE global longitudinal and circumferential strains were calculated as average of all three layers (endocardial, midmyocardial, and epicardial).

Multilayer longitudinal and circumferential strains were determined by modified 2DE strain software (Q-analysis).