Variations in Cardiac Diastolic Function in Hypertensive Patients with Different Left Ventricular Geometric Patterns

Peng QU, Yanchun DING, Daozi XIA, Hongyan WANG, and Xiaohong TIAN

To evaluate the alteration of cardiac function in hypertensive patients with different left ventricular geometric patterns. Echocardiography was used to study left ventricular geometry and cardiac diastolic function in 117 cases of essential hypertension, with 45 normal cases as controls. Echocardiographic data were used to calculated the left ventricular mass index (LVMII) and relative wall thickness (RWT), which values in turn were used to divide the subjects into four groups. The left atrial dimension of the group, with the exception of these hypertensives who showed normal geometry, was larger than that of the control group. The damage of peak of E velocity, peak of A velocity, E/A and the slope between the E and F points (E to F slope) were greater than in hypertension than in the control group. The concentric hypertrophy group and eccentric hypertrophy group suffered more serious damage of left ventricular diastolic function than the concentric remodeling group, and damage of left ventricular diastolic function in the concentric remodeling group was greater than that in the normal geometry group. The degree of cardiac diastolic function damage differed among patients with different left ventricular geometric patterns, when the cardiac structure was changed, the degree of cardiac diastolic function damage increased. (Hypertens Res 2001; 24: 601–604)

Key Words: hypertension, left ventricular geometry, diastolic function

Introduction

The left ventricle adapts to sustained hypertension through left ventricular geometric alteration and changes in cardiac function (1, 2). The relation between the alteration of left ventricular geometry and diastolic function in hypertensive patients has attracted much interest over the years. In the present study, we used echocardiographic data to classify left ventricular geometric patterns and determined the cardiac diastolic function by Echo-Doppler examination and echocardiography. The relation between left ventricular geometric alteration and cardiac diastolic function were studied.

Methods and Subjects

Study Population

One hundred and seventeen hypertensive patients and forty-five sex and age-matched normotensive controls participated in this study. Informed consent for all procedures was obtained from each patient. All hypertensive patients were untreated or had been free from medication for at least 2 weeks before the investigation. Blood pressure was determined by sphygmomanometer before Echo-Doppler examination.

Echocardiographic Measurement

Echocardiographic studies were carried out using Ultramark 9 echocardiograph with a 2.5 MHZ transducer (ATL, Inc.,

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Table 1. Clinical and Geometric Characteristics in Normotensive Controls and Hypertensive Patients

<table>
<thead>
<tr>
<th></th>
<th>Control (n=45)</th>
<th>NG (n=37)</th>
<th>CR (n=30)</th>
<th>EH (n=20)</th>
<th>CH (n=37)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>51.9±6.7</td>
<td>54.6±8.2</td>
<td>55.2±8.3</td>
<td>56.1±9.4</td>
<td>55.6±10.5</td>
</tr>
<tr>
<td>Male/Female</td>
<td>22/23</td>
<td>20/17</td>
<td>15/15</td>
<td>12/8</td>
<td>16/14</td>
</tr>
<tr>
<td>Duration (years)</td>
<td>3.7±4.6</td>
<td>5.7±6.5</td>
<td>7.3±8.1</td>
<td>6.5±5.5</td>
<td></td>
</tr>
<tr>
<td>BSA (m²)</td>
<td>1.80±0.18</td>
<td>1.81±0.16</td>
<td>1.81±0.14</td>
<td>1.85±0.16</td>
<td>1.82±0.19</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>71±7</td>
<td>71±11</td>
<td>73±10</td>
<td>73±9</td>
<td>73±12</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>120±9</td>
<td>163±13*</td>
<td>175±15*</td>
<td>165±15*</td>
<td>169±15*</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>72±5</td>
<td>97±12*</td>
<td>98±12*</td>
<td>99±13*</td>
<td>102±12*</td>
</tr>
<tr>
<td>LAD (cm)</td>
<td>2.58±0.25</td>
<td>2.90±0.46</td>
<td>3.02±0.43*</td>
<td>3.15±0.51*</td>
<td>3.19±0.50*</td>
</tr>
<tr>
<td>LVEDD (cm)</td>
<td>4.65±0.18</td>
<td>4.71±0.16</td>
<td>4.30±0.27*</td>
<td>5.39±0.25*</td>
<td>4.69±0.33*</td>
</tr>
<tr>
<td>LVESD (cm)</td>
<td>3.15±0.23</td>
<td>3.35±0.21*</td>
<td>3.10±0.19*</td>
<td>4.13±0.28*</td>
<td>3.48±0.31*</td>
</tr>
<tr>
<td>LVM (g/m²)</td>
<td>95.2±11.08</td>
<td>102.9±8.2*</td>
<td>103.7±11.2*</td>
<td>163.8±24.0*</td>
<td>154.5±28.6*</td>
</tr>
<tr>
<td>RWT</td>
<td>0.40±0.03</td>
<td>0.42±0.02</td>
<td>0.51±0.04*</td>
<td>0.43±0.02*</td>
<td>0.55±0.05*</td>
</tr>
</tbody>
</table>

NG, normal geometry; CR, concentric remodeling; EH, eccentric hypertrophy; CH, concentric hypertrophy. *p<0.05 vs. control; △p<0.05 vs. NG; □p<0.05 vs. CR; ★p<0.05 vs. ET.

Seattle, USA) according to Penn convention (3). The interventricular septal end-diastolic thickness (IVSTd), left ventricular posterior wall end-diastolic thickness (LVPWTd), left ventricular end-diastolic dimension (LVEDD) and left atrial dimension (LAD) were measured by averaging five cardiac cycles. Left ventricular mass (LVM) was estimated using the formula from Devereux and Reichek (4): 

\[ LVM(g) = 1.04 \times \left( (LVEDD + LVPWTd + IVSTd)^3 - LVEDD^3 \right) - 13.6. \]

The left ventricular mass index (LVMI) is taken as LVM/BSA, where BSA (body surface area) (g/m²) = 71.84×height (cm)⁰⁷²⁵×weight (kg)⁰⁴²⁵×10⁻⁴. The relative wall thickness (RWT) was calculated as follows: 

\[ RWT = \frac{(LVPWTd + IVSTd)}{LVEDD} \].

A normal left ventricular mass index was defined as 125 and 120 g/m² (6) in males and females, respectively. A partition value of 0.45 for relative wall thickness was used for both males and females. All patients were divided into four groups based on LVMI and RWT according to the method of Ganau et al. (7). Patients with increased LVMI and increased RWT were considered to have concentric hypertrophy, and those with increased LVMI and normal RWT were considered to have eccentric hypertrophy. Those with normal LVMI and increased or normal RWT were considered to have concentric remodeling or normal geometry, respectively.

LV Diastolic Function Determination

Two-dimensionally guided M-mode tracings of mitral valve movement was recorded, and the EF slope was determined. Pulse-wave Doppler spectra of mitral inflow were recorded from an apical four chambers view, with the sample volume placed near the tips of the mitral leaflets and adjusted to the position where velocity was maximal and the flow pattern was laminar. The peaks of the E and A velocities were determined, and the value of E/A was obtained.

Statistical Analysis

All values are expressed as the means ± SD. Differences between and within groups of normotensive and hypertensive subjects were assessed using one-factor ANOVA with post hoc comparisons by Fisher’s protected least significant difference test. Correlation coefficients were obtained using linear regression (the least squares method). A probability less than 0.05 was considered to indicate statistical significance. SPSS statistical software, version 8.0 was used to perform the analysis.

Results

Characteristics of Study Subjects and Geometry of Left Ventricle

Table 1 summarizes the clinical profiles and geometric characteristics in normotensive controls and hypertensive patients. There were no differences in sex, BSA, or heart rate (HR) among the five groups. There were no significant differences in SBP or DBP among the four different LV geometry patterns.

Cardiac Diastolic Function and Left Ventricular Geometry

Table 2 shows the variations in cardiac diastolic function among hypertensive patients with different left ventricular geometric patterns. In hypertensive patients with the normal left ventricular geometry pattern, the peak of E velocity was already decreased and the peak of A velocity increased, and thus the value of E/A was increased. These changes were more significant in eccentric and concentric hypertrophic patients. The E to F slope decreased gradually and was significantly different among the normal geometry, concentric re-
Table 2. Left Ventricular Diastolic Functional Parameters in Normotensive Controls and Hypertensive Patients with Different LV Geometries

<table>
<thead>
<tr>
<th></th>
<th>Control (n=45)</th>
<th>NG (n=37)</th>
<th>CR (n=30)</th>
<th>EH (n=20)</th>
<th>CH (n=37)</th>
</tr>
</thead>
<tbody>
<tr>
<td>E velocity (m/s)</td>
<td>0.82±0.08</td>
<td>0.70±0.12*</td>
<td>0.67±0.14*</td>
<td>0.60±0.11*</td>
<td>0.54±0.11*</td>
</tr>
<tr>
<td>A velocity (m/s)</td>
<td>0.50±0.08</td>
<td>0.61±0.14*</td>
<td>0.62±0.11*</td>
<td>0.71±0.13*</td>
<td>0.63±0.13*</td>
</tr>
<tr>
<td>EA/</td>
<td>1.66±0.23</td>
<td>1.18±0.26*</td>
<td>1.11±0.28*</td>
<td>0.86±0.14*</td>
<td>0.89±0.23*</td>
</tr>
<tr>
<td>EF slope (mm/s)</td>
<td>102.1±18.2</td>
<td>86.9±22.4*</td>
<td>74.8±17.3*</td>
<td>66.0±13.17*</td>
<td>53.8±21.1*</td>
</tr>
</tbody>
</table>

NG, normal geometry; CR, concentric remodeling; EH, eccentric hypertrophy; CH, concentric hypertrophy. *p<0.05 vs. control; ▲p<0.05 vs. NG; ▲p<0.05 vs. CR; *p<0.05 vs. ET.

Fig. 1. The significant inverse relation between LVMl and E to F slope in hypertensive patients.

The Relation between E to F Slope and LVMl

Figure 1 shows the relation between E to F slope and LVMl in hypertensive patients. There was a significant inverse relation between LVMl and E to F slope (y = -0.3052x + 110.41, R²=0.18, p < 0.01).

Discussion

The present study demonstrated that left ventricular diastolic function was damaged in the early stage of hypertension. As hypertension continued to develop, and particularly when accompanied with changes in left ventricular geometric patterns, the decrease in cardiac diastolic function became more significant. The cardiac diastolic function was inversely related to LVMl and RWT. The damage of the E to F slope and of the EA ratio was the most severe in concentric hypertrophy group among the four left ventricular geometric patterns of hypertensive patients, and progressive decreases in the EA ratio and the E to F slope were seen in eccentric hypertrophy, concentric remodeling and normal geometric patients with hypertension.

A number of studies have also reported that cardiac diastolic function is damaged at the early stage of hypertension (8–10). In the present study, it was not clear why patients with different left ventricular geometric patterns showed different degree of cardiac diastolic function damage. Factors that might contribute to this association include changes of the interstitial substances of the myocardium, abnormal transmission of intracellular calcium ions in myocytes and delayed relaxation of myocytes induced by long-term increased in blood pressure and peripheral resistance (8, 9, 11, 12).

Along with the development of hypertension, myocytes gradually became hypertrophic, and wall thickness increased. Because of the heterogeneity of myocyte hypertrophy, the left ventricular geometry changed. The accumulation of interstitial collagen and the myocyte hypertrophy resulted in an increase in myocardium rigidity and a decrease in the compliance. On the other hand, the imbalance of microvessel to myocyte often occurred during the hypertrophy development. Myocardial ischemia has been shown to increase the heterogeneity of myocardial contraction and relaxation with respect to both time and space (13, 14). Brutsaert et al. (15) emphasized that the geometric change due to the myocardial hypertrophy, the different wall segment hypertrophy, the interstitial fibrosis, the changes of myocardial contraction and conjunction between myocytes, all might contribute to the damage of cardiac diastolic function during the development of hypertrophy.

The present study also demonstrated that the left atrial dimension was increased in concentric hypertrophy, eccentric hypertrophy, concentric remodeling and normal geometric patients with hypertension. The enlargement of the left atria preceded the development of LVH and was increased with the LVMl increase (14). In hypertensive patients, enlargement of the left atria might be the only precursor to the change in the left ventricle, and it is known to be on early occurrence in hypertensive myocardial damage (16). These findings suggest that cardiac remodeling involved not only the left ventricle, but also the left atria.

In conclusion, our study suggests that the degree of cardiac diastolic function damage differed among patients with different left ventricular geometric patterns, when the cardiac structure was changed, the degree of cardiac diastolic function damage increased.
References


