Clinical Significance of Pulmonary Regurgitation Signal on Doppler Echocardiography in Patients with Hypertensive Left Ventricular Hypertrophy

Yasunori Ozawa, Sachihiko Nobuoka, Nobuyuki Mitsuya and Fumihiko Miyake

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Abstract

The purpose of this study was to assess the clinical significance of pulmonary regurgitation (PR) signal on Doppler echocardiography in patients with hypertensive left ventricular (LV) hypertrophy. Fourteen patients with hypertensive LV hypertrophy who showed PR signal on Doppler echocardiography were included (HHD group). Additional 17 patients with no evidence of heart or respiratory disease except for PR signal were included as healthy control (Control group). On Doppler echocardiography, maximum flow velocity (Vmax), end-diastolic flow velocity (Vmin), duration time (DT), ratio of decay velocity to time (Decay), and time to reach pressure difference of 1/2 (PHT) of the PR signal were measured and compared between the two groups. And the correlation between these parameters and LVM was examined. 1) There was no significant difference in Vmax, Vmin, DT, and Decay between the two groups. 2) PHT was significantly longer in the HHD group (P<0.01). 3) In the HHD group, significant positive correlations were observed between LVM and Vmax, and between LVM and Decay (r=0.592, P<0.05 and 0.738, P<0.01, respectively). Such correlations were not observed in the Control group. We propose that the elevation of the pulmonary artery pressure and decrease of the right ventricular compliance in patients with hypertensive LV hypertrophy caused by a decrease of the LV compliance, thereby generating a large, continuous pressure difference between the pulmonary artery and the right ventricle through the diastolic phase. An analysis of PR signal could be useful for evaluating hemodynamic change in patients with hypertension.

Key words

Hypertension, Left ventricular hypertrophy, Pulmonary regurgitation, Doppler echocardiography, Pulmonary artery

Introduction

In the clinical settings, pulmonary regurgitation (PR) signals were often detected on Doppler echocardiography. Since the elevation of the left sided cardiac pressure might influence the right sided cardiac hemodynamics, evaluation of the pulmonary hemodynamics is of considerable interest in patients with hypertension. Recently, we reported that regurgitation signal in the pulmonary artery in the systolic phase could be related to hypertensive left ventricular (LV) hypertrophy. However, few studies have been conducted on the clinical significance of PR signals, and there have been no reports concerning its significance in patients with hypertensive LV hypertrophy. The purpose of this study was to assess the clinical significance of PR signals on Doppler echocardiography in patients with hypertensive LV hypertrophy.
with hypertensive LV hypertrophy.

Methods

Subjects
We studied 14 consecutive patients with hypertensive LV hypertrophy (HHD group) who underwent routine echocardiographic examinations and conventional continuous wave Doppler examination.

The presence of LV hypertrophy was established when the LV mass index (LVMI) obtained from echocardiography was $>111 \text{ g/m}^2$ for men and $>106 \text{ g/m}^2$ for women. Seventeen patients with no evidence of heart disease served as control subjects (Control group). The mean age was significantly higher in the HHD group than in the control group. There was no significant difference in heart rate between the two groups. Clinical characteristics of the study subjects were summarized in Table 1.

Echocardiographic study
Echocardiograms were obtained using a TOSHIBA SSH-160A equipped with a 2.5-MHz transducer. A two-dimensional targeted M-mode echocardiogram was recorded in each patient with simultaneous recording of a II-lead electrocardiogram and phonocardiogram. All examinations were performed in the 30°left-lateral decubital position. On the M-mode LV echocardiogram, LV end-diastolic dimension (LVDd), LV end-systolic dimension (LVDs), diastolic wall thickness of interventricular septum (IVSTh), and posterior wall thickness (PWTh) were measured. The LV internal dimension and diastolic septal and posterior wall thickness were measured at the peak of R wave of the electrocardiogram, and the LVDs was measured at the peak of second heart sound of the phonocardiogram. The percentage of ejection fraction (EF) (%) was calculated as $(\text{LVDd} - \text{LVDs})/\text{LVDd} \times 100$.

Relative wall thickness (PWT) was calculated as $2 \times \text{PWTh}/\text{LVDd}$. LV mass (LVM) was calculated using the following formula validated by Devereux and Reichek:

$$\text{LVM (g)} = 1.04 \times ([\text{LVDd} + \text{IVSTh} + \text{PWTh}]^3 - (\text{LVDs})^3) - 13.6$$

Doppler study
Continuous wave (CW) Doppler imaging was performed with reference to a two-dimensional echocardiographic imaging from aortic short-axis plane view in each patient. The right ventricular outflow tract was visualized in the left parasternal short-axis view at the level of the aortic valve with a simultaneous electrocardiographic and phonocardiographic recordings. The presence of pulmonary regurgitation was determined using color Doppler imaging. The CW Doppler sample volume was placed just proximal to the pulmonary valve and within the color Doppler signal. On Doppler echocardiography, maximum flow velocity (Vmax), end-diastolic flow velocity (Vmin), duration time (DT), ratio of decay velocity to time (Decay), and time to reach pressure difference of 1/2 (PHT) of the PR signal were measured and compared between the two groups. And the correlations between these parameters and LVM were examined.

Statistical analysis
Values were expressed as the means±SD, and comparisons were assessed by the unpaired t-test. Differences were considered significant at p value of <0.05. Simple regression analysis was employed to test correlations.

Results

1. Comparison of the echocardiographic parameters between the two groups

1) There were no significant differences in LVDd, LVDs, LAD, and EF between the two groups. LVM, IVS, PW were significantly higher in the HHD group. (Table 2)

2. Comparison of the Doppler parameters between the two groups

<table>
<thead>
<tr>
<th>Table 1. Clinical Characteristics of the Study Subjects</th>
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<tbody>
<tr>
<td><strong>Number</strong></td>
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<tr>
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</tr>
<tr>
<td>Age (yr.)</td>
</tr>
<tr>
<td>Mele/Female</td>
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<tr>
<td>HR (beats/min)</td>
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</table>

Abbreviations: HR=heart rate
Table 2. Echocardiographic Findings

<table>
<thead>
<tr>
<th></th>
<th>HHD Group</th>
<th>Control Group</th>
<th>P value</th>
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</thead>
<tbody>
<tr>
<td>LVDd (mm)</td>
<td>43.9 ± 7.5</td>
<td>47.5 ± 5.6</td>
<td>NS</td>
</tr>
<tr>
<td>LVDs (mm)</td>
<td>25.7 ± 7.0</td>
<td>28.6 ± 5.6</td>
<td>NS</td>
</tr>
<tr>
<td>LAD (mm)</td>
<td>39.4 ± 11.4</td>
<td>34.6 ± 4.7</td>
<td>NS</td>
</tr>
<tr>
<td>IVS (mm)</td>
<td>16.1 ± 4.2</td>
<td>9.8 ± 1.5</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>PW (mm)</td>
<td>13.5 ± 5.0</td>
<td>9.51 ± 1.28</td>
<td>0.0032</td>
</tr>
<tr>
<td>LVM (g)</td>
<td>323.1 ± 152.7</td>
<td>188.9 ± 59.1</td>
<td>0.0023</td>
</tr>
<tr>
<td>EF (%)</td>
<td>77.5 ± 13.1</td>
<td>73.5 ± 11.6</td>
<td>NS</td>
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Abbreviations: LVDd=left ventricular end-diastolic diameter, LVDs=left ventricular end-systolic diameter, LAD=left atrial diameter, IVS=thickness of the inter ventricular septum, PW=thickness of the posterior wall, LVM=left ventricular mass, EF=ejection fraction.

Table 3. Doppler Finding of PR Signal

<table>
<thead>
<tr>
<th></th>
<th>HHD Group</th>
<th>Control Group</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vmax (m/s)</td>
<td>1.29 ± 0.56</td>
<td>1.17 ± 0.34</td>
<td>NS</td>
</tr>
<tr>
<td>Vmin (m/s)</td>
<td>0.75 ± 0.30</td>
<td>0.61 ± 0.20</td>
<td>NS</td>
</tr>
<tr>
<td>DT (sec)</td>
<td>428.4 ± 159.2</td>
<td>383.0 ± 91.0</td>
<td>NS</td>
</tr>
<tr>
<td>Decay (m/s²)</td>
<td>1.35 ± 0.98</td>
<td>1.55 ± 0.68</td>
<td>NS</td>
</tr>
<tr>
<td>PHT (sec)</td>
<td>283.5 ± 80.2</td>
<td>209.6 ± 29.2</td>
<td>0.0014</td>
</tr>
</tbody>
</table>

Abbreviations: Vmax=maximum velocity, Vmin=minimum velocity, DT=deceleration time, Decay=ratio of decay velocity to time, PHT=pressure half time.

Figure 1. Measurements of maximum flow velocity (Vmax), end-diastolic flow velocity (Vmin), duration time (DT), ratio of decay velocity to time (Decay), and time to reach pressure difference of 1/2 (PHT) of the PR signal on Doppler echocardiography.

Figure 2. Comparison of the Doppler parameters between the two groups. Example of PR signal in a subject from the HHD group (Left panel) and a subject from the Control group (Right panel). The velocity deceleration in the first half was more steep in the subject from the Control group compared with the subject from the HHD group, and the PHT in the former was shorter than in the latter.

Figure 1 shows an example of measurements of maximum flow velocity (Vmax), end-diastolic flow velocity (Vmin), duration time (DT), ratio of decay velocity to time (Decay), and time to reach pressure difference of 1/2 (PHT) of the PR signal on Doppler echocardiography.

Figure 2 shows examples of PR signal in a subject from the HHD group (Left panel) and a subject from the Control group (Right panel). The velocity deceleration in the first half was more steep in the
subject from the Control group compared with the subject from the HHD group, and the PHT in the former was shorter than in the latter.

1) There were no significant differences in Vmax, Vmin, DT, and Decay between the two groups.
2) PHT was significantly longer in the HHD group (P<0.01). (Table 3)

3. Correlations between LVM and Doppler parameters in the two groups

In the HHD group, significant positive correlations were observed between LVM and Vmax, and between LVM and Decay (r=0.592, P<0.05 and 0.738, P<0.01 respectively). (Figure 3,4)

Discussion

1. Valvular regurgitant signal on Doppler echocardiography

The Doppler echocardiographic technique has been used to assess valvular regurgitation in patients with aortic regurgitation in great detail. In cases with relatively mild aortic regurgitation, the pressure difference between aorta to left ventricle gradually decreases as the regurgitant blood flows into the left ventricle. In more severe cases, the aortic pressure drops rapidly and the increased pressure due to the large regurgitant volume results in a steep rise in the left ventricular pressure. Thus, the pressure differential decreases and the slope of the diastolic regurgitant jet increases. A larger regurgitant orifice permits a steeper fall in aortic pressure. In addition, an increasing volume of blood entering the left ventricle from the aorta accelerates the rise in the left ventricular diastolic pressure. Thus, with severe aortic regurgitation, the pressure gradient decreases rapidly and even disappears if the diastolic interval is long enough to permit equalization of aortic and left ventricular pressures. This falling pressure gradient produces an increased slope of the regurgitant velocity. The rapidity with which the aortic and left ventricular pressures equalize is a function of the severity of the aortic regurgitation.

Given that the control subjects showed no evidence of heart disease other than the PR signal, the mechanism of the PR signal demonstrated in healthy subjects should be discussed. Yock et al. detected flow in the opposite direction to right ventricular ejection during diastole in 40% of healthy adults by the continuous wave Doppler technique. Takao et al. suggested the following mechanism as causes of PR in healthy subjects; that is pulmonary valve is less tight than that of aortic valve, that is, it has a less tight fibrous ring, thinner cusps, a more hypoplastic Alantius nodule, and shallower sinuses. In our view, the movement of the blood in the reverse direction between the valvular leaflets in the valvular closure is likely to be the mechanism responsible for the PR signal demonstrated in the healthy subjects. Thus, we conjecture that the reversed blood flow velocity decreases rapidly in the control group subjects, resulting in an increase in the slope of the regurgitant velocity.

PHT was significantly longer in the HHD group than in the Control group in the present study. In several reports using the simplified Ber-
noulli equation, the velocity of the regurgitation signal reflected pressure gradients across cardiac valves. There seems to be a large, continuous pressure difference between the pulmonary artery and right ventricle through the diastolic phase in patients with hypertensive LV hypertrophy.

2. PR signal in patients with hypertensive LV hypertrophy

We propose two mechanisms of PR signal in patients with hypertensive LV hypertrophy. One is elevation of the right-sided cardiac pressure. We hypothesized that the decrease in LV compliance induced by the LV hypertrophy led to elevations of the LV end-diastolic pressure and left atrial pressure, resulting in elevations of the pulmonary arterial pressure and presenting with PR signal. Richard et al reported that the elevation of the pulmonary arterial pressure induced the PR signal on Doppler echocardiography. Furthermore, an evaluation of pulmonary regurgitation in their study allowed accurate noninvasive estimation of the diastolic pressure of the pulmonary artery. The other potential mechanism is an anatomical or morphological deformity of the pulmonary valvular leaflets due to hypertrophy of the interventricular septum. Feigenbaum described that right ventricular volume overload with abnormal septal motion and configuration may occur in patient with PR. We propose that the hypertension-induced hypertrophy of the interventricular septum may affect the morphological mechanism of the valvular closure, resulting in an insufficiency of the pulmonary valve and pulmonary regurgitation. The significant correlation between the Vmax, decay and the LVM demonstrated in the present study may support this hypothesis.

Conclusion

We propose that the elevation of the pulmonary artery pressure and decrease of the right ventricular compliance in patients with hypertensive LV hypertrophy caused by a decrease of the LV compliance, thereby generating a large, continuous pressure difference between the pulmonary artery and the right ventricle through the diastolic phase. An analysis of PR signals could be useful for evaluating hemodynamic change in patients with hypertension.

References


高血圧性心肥大例における肺動脈弁逆流の臨床的意義

小澤 泰典1  信岡 祐彦2  三宅 信之1  三宅 良彦1

抄 録

超音波検査で認める肺動脈弁逆流 (PR) シグナルを検討した。対象は、心房超音波検査を施行した患者のうち PR シグナルを認めた高血圧性心肥大例 14 例で、また超音波検査上 PR シグナルを認める以外は他に器質的異常を認めない 17 例をコントロール群として用いた。PR シグナルの最高血流速度 (Vmax), 扩張末期血流速度 (Vmin, 滅衰速度 (DT), 滅衰速度/時間比 (Decay), 压差が 1/2 までになる時間 (PHT) を計測し、両群間で比較検討した。結果は 1. Vmax, Vmin, DT, Decay は両群間で有意な差はなかった。2. PHT は HHD 群が有意に低値を示した (P<0.01)。3. HHD 群において LVM と Vmax, LVM と Decay との間には有意な相関関係が認められた。HHD 群で HHD 例における PR シグナルの解析は、HHD 例の血行動態解析や薬物治療を含めた管理に有用な手法となる可能性が示唆された。

索引用語

高血圧, 左室肥大, 肺動脈逆流, 肺動脈, 心房超音波ドップラー

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