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STUDY OF RELATION BETWEEN HYPERTENSIVE LEFT VENTRICULAR HYPERTROPHY AND NEUROCRINE FACTORS

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Abstract: In this study of the relationship between hypertensive left ventricular hypertrophy(LVH) and neurohumor factors and ventricular arrhythmias, 180 cases were divided at random into 3 groups: 60 cases with primary hypertension and LVH(PH + LVH); 60 cases with simple hypertension(PH), and with other diseases and LVH(NPH + LVH). The results showed that 1. The excitability of the sympathetic nerve was not elevated while the activity of the vagus nerve was not significantly decreased in the patients with PH + LVH. The increased sympathetic nervous tension was correlated with the ventricular premature beat which was statistically correlated with the myocardial ischemia. 2. The patients' condition in group PH + LVH was not correlated with the levels of aldosterone and insulin and was not even positively correlated with the levels of renin and angiotensin-Ⅱ. 3. The patients in group PH + LVH had high incidence of ventricular premature beat and myocardial ischemia. 4. The patients' condition in group NPH + LVH was not even positively correlated with the neurohumor factors but their heart failure was positively correlated with the levels of aldosterone, sympathetic nervous tension, myocardial ischemia and ventricular premature beat.

Key words: hypertension, left ventricular hypertrophy, neurohumor, ventricular premature, beat
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INTRODUCTION

Hypertension, one of the most common cardiovascular diseases around the world, is a serious threat against people's life and health. Hypertension with left ventricular hypertrophy(LVH) is a more important risk factor than isolated high blood pressure in causing morbidity and mortality in cardiovascular accidents such as cardiogenic sudden death, myocardial ischemia, heart failure, ventricular arrhythmia, etc(Zhang et al., 1991; Perolff 1994). This study was aimed to find the relations between hypertensive LVH and neurocrine factors; to inquire into some mechanism differences between hypertensive LVH and LVH due to other diseases; and to research the relations between hypertensive LVH and premature ventricular beats and myocardial ischemia.

PATIENTS STUDIED AND FACILITIES

There were 180 inpatients divided into 3 groups: 60 patients in the first group had primary hypertension with LVH (PH + LVH), including centritropic hypertrophy (25 cases) and centrifugal hypertrophy(35 cases); 60 cases in the second group with simple primary hypertension (PH); The other 60 cases in the third group had LVH caused by other diseases(NPH + LVH), such as rheumatic heart disease(35 cases) and dilated cardiomyopathy (21 cases) and hypertrophic cardiomyopathy(4 cases). The basic conditions including age and sex ratio were similar among the 3 groups. Equipment included Holter 3.1 Recorder and HP - 43405A analyser, Acuson Computer Sonography 128XP/10C cardiac echo and 262 γ-counter. There were 4 kinds of reagent boxes for renin, angiotensin-Ⅱ, aldosterone and insulin examination.

METHODS

Body surface area(BSA) was calculated by height and weight. Echo parameters included LVDd, LVPW, IVST and LVM, LVMI (LVM/BSA), RWT(2LVPW/LVDd), Holter and Ambulatory BP Monitor recording for the patients were synchronized. Fifteen mL blood was drawn in the morning after the patients had an overnight fast and bed rest. For AT-Ⅱ, renin and aldosterone measurement by chemical colorimetric anal-

ysis, the 3 mL of blood drawn from the patient 30 min. after the patient had taken 75 grams glucose was used to measure the peak insulin value of glucose loading. Secondary hypertension (dissected aneurysm, renal hypertension, pheochromocytoma, etc.) and normatensive cases were ruled out. Statistical analysis was done with the students't-test.

RESULTS

AT-Ⅱ and renin value were not significantly different among the three groups (PH + LVH: 81.05 ± 19.63pg/mL and 1.33 ± 1.02μg/mL, PH:77.69 ± 21.71 and 1.20 ± 0.94, NPH + VLH:76.95 ± 16.64 and 1.46 ± 0.87). The peak insulin value of the PH + LVH group was higher than that of the PH group (21.95 ± 9.08 vs 14.13 ± 9.72, μIU/mL, $P < 0.05$). The aldosterone value of the NPH + LVH group (220.18 ± 71.76) was higher than that of the other two groups (PH + LVH: 152.68 ± 68.46, PH: 112.74 ± 46.18, pg/mL, all $P < 0.01$). In the NPH + LVH group, the average aldosterone value of patients with NYHA heart function class Ⅰ and Ⅱ was 124.96 ± 41.73 pg/mL, and that of the heart function class Ⅲ was 315.40 ± 101.58 pg/mL. The high frequency(HF) value of the PH + LVH group(119.12 ± 69.44) was similar to that of the other two groups (PH: 125.25 ± 88.76, NPH + LVH: 126.75 ± 47.16, ms^2/Hz). The very lower frequency(VLF) and lower frequency (LF) value and LF/HF ratio of the PH + LVH group and PH group were significantly different (VLF: 1481.24 ± 687.71/1017.96 ± 547.08 ms^2/Hz; LF: 408.92 ± 227.43/314.61 ± 128.21 ms^2/Hz; LF/HF ratio: 3.56 ± 2.49/2.42 ± 1.76; $P < 0.01, 0.05, 0.05$, respectively). The VLF and LF of the PH + LVH group (see above) and NPH + LVH group (VLF: 1729.30 ± 791.87, LF: 499.70 ± 187.72, P all < 0.05) differed significantly, but HF and LF/HF of the two groups did not differ.

DISCUSSION

Our data demonstrated that hypertensive LVH related to increased excitability of the sympathetic nerve. Its mechanism may be that sympathetic nerve directly stimulates release of the local myocardial growth factor, enlargement of myocardial cells, and synthesis of protein(Mayet, et al., 1995). This study showed that hypertension could reduce vagal activity, but is not an important factor causing LVH. The sympathetic tone of the patients with heart failure was higher than that of patients with normal heart function.

Peak insulin value and aldosterone were higher in the cases with PH + LVH and there was postive relation between the degree of heart failure and aldosterone value. The first author's conclusion from the study results is that(Perloff, 1994; Grossman et a., 1994; Siche et al., 1994; Francis et al., 1993): 1. Aldosterone can facilate transcription of special protein, induce synthesis of $Na^+ - K^+$ ATPase and cause myocardial fibrosis by inducing interaction between the intracellular corticoid receptor and deoxyribonucle. Aldosterone can also induce cardiac local sympathetic action and influence the development of LVH indirectly. 2. There is relation between LVH and high peak insulin value. The detailed mechanism is unknown, may be related with the insulin growth factor (IGF) 3. LVH is not related to renin and AT - Ⅱ.

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