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Pathogenesis and Clinical Implications of Left Ventricular Hypertrophy in Hypertension, Especially in Patients with Borderline and Low Grade Hypertension

Tamar Gaprindashvili[a*], Zurab Pagava[b], Rusudan Agladze[c]

[a,b,c]*Bokhua Memorial Cardiovascular Center,Tbilisi,Georgia*

[a]*Email: mantskavamaka@bk.ru*

Abstract

Physical exercise and systemic hypertension are associated with cardiac adaptive mechanisms. As left ventricle is more prone to BP changes, the impact is most prominent on the left side of the heart, which leads to left ventricular remodeling and in consequence left ventricular hypertrophy. [1]Since the sensitivity of the different ECG criteria may be as low as 7 to 35 percent with mild LVH and only 10 to 50 percent with moderate-to-severe disease, echocardiography is the diagnostic procedure of choice [2] Although it is relatively insensitive, electrocardiography (ECG) does have prognostic significance [3] Left ventricular hypertrophy (LVH) is a common finding in patients with fixed or borderline hypertension. Previous studies have already shown that exaggerated response of BP to exercise is a valid risk factor for future developing of hypertension. It often does not correlate with a grade of hypertension the objective or the article is to collect information about studies that review structural and functional cardiac changes seen in patient with High normal and Grade I hypertension.

KeyWords: hypertrophy; hypertension; left ventricle; remodeling.

1. Introduction

Hypertension is one of the most important risk factors for cardiovascular morbidity and mortality. Due to consecutive cardiac remodeling and high evidence of fatal cardiovascular abnormalities, makes early identification of persons with high risk of for developing hypertension a leading mission. Left Ventricular Hypertrophy LVH is defined as an increase in the mass of the left ventricle. It can de due to increase in wall thickness, an increase in cavity size, or both. As a consequence of hypertention LV responds with cardiac remodeling, due to increased afterload presented by increase in wall thickness, without increace of LV cavity type LVH, although there is also a genetic component.

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* Corresponding author.

Pathophisiologic mechanism incueds significant increase in number and/ir size of sarcomeres in cardiomyocite. As echocardiography is a most sencetive and cheap method of early revialing of LVH, the estimation of LV mass is commonly derived from LV measurements obtained by 2D echocardiography. According to the American Society of Echocardiography and the European Association of Echocardiography, left ventricular hypertrophy is derived from relative wall tickness (sezes of inter ventricular septum IVS and LV end dyastolic diameter EDD. [4]. Using LV mass = 0.8[1.04 (LVIDD + IVST + PWT)3 2 (LVIDD)3] + 0.6, formulae. Wall thickness and cavity dimensions, are an estimated LV mass (indexed for body surface area) of >102 g/m^2 for men and >88 g/m^2 for women According to mentioned guidline we have normal LV geometry (normal RWT+Normal Mass), Concentric Hypertropy (RWT > 0.42 and elevated LV mass Mass index), Eccentric hypertrophy (Normal RWT with increased LV Mass), concentric remodeling (normal Mass with elevated RWT) (Table 1)

Table 1: Types of LV Hypertrophy.

LV geometry	LV Mass	(RWT)
Normal	≤115 g/m2 (men) or ≤95 g/m2 (women)	<0,42
Concentric Hypertrophy	≥115 g/m2 (men) or >95 g/m2 (women)	>0,42
Eccentric Hypertrophy	>115 g/m2 (men) or >95 g/m2 (women)	<0,42

So in addition to the absolute increase in mass, the geometric pattern of LVH also may be important [5]. Studies Showe that patients without an increase in absolute mass, but with an increase in relative wall thickness (concentric remodeling), have the same adverse risk as those with concentric hypertrophy. Evaluation of concentric hypertrophy is independent of grade of hypertension but shows elevation in risk with either finding is for both cardiovascular disease and death and is independent of the level of blood pressure [6]. Remodeling process also results in LV fibrosis and both factors contribute to an increase in LV stiffness, resulting in diastolic dysfunction and an elevation in LV end diastolic pressure. Although ECG is a less sensitive method to reveal early signs of hypertrophy, hypertensive patients with echocardiographically proven LVH who also meet ECG criteria have a greater LV mass than those without the expected ECG changes. In particular, changes in ECG voltage over time may reflect changes in LV mass and correlate with cardiovascular risk.As cardiovascular risk associated with LVH is very high and it can be reduced by effective antihypertensive therapy that leads to regression of LVH, early detection of patients more prone to develop cardiac remodeling can save them from major cardiac adverse events.

2. Materials and Methods

Present article is a systemic review of 12 articles using as a source Pub med and Google scholar. All the citations are according Google scholar MLA model.

3. Results

The close analysis of articles in question showed that presence of LVH ether in ECG or echocardiography, has a

great clinical importance due to direct association with increased risk in incidence of the major cardiovascular events such as heart failure, death following Acute coronary syndrome, fatal ventricular arrhythmias, other types of sudden cardiac death, cerebrovadcular events an effect that is independent of blood pressure [Table 2].

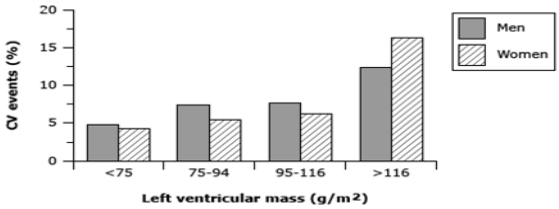

Figure 1

Four-year, age-adjusted incidence of cardiovascular events in men and women in the Framingham Study according to left ventricular mass determined by echocardiography. Subjects with increased left ventricular mass (far right panel) had a marked increase in cardiovascular risk. Adapted from: Levy D, Garrison RJ, Savage DD and his colleagues Prognostic implications of echocardiographically determined left ventricular mass in the Framingham Heart Study. N Engl J Med 1990; 322:1561.

●In a prospective study, 1033 subjects over the age of 50 years with primary hypertension and no previous cardiovascular events were followed for a median of three years .The rate of major cardiovascular events (fatal and nonfatal myocardial infarction; all-cause, sudden, or cardiovascular mortality; severe heart failure; or severe renal failure requiring dialysis) was significantly higher in the 29 percent of patients with an increased left ventricular mass, defined as ≥125 g/m^2 body surface area. After adjustment for other risk factors, LVH was associated with an increase in cardiovascular events (relative risk 2.08); for each 39 g/m^2 increase in left ventricular mass, there was a 40 percent increased risk of a major cardiovascular event (figure 3).

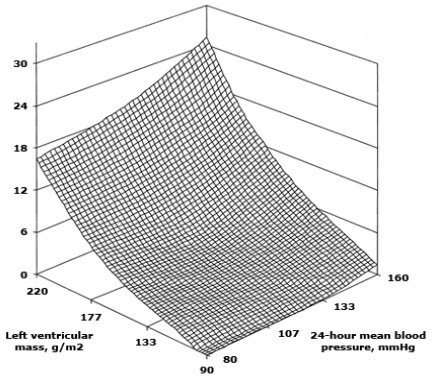

Figure 2

In a study of 1033 hypertensive subjects the age-adjusted five-year probability of cerebrovascular events is related to left ventricular mass, as assessed with echocardiography. *Data from Verdecchia, P, Porcellati, C, Reboldi, G and his colleagues Circulation 2001; 104:2039.*

●A report from the Framingham Heart Study examined the relationship between left ventricular mass and hypertrophy and sudden death in 3661 subjects over the age of 40 years who were followed for 14 years The prevalence of LVH was 22 percent and the risk factor adjusted hazard ratio for sudden death was 2.16 (p=0.008). For each 50 g/m increment in left ventricular mass, the risk-factor adjusted hazard ratio for sudden death was 1.45 (p=0.008) (figure 1).

As numerous studies showed, LVH is associated with elevated cardiac risk which is probably due to myocardial ischemia that can be induced by a variety of factors. In hypertrophied myocardium, there is a reduced density of capillaries leading to enlarged muscle mass that limits the ability of the coronary arteries to dilate in response to decreased perfusion, reduced coronary flow reserve [9]; and direct compression of the endocardial capillaries. Both of these factors can decrease coronary reserve and can have a number of important clinical implications.

● LVH in patients with acute MI is associated with a greater infarct size and a higher mortality rate than seen in the absence of LVH [10].

●The hypertrophied myocardium may be more susceptible than normal myocardium to the effects of ischemia. In a study of patients with sudden cardiac death, the patients with hypertension and LVH who died suddenly had less extensive coronary disease and were less likely to have thrombi in the coronary vessels than normotensives who died suddenly [11].

The development of heart failure with LVH results from ether decreased left ventricular systolic function due to precious MI or diastolic dysfunction, so called Heart Failure with preserved Ejection Fraction EF, which represents almost 50% of HF patients. So the deleterious effect of LV remodeling may be an important determinant of progression to overt heart failure.

4. Discussion

Studies showed that regression of LVH was associated with improved systolic function , enhanced stroke volume, and no increase in the risk of decomposition in hypertensive occasions. It may also have other benefits as a reduced number of ventricular premature contraction PVCs, decreased risk of ventricular and atrial fibrillation. As diastolic dysfunction is one of the earliest changes after LV remodeling and consecutive LVH, the effect of LVH regression on diastolic dysfunction, one of the earliest changes in hypertension, has been less well studied. This issue was addressed in the LIFE trial, which randomly assigned 728 hypertensive patients with LVH by ECG criteria to antihypertensive therapy with ARB and BB [12]. Study also showed that after one year LVH regression was associated with significant improvement in left ventricular diastolic filling parameters and no change in those without regression.

Most important parameter, Reduction in cardiovascular risk

Regression of LVH, either by ECG or echocardiography is associated with a reduction in cardiovascular risk. Framingham Heart Study of 524 patients with LVH; during follow-up there were 269 new cardiovascular events showed the magnitude of its benefit. Changes in LVH on the ECG were measured as the sum of the R wave in

aVL and the S wave in V3. Compared to patients with no serial change, those with a serial decline in voltage were at lower risk for cardiovascular disease. By contrast, patients with a serial increase in voltage were at higher risk (adjusted odds ratio 1.86 in men and 1.61 in women).

5. Conclusions

- Left ventricular hypertrophy (LVH) is associated with increased incidence of heart failure, ventricular arrhythmias, death following myocardial infarction, decreased LV ejection fraction, sudden cardiac death, aortic root dilation, and a cerebrovascular event. The increase in cardiovascular risk is directly related to the degree of increase in left ventricular mass
- LVH can be diagnosed either by ECG or by echocardiography. Echocardiography is more sensitive and is the preferred test.(see section “Introduction”)
- As an early response to hypertension, the most acurate method of detection of development of LVH, is 2D echocardiography. Risk factors for the development of LVH include exaggerated transient elevations in blood pressure during mild exercise od mental stress, a high percentage of pressures above 135/85 during the day and 120/80 mmHg at night, nocturnal hypertension. (non-Dipperes)
- It is important that LV mass may be increased **prior** to the development of overt hypertension. This observation may be explained by the fact that the same factors, that regulate blood pressure can promote both hypertension and LVH. Also the tendency to LVH may be an inherited trait that predisposes to the development of hypertension.

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References

[1]. Miyai, Nobuyuki, et al. "Blood pressure response to heart rate during exercise test and risk of future hypertension." Hypertension. 2002; 39(3):761-766

[2]. Devereux, Richard B. "Is the electrocardiogram still useful for detection of left ventricular hypertrophy?." Circulation. 1990; 81(3):1144-1146

[3]. Okin, Peter M., et al. "Electrocardiographic strain pattern and prediction of cardiovascular morbidity and mortality in hypertensive patients." Hypertension. 2004; 44(1):48-54

[4]. Cuspidi, C., et al. "Prevalence of left-ventricular hypertrophy in hypertension: an updated review of echocardiographic studies." Journal of human hypertension. 2012; 26(6):343-349

[5]. Lang, Roberto M., et al. "Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging." European Heart Journal-Cardiovascular Imaging. 2015; 16(3):233-271

[6]. Krumholz, Harlan M., Martin Larson, and Daniel Levy. "Prognosis of left ventricular geometric patterns in the Framingham Heart Study." Journal of the American College of Cardiology. 1995;

25(4):879-884

[7]. Verdecchia, Paolo, et al. "Adverse prognostic significance of concentric remodeling of the left ventricle in hypertensive patients with normal left ventricular mass." Journal of the American College of Cardiology. 1995; 25(4):871-878.

[8]. Zhao, Lin, et al. "Deletion of interleukin-6 attenuates pressure overload-induced left ventricular hypertrophy and dysfunction."Circulation research. 2016; 118(12):1918-1929

[9]. Beache, Garth M., et al. "Attenuated myocardial vasodilator response in patients with hypertensive hypertrophy revealed by oxygenation-dependent magnetic resonance imaging."Circulation. 2001; 104(11):1214-1217

[10]. Carluccio, Erberto, et al. "Prognostic value of left ventricular hypertrophy and geometry in patients with a first, uncomplicated myocardial infarction." International journal of cardiology. 2000; 74(2-3):177-183

[11]. Burke, Allen P., et al. "Effect of hypertension and cardiac hypertrophy on coronary artery morphology in sudden cardiac death." Circulation. 1996; 94(12):3138-3145.

[12]. Wachtell, Kristian, et al. "Change in diastolic left ventricular filling after one year of antihypertensive treatment: The Losartan Intervention for Endpoint Reduction in Hypertension (LIFE) Study." Circulation. 2002; 105(9):1071-1076