Left ventricular hypertrophy with and without hypertension: what is the differential in cardiovascular risk?

A. Ryabikov, S. Malyutina, S. Shakhmatov, G. Simonova, V. Gafarov, E. Veryovkin

Novosibirsk State Medical University
Research Institute of Internal Medicine
Novosibirsk, Russia
ALL THE AUTHORS DECLARED NO CONFLICT OF INTEREST RELATED TO REPORTED STUDY RESULTS
INTRODUCTION

The left ventricular hypertrophy (LVH) is an established predictor of coronary and cerebrovascular events and mortality. Much less is known about specific impact of LVH without hypertension on CVD risk.

OBJECTIVES

To assess the impact of composite patterns of LVH with/without hypertension (HT) on CVD events and mortality in a general population.
STUDIED POPULATION AND DESIGN

The data came from Novosibirsk (Russia) MONICA study. Population surveys in 1988/89 and 1994/95 recruited about 7000 men and women aged 25-64 in two city districts. Random sub-sample was examined with echocardiography and prospectively followed-up.

ECHO-cohort characteristics:
- men and women, n=2006 (w.-41%)
- aged 25-64 years at baseline
- mean follow-up period 12.2 years
- analyzed end points
  - acute myocardial infarction (MI) (ICD-9 410)
  - fatal MI (ICD-9 410)
  - acute stroke (ICD-9 430-434, 436)
  - death from CVD (ICD-9 390-459)
  - death from all causes
METHODS

LV mass was assessed echocardiographically by anatomically validated formula (Devereux) and then indexed on BSA (LVMI).

The LVH was defined based on the population-specific reference value for myocardium mass index: 124 g/m² for men and 100 g/m² for women (90% cut-off point of LVMI distribution in apparently healthy group).

The intra-observer inter-session reproducibility coefficient for LV mass according to Bland and Altman’s method was 2.0%.
BP was measured in survey center 2 times and averaged for further analysis.

We distinguished 4 patterns by hypertension and LV hypertrophy:

(i) Normotension without LVH (ref. group, n=856)
(ii) HT without LVH (n=679),
(iii) LVH without HT (n=106),
(iv) HT with LVH (n=346)
Prevalence of LVH by gender in Novosibirsk population (men & women, 25-64)

Reference criteria for LVH:

LVMI > 124 g/m² (men)
> 100 g/m² (women)

n=2006

22.8%

19.2%

27.9%

p<0.001
**Combination of LVH with HT**

*(general population sample, men & women, 25-64 yrs, n=2006)*

<table>
<thead>
<tr>
<th>Criteria</th>
<th>LVH (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HT (+)</td>
<td>33.8%</td>
</tr>
<tr>
<td>HT (-)</td>
<td>11.0%</td>
</tr>
</tbody>
</table>
Follow-up period 12.2 years (median)

Totally: 220 end points

MI 67 events (49 fatal),
Stroke 31 events (25 fatal)
Fatal CVD 90 events
All-cause death 196 events

We applied multivariable Cox-regression for cohort analysis (adjustment for age, SBP, antihypertensive treatment, BMI, TC, HDL-C, TG, smoking, alcohol, physical activity, gender)
12-year risk* of fatal MI by combination of HT and LVH

*(men and women, 25-64 yrs, Novosibirsk)

* - multivariable Cox-regression

Fig 2.
12-year risk* of cardiovascular death by combination of HT and LVH

*men and women, 25-64 yrs, Novosibirsk*

![Bar chart showing risk of cardiovascular death by combination of HT and LVH](image)

- **HT- LVH-**: 1.7
- **HT+ LVH-**: 3.3
- **HT- LVH+**: 2.9
- **HT+ LVH+**: 0.155

*p* values: 0.155, 0.023, 0.013

* - multivariable Cox-regression

Fig 2.
12-year risk* of all-cause death by combination of HT and LVH

(men and women, 25-64 yrs, Novosibirsk)

Fig 3.

* - multivariable Cox-regression
12-year risk* of incident cardiovascular events and death by combination of HT and LVH

(men and women, 25-64 yrs, Novosibirsk)

* - multivariable Cox-regression

Fig 4.
12-year risk* of incident cardiovascular events and death in HT without LVH (HT+LVH-)

*(men and women, 25-64 лет, Novosibirsk)

**Fig 5.**

*age-adjusted and multivariable-adjusted Cox-regression
Clinical profile of the groups

<table>
<thead>
<tr>
<th></th>
<th>HT- LVH+</th>
<th>HT+ LVH+</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yrs</td>
<td>47.3*</td>
<td>52.3</td>
</tr>
<tr>
<td>Women, %</td>
<td>50.0</td>
<td>48.9</td>
</tr>
<tr>
<td>BP, mm Hg</td>
<td>123.3/80.4***</td>
<td>157.1/100.0</td>
</tr>
<tr>
<td>Heart rate, bpm</td>
<td>69</td>
<td>70</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>27.8***</td>
<td>30.1</td>
</tr>
<tr>
<td>LV Diam Index, mm/m²</td>
<td>28.8***</td>
<td>27.5</td>
</tr>
<tr>
<td>LV mass Index, g/m²</td>
<td>126.7**</td>
<td>132.7</td>
</tr>
<tr>
<td>LVH Eccentric dilated geometry, %</td>
<td>29***</td>
<td>12</td>
</tr>
<tr>
<td>Alcohol (occ.dose), g.eth.</td>
<td>64.4**</td>
<td>62.4</td>
</tr>
</tbody>
</table>

P < * - 0.05; ** - 0.01; *** - 0.001-.0001
Potential explanations for unfavourable prognosis in normotensives with LVH

- Genetic background (HCM, DCM)
- “Masked” HT and high normal BP
  - inappropriate hypertrophic response
- (Cardio)Metabolic syndrome associated with LV
- Alcohol consumption (?)
- Arrhythmias (?)
CONCLUSION

- In a general population sample ~11% of normotensives have LV myocardium hypertrophy.

- Echocardiographic LVH with hypertension or without hypertension independently increased CVD risk in general population.

- The excess risk of CVD outcomes was 15-70% higher among normotensive patients with LVH compared to hypertensive counterparts.

- Hypertension without LVH was the less powerful predictor of CVD risk, confined to fatal coronary outcomes.
Geometric types of LVH in population

(men and women, general population sample 25-64 years, Novosibirsk)

- Eccentric dilated: 16.7%
- Eccentric-nondilated: 53.9%
- Concentric: 21.9%
- Disproportionate septal: 7.5%

(Framingham stratification; Savage et al, 1987)