THE 5-YEAR DYNAMICS OF CARDIAC STRUCTURE AND FUNCTION IN PATIENTS WITH CORONARY ARTERY DISEASE AFTER MYOCARDIAL REVASCULARIZATION

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ABSTRACT
The aim: To estimate the dynamics of echocardiographic parameters in patients with CAD within 5 years after revascularization.

Material and methods: 50 persons (males/females 39/11; mean age 59.9±9.3 years; STEMI 76%, non-STEMI 24%) were divided into two groups: n=38 after PCI with stenting (PCIwS); n=12 after CABG. Observation included regular echocardiography with LV myocardial mass (LVMM) and geometry estimation.

Results: Groups were comparable by age, co-morbidity, BP, heart rate and BMI. Significantly severe baseline LV hypertrophy (LVH) and left atrial enlargement (LAE) in group 2 explained by spread coronary atherosclerosis. Later progressive LAE (4.37±0.22 cm, \(P_{0.05} < 0.05\)) in group 1, and aortic/LV dilatation (+0.4/+1.0 cm, respectively, both \(P_{0.05} < 0.05\)) in group 2 developed. In two years LVMM index increased by 13.4/17.5% in groups 1/2, respectively. Normal geometry and concentric remodeling completely disappeared in 3/1.5 years after PCIwS/CABG, respectively.

Conclusions: Within the 1st year after revascularization, patients with CABG had more severe LVH. In 5 years after PCIwS the ratio between concentric/eccentric LVH was 2:1, whereas after CABG = 1:2.

KEY WORDS: coronary artery disease, myocardial revascularization, coronary stenting, coronary artery bypass grafting, left ventricular geometry, left ventricular myocardial mass

INTRODUCTION
In patients with coronary artery disease (CAD) the period after myocardial revascularization is a complicated recovery process that may be assessed by echocardiographic parameters and left ventricular (LV) indices. The process of LV remodeling may last for several years after an acute cardiovascular event [1]. During the recovery period, left ventricular hypertrophy (LVH) is not always a positive phenomenon, because changes in LV geometry may cause disturbances of contractility and increased myocardial stiffness [2]. Late remodeling involves LV hypertrophy and dilatation, resulting in both systolic and diastolic dysfunction. It is being considered as an independent risk factor for cardiovascular complications [3]. Some prospective studies demonstrated the correlations between LV mass and chamber size and the risk of cardiovascular complications.

Despite the presence or absence of traditional risk factors (e.g., dyslipidemia, overweight, diabetes mellitus, high blood pressure, endogenous intoxication, older age), the incidence of cardiovascular complications is doubled in patients with LVH, comparing to those without [4, 5, 6, 7]. LV wall thickening by 1 mm above the upper reference limit increases the risk of sudden cardiac death by 7 times, as well as the incidence of acute coronary syndrome and the need for myocardial revascularization [5, 8].

Usually patients stay under the supervision of a surgeon or an interventional cardiologist within several months after a revascularization procedure with further long-term surveillance by cardiologist in outpatient setting. To our knowledge, only few studies analyzed the influence of LV remodeling after myocardial revascularization on further course of CAD [9, 10] that prompted our study.

THE AIM
The aim of the study was to estimate the dynamics of echocardiographic parameters in patients with CAD during a 5-year period after myocardial revascularization.

MATERIALS AND METHODS
The study had been carried out at the clinical bases of the Department of Internal Medicine No 2 Danylo Halytsky Lviv National Medical University. After obtaining a written consent in accordance with the principles of the Declaration of Helsinki, European Convention on Human Rights and Biomedicine, and relevant laws of Ukraine, fifty patients with CAD (39 men and 11 women; mean age 59.9±9.3 years) were included into the study. Standard recommended examination revealed ST-Elevation Myocardial Infarction (STEMI) with Q wave in 48%, STEMI without Q wave in 28%, non-STEMI in 24%. Depending on the type of revascularization, all partici-
Table I. Echocardiographic parameters during a 5-year follow-up period after myocardial revascularization

<table>
<thead>
<tr>
<th>Time, month</th>
<th>RVD, cm</th>
<th>LAD, cm</th>
<th>IVST, cm</th>
<th>PWT, cm</th>
<th>LVID, cm</th>
<th>ARD, cm</th>
<th>LVEF, %</th>
<th>LVMM, g</th>
<th>iLVMM, g/m²</th>
<th>RWT, units</th>
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<td>3.72±0.09</td>
<td>1.12±0.03</td>
<td>1.06±0.03</td>
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<td>3.12±0.06</td>
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<td>223±12</td>
<td>107±6</td>
<td>0.45±0.01</td>
</tr>
<tr>
<td>3</td>
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<td>3.83±0.12</td>
<td>1.12±0.03</td>
<td>1.07±0.03</td>
<td>4.87±0.13</td>
<td>3.05±0.07*</td>
<td>54±3</td>
<td>236±17</td>
<td>126±11</td>
<td>0.44±0.02</td>
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<td>6</td>
<td>2.30± 0.06</td>
<td>3.95±0.09</td>
<td>1.17±0.05</td>
<td>1.11±0.05</td>
<td>4.93±0.12</td>
<td>3.21±0.11</td>
<td>52±2</td>
<td>256±18*</td>
<td>127±9*</td>
<td>0.45±0.02</td>
</tr>
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<td>12</td>
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<td>4.03±0.10</td>
<td>1.28±0.05</td>
<td>1.14±0.05</td>
<td>5.07±0.13*</td>
<td>3.24±0.01</td>
<td>53±2</td>
<td>297±23*</td>
<td>122±9</td>
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<td>4.07±0.16</td>
<td>1.26±0.09</td>
<td>1.13±0.08</td>
<td>5.2±0.29 12</td>
<td>3.2±0.16</td>
<td>54±3</td>
<td>313±54</td>
<td>122±20</td>
</tr>
<tr>
<td>48</td>
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<td>4.17±0.24</td>
<td>1.22±0.09</td>
<td>1.14±0.06</td>
<td>5.2±0.21 13</td>
<td>3.13±0.16</td>
<td>55±5</td>
<td>299±43</td>
<td>133±19</td>
<td>0.44±0.01</td>
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<td>60</td>
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<td>1.12±0.04</td>
<td>5.2±0.24 15</td>
<td>3.33±0.24</td>
<td>47±4</td>
<td>385±53</td>
<td>176±20</td>
<td>0.44±0.01</td>
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<tr>
<td>Group 2</td>
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<td>1.23±0.06</td>
<td>1.13±0.04</td>
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<td>1.15±0.06</td>
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<td>3.39±0.15</td>
<td>52±3</td>
<td>311±17*</td>
<td>160±13</td>
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<tr>
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<td>3.18±0.14</td>
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<td>1.3±0.17</td>
<td>1.25±0.12</td>
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<td>3.48±0.21</td>
<td>54±5</td>
<td>351±42*</td>
<td>176±20*</td>
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<td>47±4</td>
<td>325±43</td>
<td>171±25</td>
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<td>45±4</td>
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<td>48</td>
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<td>5.5±0.35</td>
<td>3.42±0.17</td>
<td>48±5</td>
<td>317±22</td>
<td>155±8</td>
<td>0.40±0.06</td>
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<td>60</td>
<td>2.50± 0.10</td>
<td>4.7±0.42</td>
<td>1.2±0.15</td>
<td>1.07±0.07</td>
<td>6.1±0.44</td>
<td>3.57±0.19</td>
<td>45±3</td>
<td>363±43</td>
<td>164±38</td>
<td>0.35±0.04</td>
</tr>
</tbody>
</table>

Notes: * – intergroup differences (P<0.05); 1-28 – comparison of baseline and dynamic parameters within the group (P<0.05).
Figure 1. The prevalence of geometric LV patterns in patients after stenting (A) and CABG (B)
Notes: 1–7 – comparison of baseline and dynamic parameters within the group (P<0.05).
RESULTS AND DISCUSSION

Although both groups did not significantly differ by the age, co-morbidity, blood pressure levels, heart rate and body mass index, some echocardiographic parameters and their dynamic changes were significant (Table I).

Immediately after myocardial revascularization, group 2 patients had significantly more severe LV hypertrophy/dilatation and left atrial enlargement according to their values of LVMM, IVLMM, IVST, LVID, and LAD than group 1 patients. Prominent cardiac remodeling in group 2 patients may be explained by more spread atherosclerosis as demonstrated by coronary angiography. Several studies showed that coronary atherosclerosis often results in LVH [4, 12]. However, after 24 months and during the rest of follow-up period, no significant differences between groups were observed (Table I), regardless the type of revascularization.

Estimation of the 5-year dynamics of echocardiographic parameters revealed no significant changes in the RVD, RWT and LVEF, although the tendency to negative changes was still being traced in both groups. Left heart chambers (i.e., LAD, IVST, LVID, and LVMM) in group 1 patients were increasing gradually with a significant difference already in a year after procedure. Thus, the left atrial size in 12 months was above reference ranges – 4.03 ± 0.10 cm, and continued to enlarge: in 2 years – 4.07 ± 0.16 cm (P <0.05), in 3 years – 4.17 ± 0.2 cm (P <0.05), in 5 years – 4.37 ± 0.22 cm (P <0.05). Atrial remodeling is simultaneously a consequence and an evidence of diastolic dysfunction; it predisposes to lung congestion and arrhythmias, mainly atrial fibrillation.

Similar changes in the LV structure and geometry had been observed. LVID values increased significantly, from 4.71 ± 0.08 cm to 5.07 ± 0.13 cm in 12 months (P <0.05), 5.2 ± 0.29 cm in 2 years (P <0.05), and 5.57 ± 0.18 cm in 5 years (P <0.05). Patients after PCIwS initially had slightly increased values of IVST (1.2 ± 0.03 cm) and PWT (1.06 ± 0.03 cm). In one year after procedure the value of IVST significantly increased (1.28 ± 0.05 cm, P <0.05), and in 5 years its value had become 25% higher than baseline (1.40 ± 0.05 cm, P <0.05). The mean value of PWT also gradually increased, however, more slowly than IVST, and the difference became significant only in 5 years after PCIwS (1.23 ± 0.07 cm, P <0.05). Besides, a significant increase in LVMM and its index had been found after CABG significantly increased only in 5 years, partic-

larly LVID (from 5.11 ± 0.24 to 6.13 ± 0.44 cm, P <0.05) and ARD (from 3.17 ± 0.10 to 3.57 ± 0.19 cm, P <0.05). Besides, in 3 years after CABG a tendency toward moderate reduction of LVEF was observed (from 52 ± 3% to 45 ± 4%, P <0.05).

Our results suggest the constant progression of LVH and changes in the type of LV remodeling after both types of revascularization. According to the literature, patients with CAD usually have enlargement of LV sizes prior to surgery and at all stages of observation after CABG with increase in iLVMM by 8% till the end of the second year [13]. In our study during the same period of time, the LVMM index increased by 13.4% and 17.5% in groups 1 and 2, respectively.

The prevalence of different geometric patterns of LV remodeling also had been estimated during 5 years of observation (Fig. 1). It is known that abnormal LV geometry is associated with worse prognosis, e.g., increased risks of systolic and/or diastolic LV dysfunction, cardiovascular events, and death [14]. The highest risk and worst prognosis had patients with a concentric LVH [15].

All four LV geometric patterns were encountered in both groups prior to revascularization. Both normal geometry and concentric remodeling were absent in all patients in 3 years after PCIwS, whereas in those after CABG it was observed already in 1.5 years. During the last two years of the observation, no changes in the prevalence of LV geometric patterns had been observed in both groups (Fig. 1).

Among group 1 patients, concentric LVH occurred 2.8 times more often than normal LV geometry and 4.7 times more often than eccentric LVH (45.2%, 16.1%, and 9.7%, respectively); in 5 years its prevalence increased by 21.5% (from 45.2% to 66.7%). Besides, 12 months after procedure, the prevalence of concentric remodeling reduced by 4.9 times (from 29.0% to 5.9%, p<0.05), and in 3 years this type of geometry was not detected at all. Thus, in 5 years after PCIwS 2/3 patients had a concentric LVH, and 1/3 an eccentric LVH associated with LV dilatation.

The prevalence of geometric LV patterns in group 2 differed from that in group 1, although at the beginning it looked the same: there were significantly more patients with eccentric LVH (54.5%) that was 6 times higher than the prevalence of normal geometry and concentric LVH. The percentage of concentric LVH steadily increased, reaching statistical significance in 6 months, i.e., 6.9 times (Fig. 1). Subsequently, the percentage of eccentric LVH began to increase, suggesting the emergence of secondary remodeling, namely, LV dilatation. These findings somewhat contradicted the reports of other authors, which indicated that 24% of patients after revascularization had concentric remodeling [16]. We, in turn, noted that during the third year of observation all patients had LVH.

CONCLUSIONS

Immediately after CABG, patients with CAD had more severe LVH than patients after PCIwS, but starting from the second year of the observation these differences became not significant. In 5 years 2/3 of patients after PCIwS had the concentric LVH and 1/3 eccentric LVH, whereas those after CABG had opposite percentage, i.e., 2/3 eccentric LVH and 1/3 concentric LVH.

Perspective is further study of cardiac structure and function after revascularization, depending on the level of biomarkers.
REFERENCES


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Conflicts of interest:
Authors declare no conflict of interest.

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