ORIGINAl ARTiClE

Left atrial volume index as a predictor of left ventricular remodeling in patients with anterior STEMI treated with primary PCI

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KEYWORDS
Left atrial volume; Myocardial infarction; Remodeling

Abstract Background: Left ventricular remodeling (LVR) after myocardial infarction (MI) is associated with adverse cardiovascular events. Left atrial volume index (LAVI) was emerged as a prognostic factor for cardiovascular outcome. The aim of the study was to assess the value of measuring LAVI at discharge as a predictive factor for the development of LVR after anterior ST-elevation MI (STEMI) treated with primary percutaneous coronary intervention (PPCI).

Methods: The study included 100 consecutive patients with first anterior STEMI successfully treated with primary percutaneous coronary intervention (PPCI). Echocardiographic evaluation was performed at the time of discharge and at 6 months. LVR was defined as >20% increase in LVEDVI at 6 months compared with that at discharge.

Results: The prevalence of LVR was 31%. Patients who developed LVR had higher LVESVI at baseline, lower EF%, higher WMSI, higher LAVI, and longer time to perfusion. Multivariable logistic regression analysis showed that LAVI was the only predictive factor for LVR ($p = 0.003$, 95% CI = 1.87–19.7). ROC curve analysis showed that LAVI was predictive for LVR with a cut-off value 38 ml/m$^2$ (sensitivity 96.8% and specificity 77%, $p = 0.001$, 95% CI = 0.93–0.99).

Conclusion: LAVI is a simple and non-invasive echocardiographic marker that can predict the development of LVR in patients with anterior STEMI treated with PPCI.

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1. Introduction

Left ventricular remodeling (LVR) following myocardial infarction (MI) includes complex alternations in the architecture of the left ventricle involving both the infarcted and non-infarcted areas.$^{1-4}$ These changes manifest clinically as changes in the chamber size, shape and function.$^{5}$ Progressive post-infarction left ventricular (LV) dilatation is associated with increased cardiovascular death$^{5}$ and the development of chronic heart failure.$^{2,6}$

Mechanical reperfusion using primary percutaneous coronary intervention (PPCI) was associated with reduction in the infarct size and preservation of LV function resulting in an improved in-hospital survival.$^{7}$
Left ventricular remodeling is associated with worsening of diastolic dysfunction. During ventricular diastole, the left atrium is directly exposed to the left ventricular pressure; therefore the left atrial size is affected by changes in the LV diastolic filling pressure. So, the left atrial volume increases with increasing severity of LV diastolic dysfunction.

Left atrial volume index (LAVI) was associated with adverse events after myocardial infarction.

The aim of this study was to determine the value of measuring LAVI at discharge as a predictor of LVR in patient with anterior (ST elevation MI) STEMI treated with PPCI.

2. Methods

- **Study population:**

  Consecutive 100 patients with recent first STEMI who underwent successful PPCI in Tanta University, cardiology department were prospectively enrolled in the study.

  The study inclusion criteria were as follows: (1) confirmed first acute STEMI based on the presence of typical angina pain more than 30 min, new ST-segment elevation at J point in 2 or more contiguous chest leads $\geq 0.2$ mV, and elevation in troponin or ck-MB and (2) onset of symptoms $< 12$ h before hospital admission.

  The exclusion criteria were as follows: (1) Unsuccessful PPCI (residual stenosis $> 30\%$ and or thrombolysis in myocardial infarction (TIMI) flow $< III$). (2) Atrial fibrillation or flutter. (3) Poor echogenic window. (4) Patients with mitral stenosis or more than mild mitral regurgitation. (5) Prior MI. (6) Cardiomyopathy. (7) Reinfarction during follow up course. (8) Prior Coronary artery bypass grafting (CABG) or percutaneous coronary intervention (PCI).

- **Angiographic procedure:**

  Coronary angiography and percutaneous coronary intervention were done through the femoral approach. All patients received the following regimen: (1) Clopidogrel 300–600 mg loading dose orally followed by maintenance dose of 75 mg/day. (2) Aspirin 300 mg followed by 81–325 mg/day. (3) During the procedure patients received unfractionated heparin (100 IU/kg), and the dose was reduced to (70 IU/kg) in case of administration of glycoprotein IIb/IIIa inhibitor (eptifibatide). (4) All patients received FDA approved bare metal stents (BMS).

  At the end of the procedure TIMI flow rate$^{13}$ and myocardial blush grade (MBG)$^{14}$ were assessed.

- **Echocardiographic evaluation:**

  All patients underwent two dimensional transthoracic echocardiographic and Doppler studies using the commercially available GE Vivid 7 echocardiograph with 2.5 MHz transducer and echocardiographic evaluation was performed at the time of discharge and after 6 months. At discharge, echocardiography was done to evaluate LV ejection fraction (EF), LV end-systolic volume, LV end diastolic volume (using biplane method of disks). LV volume indices were measured by dividing the volume by the body surface area (BSA). Wall motion score index (WMSI) was obtained in all patients, the LV was divided into 16-segments model, and the score for each segment was graded according to the following system: normal, 1; hypokinesia, 2; akinesia, 3; dyskinesia, 4. The total wall motion score (WMSI) was obtained by adding the score for each segment. The WMSI was calculated by dividing the total wall motion score by 16.$^{16}$

  From the trans-mitral flow profile, the E and A waves peak velocities were calculated. Doppler tissue imaging of the mitral annulus was performed in the apical 4 chamber view using 1- to 2-mm sample volume placed in the septal mitral valve annulus. The value of e was measured and E/e was obtained.$^{17}$

  Left atrial volume was measured in the apical 4-chamber view at the ventricular end-systole from the frame preceding mitral valve opening using the biplane methods of disks, left atrial borders were traced using planimetry excluding pulmonary veins and left atrial appendages, and left atrial volume index was calculated by dividing LAV by (BSA)$^{18}$.

  At 6 month follow up 2D echocardiography was repeated to evaluate LVEDVI, LVESVI and EF. Left ventricular remodeling was defined as $> 20\%$ increase in LVEDVI at 6 months compared with that at discharge.$^{3}$

3. Results

One hundred patients with first anterior wall STEMI, who were treated with PPCI were included in the study.

The study population was divided into two groups according to the presence or absence of LVR. The “+ve remodeling” group included 31(31%) patients who developed LVR and the “–ve remodeling” group included 69(69%) patients who did not develop LVR.

3.1. Baseline clinical characteristics

Neither groups showed any significant differences regarding age, sex, BSA, hypertension, dyslipidemia, diabetes mellitus nor family history of premature coronary artery disease. Both groups did not differ regarding major medications prescribed at discharge (Table 1).

Table 1 Baseline clinical characteristics.

<table>
<thead>
<tr>
<th></th>
<th>+ve remodeling (N = 31, 31%)</th>
<th>–ve remodeling (N = 69, 69%)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>N</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>Age (year)</td>
<td>57.4 ± 7.6 years</td>
<td>60.6 ± 8.7 years</td>
<td>0.09</td>
</tr>
<tr>
<td>Male</td>
<td>24</td>
<td>55</td>
<td>0.7</td>
</tr>
<tr>
<td>Hypertension</td>
<td>16</td>
<td>29</td>
<td>0.3</td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td>11</td>
<td>29</td>
<td>0.5</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>7</td>
<td>16</td>
<td>0.49</td>
</tr>
<tr>
<td>Smoker</td>
<td>10</td>
<td>21</td>
<td>0.8</td>
</tr>
<tr>
<td>Family history of premature CAD</td>
<td>4</td>
<td>6</td>
<td>0.5</td>
</tr>
<tr>
<td>BSA</td>
<td>1.95 ± 0.16</td>
<td>1.90 ± 0.16</td>
<td>0.2</td>
</tr>
<tr>
<td>Major medications</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beta-blocker</td>
<td>28</td>
<td>90.6</td>
<td>0.7</td>
</tr>
<tr>
<td>ACEI or ARB</td>
<td>30</td>
<td>96.7</td>
<td>0.6</td>
</tr>
<tr>
<td>Statin</td>
<td>29</td>
<td>93.5%</td>
<td>0.6</td>
</tr>
<tr>
<td>Spironolactone</td>
<td>29</td>
<td>93.5%</td>
<td>0.4</td>
</tr>
</tbody>
</table>

CAD = Coronary artery disease. ACEI = angiotensin-converting enzyme inhibitor. ARB = Angiotensin II receptor blocker.
### 3.2. Echocardiographic characteristics

Regarding the baseline echocardiographic characteristics, both groups did not differ with respect to LVEDVI at baseline, peak E, peak A, or E/e₀ ratio. LVESVI at baseline, LAVI and WMSI, were significantly higher and EF% was significantly lower, in the “+ve remodeling” group (\( p = 0.001, 0.0001, 0.001 \) and 0.002 respectively) (Table 2).

### 3.3. Angiographic characteristics

There was no significant differences between both groups regarding, the number of diseased vessels, rate of use of G IIb/IIIa inhibitors, rate of use of thrombus aspiration device, reference vessel diameter, stent length or stent diameter. The time to perfusion was significantly longer in the “+ve remodeling” group (\( p = 0.01 \)) (Table 4).

Multivariable logistic regression analysis to assess factors predictive of LVR included LVESVI, LAVI, EF, WMSI and time to perfusion, and showed that LAVI was the only predictive factor for LVR (odds ratio = 6.07, 95% confidence interval, 1.87–19.7) (Table 5).

Roc curve analysis showed that LAVI was predictive for LVR with a cut-off value 38 with sensitivity 96.8% and specificity 77% (Fig. 1).

### 4. Discussion

The benefits of PPCI in patients with STEMI have been related to the achievement of early patency of the infarct related artery leading to better myocardial salvage compared with thrombolytic treatment.\(^1\)

The favorable effects of the early reperfusion limit the remodeling process or at least attenuate its effects on the left ventricular shape and geometry resulting in improved survival.\(^2\)

LVR occurs in a relevant proportion of patients despite successful treatment with PPCI and is an important predictor

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**Table 2** Baseline echocardiographic characteristics.

<table>
<thead>
<tr>
<th></th>
<th>+ve remodeling (N = 31, 31%)</th>
<th>−ve remodeling (N = 69, 69%)</th>
<th>( P ) value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ESVI – at baseline (ml/m²)</td>
<td>47.8 ± 15.75</td>
<td>36.6 ± 9.8</td>
<td>0.001*</td>
</tr>
<tr>
<td>EDVI – at baseline (ml/m²)</td>
<td>87.5 ± 16.2</td>
<td>81.8 ± 12.3</td>
<td>0.05</td>
</tr>
<tr>
<td>EF%</td>
<td>48.9 ± 6.8</td>
<td>53.6 ± 5.2</td>
<td>0.002*</td>
</tr>
<tr>
<td>Left atrium volume index (ml/m²)</td>
<td>42.9 ± 3.7</td>
<td>34.33 ± 3.9</td>
<td>0.0001*</td>
</tr>
<tr>
<td>Wall motion score index</td>
<td>1.58 ± 0.25</td>
<td>1.42 ± 0.21</td>
<td>0.001*</td>
</tr>
<tr>
<td>Peak E (m/s)</td>
<td>0.732 ± 0.1</td>
<td>0.73 ± 0.1</td>
<td>0.9</td>
</tr>
<tr>
<td>Peak A (m/s)</td>
<td>0.777 ± 0.1</td>
<td>0.78 ± 0.11</td>
<td>0.8</td>
</tr>
<tr>
<td>E/e₀ ratio</td>
<td>11.2 ± 2.47</td>
<td>10.94 ± 1.47</td>
<td>0.6</td>
</tr>
</tbody>
</table>

ESVI = end-systolic volume index. EDVI = end-diastolic volume index. EF = Ejection fraction. E: peak mitral valve flow velocity during the early rapid filling phase; A: peak mitral valve flow velocity during atrial contraction. E/e₀, the ratio of early transmural velocity to the early mitral annular velocity.

* Significant \( P \) value.

**Table 3** Six-month follow-up echocardiographic characteristics.

<table>
<thead>
<tr>
<th></th>
<th>+ve remodeling (N = 31, 31%)</th>
<th>−ve remodeling (N = 69, 69%)</th>
<th>( P ) value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVESVI-6 month (ml/m²)</td>
<td>57.36 ± 19</td>
<td>37.4 ± 10</td>
<td>0.001*</td>
</tr>
<tr>
<td>LVEDVI-6 month (ml/m²)</td>
<td>109.3 ± 22</td>
<td>78.79 ± 19</td>
<td>0.01*</td>
</tr>
<tr>
<td>EF-6 month (ml/m²)</td>
<td>48.5 ± 6.2</td>
<td>54.5 ± 5.3</td>
<td>0.001*</td>
</tr>
</tbody>
</table>

ESVI = end-systolic volume index. EDVI = end-diastolic volume index. EF = Ejection fraction.

* Significant \( P \) value.

**Table 4** Angiographic characteristics.

<table>
<thead>
<tr>
<th></th>
<th>+ve remodeling (N = 31, 31%)</th>
<th>−ve remodeling (N = 69, 69%)</th>
<th>( P ) value</th>
</tr>
</thead>
<tbody>
<tr>
<td>RD</td>
<td>2.6 ± 0.55</td>
<td>2.7 ± 0.4</td>
<td>0.4</td>
</tr>
<tr>
<td>Stent length</td>
<td>22.9 ± 4.8</td>
<td>21.5 ± 5.3</td>
<td>0.1</td>
</tr>
<tr>
<td>Stent diameter</td>
<td>3.17 ± 0.37</td>
<td>3.13 ± 0.35</td>
<td>0.975</td>
</tr>
<tr>
<td>Time of perfusion (mins)</td>
<td>254.9 ± 123</td>
<td>202 ± 87.9</td>
<td>0.01*</td>
</tr>
<tr>
<td>G IIb/IIIa inhibitors</td>
<td>25 (80.6%)</td>
<td>57(84%)</td>
<td>0.7</td>
</tr>
<tr>
<td>Thrombus aspiration</td>
<td>12 (38.7%)</td>
<td>25(36.2%)</td>
<td>0.8</td>
</tr>
<tr>
<td>MB grade</td>
<td>0 (6.4%)</td>
<td>7 (10.1%)</td>
<td>0.17</td>
</tr>
<tr>
<td>1</td>
<td>6 (19.4%)</td>
<td>27 (39.1%)</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>10 (32.3%)</td>
<td>14 (20.4%)</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>13(41.9%)</td>
<td>21(30.4%)</td>
<td></td>
</tr>
</tbody>
</table>

RD = reference diameter. G IIb/IIIa inhibitors = glycoprotein IIb/IIIa inhibitor. MB = myocardial blush.

Significant \( P \) value.
of mortality and development of heart failure.\textsuperscript{3,5,21} Therefore, identifying patients who are prone to develop progressive post-infarction left ventricular dilatation who are at increased risk to have adverse cardiovascular events, may have important therapeutic implication.

Many factors have been studied in previous studies to predict LVR.\textsuperscript{6,22–25} This study was undertaken to evaluate the value of measuring LAVI (which is a simple and non-invasive echocardiographic marker) at discharge as a predictive factor for the development of LVR after anterior STEMI.

Left atrial enlargement becomes a powerful predictor of long term outcome, and left atrial dilatation has been shown to predict both all-cause and cardiovascular mortality\textsuperscript{11,26–28} atrial fibrillation\textsuperscript{29–33} and heart failure.\textsuperscript{34,35} Left atrial dilatation was also evaluated in certain groups of patients like those with ischemic\textsuperscript{36} dilated,\textsuperscript{37,38} or hypertrophic cardiomyopathy,\textsuperscript{39} atrial arrhythmias,\textsuperscript{40} acute myocardial infarction\textsuperscript{12,41–43} and in patients with asymptomatic severe aortic stenosis.\textsuperscript{44}

In the present study LVR occurred in 31% of patients despite successful treatment with PPCI, a rate close to that published in previous studies.\textsuperscript{1,21}

Our data showed that patients with LVR have higher LVESVI at baseline, lower EF%, higher WMSI, higher LAVI, and longer time to perfusion.

Multivariable logistic regression analysis showed that LAVI was the only predictive factor for LVR. ROC curve analysis showed that LAVI was predictive of LVR with a cut-off value 38 with sensitivity 96.8% and specificity 77%.

The clinical usefulness of LAVI at discharge as a predictive factor for LVR after anterior STEMI may be explained by the fact that LVR is associated with progression of diastolic dysfunction. Deterioration of diastolic dysfunction is associated with a rise in LVEDP.\textsuperscript{8} As LAVI is influenced by LV filling pressure, it may be a powerful predictor for the development of LVR.

Furthermore, in response to decreased LV compliance, LA pressure rises, leading to increased LA wall tension. The secretion of cardiac peptides increases in response to both LA stretch and increased LV pressure.\textsuperscript{45}

5. Conclusion

The present study demonstrates that LAVI may predict LVR after successful PPCI of anterior STEMI. Therefore measurement of LAVI at discharge could emerge as a simple tool to risk stratify and guide therapy in patients with anterior STEMI.

Conflict of interest

The authors declare that they have no conflict of interest.

References

Left atrial volume index as a predictor of left ventricular remodeling


