Cardiac resynchronization therapy restored ventricular septal myocardial perfusion and enhanced ventricular remodeling in patients with nonischemic cardiomyopathy presenting with left bundle branch block

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Cardiac Resynchronization Therapy (CRT)
The efficacy of CRT was observed only among LBBB patient.

Why do patients with LBBB greatly benefit from CRT?
45 yo male: Chest discomfort during exercise

LVEF 29%
Diffuse hypokinesis

DM, HT, dyslipidemia +
Myocardial perfusion SPECT with Technetium-99m sestamibi
Coronary angiography
Objective

To investigate whether CRT changes septal myocardial perfusion change of septal myocardial perfusion has any relationships for LV reverse remodeling
Inclusion criteria

- NYHA Class II, III or IV
- LV ejection fraction <35%
- QRS duration >120msec
- Non-ischemic HF
- LBBB
- Sinus rhythm

CRT implantation

Myocardial perfusion SPECT with technetium-99m sestamibi was performed before and 6 months after CRT.
Mean count rate with 30 pixels in the LV septal and lateral wall was calculated.

**LV septal perfusion index (LV SPI)**

\[
\text{LV SPI} = \frac{\text{mean count rate (septum)}}{\text{mean count rate (lateral)}}
\]

\[
\text{LV SPI} < 0.9 : \text{LV septal perfusion defect}
\]
Methods

Following parameters were evaluated as CRT efficacy:

- NYHA Class, QRS duration, LVEF, LVESV and LVEDV

**Parameter for LV reverse remodeling:** Reduction of LVESV

\[
\text{Reduction of LVESV} = \frac{\text{LVESV (baseline)} - \text{LVESV (6 months after CRT)}}{\text{LVESV (baseline)}} \times 100
\]

**Parameter for myocardial perfusion SPECT:** Improvement of LV SPI

\[
\text{Improvement of LV SPI} = \frac{\text{LVSPI (6 months after CRT)} - \text{LVSPI (baseline)}}{\text{LVSPI (baseline)}}
\]
## Results

### Enrollment N=26

<table>
<thead>
<tr>
<th></th>
<th>LVSPI&gt;0.9</th>
<th>LVSPI&lt;0.9</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>perfusion defect (-)</td>
<td>N=7 (27%)</td>
<td>N=19 (73%)</td>
<td></td>
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</tbody>
</table>

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<thead>
<tr>
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</thead>
<tbody>
<tr>
<td>Sex: male</td>
<td>5 (71.4)</td>
<td>10 (52.6)</td>
<td>0.658</td>
</tr>
<tr>
<td>Age (year)</td>
<td>73 ± 8</td>
<td>69 ± 12</td>
<td>0.543</td>
</tr>
<tr>
<td>NYHA Class II</td>
<td>2 (28.6)</td>
<td>3 (15.8)</td>
<td>0.255</td>
</tr>
<tr>
<td>Class III</td>
<td>5 (71.4)</td>
<td>13 (68.4)</td>
<td></td>
</tr>
<tr>
<td>Class IV</td>
<td>0 (0.0)</td>
<td>3 (15.8)</td>
<td></td>
</tr>
<tr>
<td>QRS duration (ms)</td>
<td>170.0 ± 36.8</td>
<td>171.1 ± 36.7</td>
<td>0.468</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>29.4 ± 5.7</td>
<td>23.1 ± 10.1</td>
<td>0.183</td>
</tr>
<tr>
<td>LVEDV (mL)</td>
<td>171.9 ± 75.4</td>
<td>261.2 ± 139.1</td>
<td>0.174</td>
</tr>
<tr>
<td>LVESV (mL)</td>
<td>113.7 ± 65.6</td>
<td>210.7 ± 137.7</td>
<td>0.078</td>
</tr>
<tr>
<td>ACE-I/ARB</td>
<td>7 (100.0)</td>
<td>14 (73.7)</td>
<td>0.278</td>
</tr>
<tr>
<td>β-blockers</td>
<td>7 (100.0)</td>
<td>13 (68.4)</td>
<td>0.146</td>
</tr>
<tr>
<td>Diuretics</td>
<td>5 (71.4)</td>
<td>14 (84.2)</td>
<td>0.588</td>
</tr>
</tbody>
</table>

Perfusion defect (+) N=19 (73%)

Perfusion defect (-) N=7 (27%)
## Results

<table>
<thead>
<tr>
<th></th>
<th>perfusion defect (-) (n=7)</th>
<th>perfusion defect (+) (n=19)</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Before CRT</td>
<td>After CRT</td>
</tr>
<tr>
<td><strong>NYHA Class</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>0 (0.0)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>II</td>
<td>2 (28.6)</td>
<td>6 (85.7)</td>
</tr>
<tr>
<td>III</td>
<td>5 (71.4)</td>
<td>1 (14.3)</td>
</tr>
<tr>
<td>IV</td>
<td>0 (0.0)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td><strong>QRS duration (ms)</strong></td>
<td>160.0±38.6</td>
<td>137.9±0.2</td>
</tr>
<tr>
<td><strong>LVEF (%)</strong></td>
<td>29.4±5.8</td>
<td>28.0±8.3</td>
</tr>
<tr>
<td><strong>LVEDV (mL)</strong></td>
<td>171.9±75.4</td>
<td>153.7±64.6</td>
</tr>
<tr>
<td><strong>LVESV (mL)</strong></td>
<td>113.7±65.6</td>
<td>106.1±57.4</td>
</tr>
<tr>
<td><strong>Septal perfusion index</strong></td>
<td>106.2±14.2</td>
<td>117.8±32.3</td>
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77 yrs Female: DCM

Pre-CRT: LVSPI = 0.71

Post-CRT: LVSPI = 1.03

Ogano M et al. Heart Rhythm 2014:5:836-841
Results

Ogano M et al. Heart Rhythm 2014:5:836-841

Improvement of LVSP1

Reduction of LVESV

\[ r = 0.561 \]

\[ p = 0.012 \]
Summary

1: 70% of the patients with non-ischemic HF and LBBB revealed LV septal perfusion defect.

2: The LV septal perfusion defect group showed better CRT response than did the non-LV septal perfusion defect group.

3: The improvement ratio of LV septal perfusion index significantly correlated with LV volumetric reverse remodeling.
Delayed contraction of LV lateral wall in LBBB results in increased pressure load to septal wall and impairs septal coronary blood flow.
Intramyocardial pressure (IMP) in septum and LAD flow

LBBB and Coronary flow reserve (CFR)

Group I: LBBB (+)、Septal hypoperfusion (+)

Group II: LBBB (+)、Septal hypoperfusion (―)

Control: LBBB (―)

Skalidis E. et al. J Am Coll Cardiol. 1999;33;1338-46
Coronary artery disease
- PCI/CABG
  - Reduced coronary flow
  - Myocardial hibernating

LBBB
- CRT
  - Reduced LAD flow
  - Impairment of coronary microvascular dysfunction
LBBB and Coronary flow reserve (CFR)

Group I: LBBB (+) 、 Septal hypoperfusion (+)

Group II: LBBB (+) 、 Septal hypoperfusion (−)

Control: LBBB (−)

Skalidis E. et al. J Am Coll Cardiol. 1999;33;1338-46
LBBB(+)  
Septal hypoperfusion(−)
There is a heterogeneity among LBBB pattern.
LBBB pattern is qualitatively equivalent to induce large intra-LV pressure burden for septum.
Requisite for CRT responder

- Burden of cardiac dyssynchrony
- Potential to improve cardiac function

Perfusion defect (+)

Perfusion defect (-)

Responder wall

Burden of cardiac dyssynchrony
Conclusions

A latent and reversible myocardial perfusion defect at the LV septum was confirmed in patients with non-ischemic cardiomyopathy and LBBB.

CRT improved myocardial perfusion and enhance LV reverse remodeling in these patients.
Thank you for your attention