importance of the serum LOX-1 levels in the diagnosis and assessment of left
ventricular systolic heart failure and its relationship with serum pro-BNP.
Methods: Fifty-five patients with a diagnosis of systolic heart failure and 25 patients
without systolic heart failure were enrolled in this study. The study took place in the
department of cardiology at Uludag University School of Medicine between October
2011 and April 2012. Echocardiography was performed in all cases. Serum C-reactive
protein, pro-BNP and LOX-1 levels were studied.
Results: Serum LOX-1 and pro-BNP levels were significantly higher in the heart
failure group and showed negative correlations with left ventricular ejection fraction.
However, there was no significant correlation between serum LOX-1 and pro-BNP
levels. In addition, LOX-1 level in patients with ischemic cardiomyopathy was
significantly higher than the patients with dilated cardiomyopathy. ROC analysis was
done for the studied sample of serum LOX-1, the ‘cut off’ level was determined as
1.31 ng/ml for LOX-1 giving a sensitivity of 56.3% and specificity of 96% for the
diagnosis of the systolic heart failure.
Conclusion: Our study demonstrates the utility of the serum LOX-1 levels in the
diagnosis of left ventricular systolic heart failure. LOX-1 may have an important place
in the diagnosis of heart failure, especially when the etiology is ischemic cardiomy-
opathy. Further prospective studies with larger sample sizes are needed to better
understand the exact role of LOX-1 in the diagnosis and assessment of heart failure.

Table 1. The demographic and baseline clinical characteristics of the heart
failure and control groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>Heart failure group</th>
<th>Control group</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>64 (22-83)</td>
<td>62 (50-78)</td>
<td>0.306</td>
</tr>
<tr>
<td>Hypertension (n, %)</td>
<td>30 (54.5%)</td>
<td>14 (56%)</td>
<td>1.000</td>
</tr>
<tr>
<td>Hyperlipidemia (n, %)</td>
<td>21 (38.2%)</td>
<td>14 (56%)</td>
<td>0.323</td>
</tr>
<tr>
<td>Smoking (n, %)</td>
<td>9 (16.4%)</td>
<td>2 (8%)</td>
<td>0.511</td>
</tr>
<tr>
<td>Left ventricular ejection fraction (%)</td>
<td>27 (18-44)</td>
<td>65 (54-73)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Table 2. Laboratory data of the heart failure and control groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>Heart failure group</th>
<th>Control group</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood glucose (mg/dl)</td>
<td>91.4 (17.2)</td>
<td>90.9 (12)</td>
<td>0.882</td>
</tr>
<tr>
<td>Urea (mg/dl)</td>
<td>45 (21-99)</td>
<td>32 (19-44)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Creatinine (mg/dl)</td>
<td>0.9 (0.6-1.4)</td>
<td>0.7 (0.6-1.1)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>GFR (ml/min/1.73 m²)</td>
<td>84 (44-148)</td>
<td>96 (65-144)</td>
<td>0.012</td>
</tr>
<tr>
<td>Sodium (mg/dl)</td>
<td>137 (125-144)</td>
<td>141 (138-144)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Potassium (mg/dl)</td>
<td>4.3 (0.5)</td>
<td>4.4 (0.44)</td>
<td>0.602</td>
</tr>
<tr>
<td>Calcium (mg/dl)</td>
<td>9.0 (6.3-10.5)</td>
<td>9.5 (8.6-11.2)</td>
<td>0.001</td>
</tr>
<tr>
<td>Hemoglobin (g/dl)</td>
<td>127.1 ± 1.97</td>
<td>143 ± 0.86</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LOX-1 (ng/ml)</td>
<td>1.46 (0.56-4.09)</td>
<td>0.99 (0.58-1.7)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Pro-BNP (µg/ml)</td>
<td>3560 (211-20806)</td>
<td>97 (18-184)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>CRP (mg/L)</td>
<td>0.8 (0.3-5.5)</td>
<td>0.33 (0.05)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Table 3. Comparison of patients according to the etiology of heart failure

<table>
<thead>
<tr>
<th>Ejection fraction (%)</th>
<th>DCMP (n = 24)</th>
<th>ICMP (n = 31)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>25 (18-44)</td>
<td>30 (19-44)</td>
<td>0.165</td>
<td></td>
</tr>
<tr>
<td>LOX-1 (mg/ml)</td>
<td>1.16 (0.56-2.84)</td>
<td>1.65 (0.64-4.09)</td>
<td>0.027</td>
</tr>
<tr>
<td>Pro-BNP (µg/ml)</td>
<td>2806 (211-17277)</td>
<td>5200 (248-20806)</td>
<td>0.044</td>
</tr>
<tr>
<td>CRP (mg/L)</td>
<td>0.52 (0.3-3.5)</td>
<td>1.3 (0.35-5.4)</td>
<td>0.058</td>
</tr>
</tbody>
</table>

Table 1

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>EF (%)</th>
<th>Hypertension (%)</th>
<th>Diabetes mellitus (%)</th>
<th>Smoking (%)</th>
<th>Ischemic cardiomyopathy (%)</th>
<th>Neutrophil-to-lymphocyte ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>63.2 ± 12.7</td>
<td>26.5 ± 6.4</td>
<td>62.3</td>
<td>28.9</td>
<td>17.3</td>
<td>82.7</td>
<td>6.5 ± 4.7</td>
</tr>
</tbody>
</table>

Comparison of changes in hematological variables of the patients who survived (group 1) and
died (group 2) after levosimendan therapy

OP-167
Utility of the Neutrophil to Lymphocyte Ratio in Predicting In-hospital Mortality in Patients That Received Levosimendan Treatment for Acute Decompensated Heart Failure

Abdullahnam Taxel, Özgür Sarıgül, Ahmet Bacakci, Ömer Goktekin, Huseyin Uyarel, Mehmet Ergelen, Erkan Erdogan, Seref Kafı, Osman Sonmez, Murat Tarfan, Sitti Kucukbuzcu, Mehmet Akif Vatankulu, Aylin Hatice Yamac, Cemal Savcany, Ugur Berber, Mehmet Akif Ersoy

Aim: The aim of the study was to investigate the effect of levosimendan infusion on
hematological variables in patients with acute heart failure. Also, predictive value of
these variables over in hospital mortality evaluated.

Methods: Two hundred and nineteen patients (168 male, 51 female, mean age 63.2±12.7 years)
with acute exacerbation of advanced heart failure (ejection fraction ≤35%) were included in this study. Levosimendan was initiated as a bolus of 6 µg/kg
followed by a continuous infusion of 0.1 µg/kg/min for 24 hours. Changes of
hematological variables between admission and on third day after levosimendan
infusion were evaluated. Categorical variables were expressed as frequencies and
percentages. Continuous variables were compared using analysis of variance and
Kruskal-Wallis tests for those with normal and skewed distributions, respectively.
Chi-square tests were used to compare categorical variables. Univariate and multi-

Table 2

<table>
<thead>
<tr>
<th>Variables</th>
<th>Group 1</th>
<th>Group 2</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Δ WBC</td>
<td>0.5 ± 2.2</td>
<td>1.1 ± 5.1</td>
<td>0.216</td>
</tr>
<tr>
<td>Δ Neutrophil</td>
<td>3.3 ± 7.8</td>
<td>-1.1 ± 7.7</td>
<td>0.001</td>
</tr>
<tr>
<td>Δ Lymphocyte</td>
<td>-1.4 ± 6.1</td>
<td>1.1 ± 5.4</td>
<td>0.012</td>
</tr>
<tr>
<td>Δ NLR</td>
<td>1.3 ± 3.7</td>
<td>-2.7 ± 11.6</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Comparison of changes in hematological variables of the patients who survived (group 1) and
died (group 2) after levosimendan therapy

OP-168
Post-discharge Heart Failure Monitoring Program in Turkey: HitPoint

Yüksel Çavgunozlu, Mehdi Zoghi, Mehmet Eren, Evin Bocail, Guliz Kocazag, Tuncay Şentürk, Güray Aşıcı, Korhan Soylu, Ibrahim Sarı, Ahmet Temizhan, Okay Ergene, Özlem Soran

Izmir, Turkey

Introduction: Disease management programs for the treatment of patients with heart
failure (HF) have been advocated by society guidelines in order to improve patient
compliance and decrease hospitalizations. However, there are several different HF
management programs; most of them are costly and are not feasible to use in various
geographic areas. The aim of this study was to assess the efficacy and feasibility of
a cardiologist lead enhanced HF education at the time of hospital discharge with a 6
month phone follow-up program in chronic HF patients.

Methods: The Hit Point trial was a multicenter, randomized, controlled trial of
enhanced HF education with a 6 month phone follow-up program (EHFP) vs routine
management care (RC) in patients who carried the diagnosis of HF secondary to systolic
dysfunction, had been hospitalized for HF within six months of randomization, and
had symptoms despite optimal medical therapy. Education included information on
the adherence to treatment, symptoms recognition, diet and fluid intake, weight
monitoring, activity, exercise training and when to contact cardiologist. Patients were

C74  JACC Vol 62/18/Suppl C  October 26–29, 2013  TSC Abstracts/ORALS
contacted by phone at 1, 3, and 6 months. The primary study endpoint was cardiovascular death.

Results: A total of 248 patients from 10 centers in various geographical areas were randomized: 125 to EHF and 123 to RC at the time of hospital discharge. The mean age of all patients was 60.8 ± 13.8 years. The patients were predominantly men (73%) with NYHA class II-IV heart failure symptoms who had a mean ejection fraction of 26.8 ± 7.3%.

Demographic characteristics, including age, sex, laboratory evaluations and assessments of cardiac function, and functional capacity were equivalent in both treatment groups. Use of pharmacologic therapy at baseline in the patients demonstrated compliance with guidelines recommended therapy. Six-month cardiovascular mortality was significantly higher in the RC group (p < 0.05). At baseline 60% in EHF and 61% in RC were in NYHA Class III and IV; at 6-month follow up only 12% in EHF and 32% in RC were in NYHA Class III and IV. The mean number of hospitalization was 0.58 ± 0.91 in EHF and 0.64 ± 1.05 in RC (p = NS) and the mean number of emergency room visits was 0.84 ± 1.15 and 1.21 ± 1.47, respectively at 6-month follow-up (p < 0.05).

Conclusion: Our study results showed that an enhanced education lead by a cardiologist and number of emergency room visit within 6 months after discharge in HF patients.

Interventional Cardiology
Tuesday, October 29, 2013, 10:15 AM–11:30 AM Hall: LEFKOŠA
Abstract nos: 169-174

OP-169
Middle-term Results of Percutaneous Closure of Congenital Ventricular Septal Defect Using Different Devices
Ahmet Celebi, Ilker Kemal Yucel, Abdullah Erdem, Rehyan Dedegil, Mustafa Orhan Bulut, Aysan Cecit, Nurdan Erol
Department of Pediatric Cardiology, Dr Siyami Ersek Thoracic and Cardiovascular Surgery Center, Istanbul

Introduction: The middle-term results of VSD closure using different devices in our clinic are presented.

Patients and Methods: Patients undergoing transcatheter VSD closure in our clinic between April 2007 and June 2013 were reviewed. Defects were closed in cases with a large VSD on echocardiography, left chamber hypertrophy and hemodynamically a Qp/Qs > 1.5 and or reversible pulmonary hypertension. In perimembranous VSD the left disk of the occluder was placed on the left of the septum in the absence of aneurysm. If an aneurysm was present, the left disk was placed inside it, to reduce the risk of AV block.

Results: Median age of the patients was 8 (range, 10 months - 55 years); their average follow-up was 37 ± 20 months and average Qp/Qs 2.0 ± 0.63. Average VSD diameter was 9.1 ± 3.3 mm (3-20). Implantation was successful in 74 (92.5%) of the 80 patients. Of these, 51 had perimembranous and 23 muscular defects. For cases with perimembranous defects, an eccentric Amplatzer perimembranous VSD device was placed in 18, a Cardiofix muscular VSD device in 17, an Amplatzer or Cardiofix duct occluder in 9, an Amplatzer muscular VSD device in 3, a Lifetech muscular VSD device in another 3 and an ADO-II device in one patient. The device was placed within the aneurysm tissue in 18 cases. As for muscular VSD, Cardiofix muscular VSD device was used in 18 patients, an Amplatzer muscular VSD device in 2, a Lifetech muscular VSD in another 2 and a Cardiofix ASD occluder in one patient. Complete occlusion immediately post-intervention was achieved in 52% (39/74), 70% (52/74) on the next day and 85% (63/74) at the 6-month follow-up. The residual defect ratio was significantly higher where the device had been placed inside the aneurysm (p < 0.05). One patient had a reversible complete AV block and another one experienced hemolysis, no other major complications were observed. One patient underwent open surgery for a significant residual defect. During follow-up, two patient had de novo presentation of non-progressive, minimal aortic insufficiency.

Conclusion: VSD occlusion by different devices is safe and efficacious in selected patients. While placement of the device inside the aneurysm increases the proportion of residual shunt, it is believed that it may reduce the risk of AV block, the most feared complication.

OP-170
Percutaneous Closure of Perimembranous Ventricular Septal Defects Associated With Septal Aneurysm
Oktay Ergene1, Nihan Kaya Erent1, Hamza Dayigu2, Uğur Kocabuçak2, Cem Naçi2
1Docku Eyal University, Faculty of Medicine, Cardiology Department, İzmir, 2İzmir Katip Çelebi University Ataturk Education and Research Hospital, Cardiology Department, İzmir

Introduction: Ventricular septal defects (VSD) are the most common congenital heart disease. Of these defects 80% involves the membranous septum. With the introduction of eccentric perimembranous VSD (PmVSD) occluder devices, percutaneous closure of PmVSDs has become an accepted alternative to surgical closure. However, closure of PmVSDs associated with septal aneurysms is more challenging. We report our experience of device closure of PmVSD associated with septal aneurysm.

Material-Methods: Between 2008 and 2012, percutaneous closure of PmVSD associated with a septal aneurysm was attempted in 11 patients in our institution. The indication for VSD closure was the presence of hemodynamically significant PmVSD demonstrated by cardiac catheterisation (Qp/Qs > 1.5). We used 2 methods to occlude PmVSD associated with septal aneurysm: 1) to close the defect at the left ventricular opening of the aneurysm by anchoring the left disc of the occluder at the inlet portion of the aneurysm and the defect between the left and the right discs of the device; 2) to close the defect at the outlet by anchoring the left disc of the device at the left side of the outlet portion of the aneurysm. We preferred to use the first method when the aneurysm was small and there is adequate distance from the aortic valve. The patients were followed up at 1st, 3rd, 6th and 12th months after the closure procedure by TTE and ECG.

Results: Mean age of the patients was 36.2 ± 1.3 and 64% were male. The demographic and clinical characteristics of patients are shown in Table 1. The average diameter of the VSD was 5.9 ± 2.4 mm by angiography. One patient had 2 defects within the aneurysm and 1 patient had dextrocardia. Large aneurysm (the inlet portion of the aneurysm > 10 mm) was present in 7 patients. The procedure was successful in all patients. We used Amplatzer PmVSD occluder device in 3 patients, Amplatzer Muscular VSD occluder device in 5 patients, Amplatzer Duct occluder-I (ADO-I) in 1 patient and ADO-II device in 2 patients. We preferred to occlude the defect by the first method in 4 patients who had a small aneurysm and PmVSD occluder was used in 3 cases and a muscular VSD defect was present in 1 patient. Second method was preferred in 7 patients who had larger aneurysmas. A trivial residual shunt was detected by ventriculography in 4 patients immediately after the procedure. Complete closure was observed by transthoracic echocardiography in all patients at the time of discharge. The patients were followed-up at mean of 22.1 ± 1.9 months. There was no device or procedure related complications at the acute setting or midterm follow-up.

Conclusion: Percutaneous closure of PmVSDs associated with aneurysm is more challenging than simple defects. The selection of the device type and size should be made according to the configuration and size of the aneurysm and the defect.