Heart Failure: Adjunctive Therapies and Their Mechanisms

Abstract 2370: LDL Independent Reduction In Cardiovascular Morbidity And Mortality With Rosuvastatin In Heart Failure Patients With A Raised C-reactive Protein: A Retrospective Analysis Of The Controlled Rosuvastatin Multinational Trial In Heart Failure (CORONA)

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Objective: The anti-inflammatory action of statins may contribute to the clinical benefits of these drugs. Heart failure (HF) is an inflammatory state in which the usual epidemiologic relationship between cholesterol and cardiovascular outcomes is reversed, representing an excellent disease model in which to test the statin anti-inflammatory hypothesis.

Methods: We carried out a retrospective subgroup analysis of CORONA, which randomized 5011 patients with low ejection fraction HF of ischemic etiology to placebo or rosuvastatin 10 mg daily. We...
examined the effect of rosuvastatin according to baseline plasma high-sensitivity C-reactive protein concentration (hs-CRP) with patients divided into two groups: < 2 mg/L (779 placebo/777 rosuvastatin) versus >/= 2 mg/L (1694 placebo/1711 rosuvastatin). Results (table): Baseline LDL was the same in the two hsCRP groups (approx. 138 mg/dL) and was reduced equally by 45% in each group with rosuvastatin. In patients with a hsCRP >/= 2 mg/L, rosuvastatin treatment was associated with nominally statistically significant reductions in the primary outcome (cardiovascular death, myocardial infarction or stroke), all cause mortality and the pre-specified coronary endpoint (sudden death, fatal or nonfatal myocardial infarction, PCI or CABG, ventricular defibrillation by an ICD, resuscitation after cardiac arrest or hospitalization for unstable angina). Importantly, rosuvastatin also reduced the secondary outcome of HF hospitalizations: hsCRP < 2.0 mg/L 267 placebo/264 rosuvastatin (p n.s.); hsCRP >/= 2.0 mg/L 1015 placebo/827 rosuvastatin (p = 0.0044)  

Conclusions: Our findings in patients with HF support and extend previous retrospective analyses in patients with acute and stable coronary heart disease and add more evidence that the anti-inflammatory action of statins may be clinically important, not just in reducing atherosclerotic events but also HF hospitalizations.

<table>
<thead>
<tr>
<th>Primary endpoint</th>
<th>Placebo</th>
<th>Rosuvastatin</th>
<th>HR**</th>
<th>P value***</th>
</tr>
</thead>
<tbody>
<tr>
<td>hSCR &lt; 2.0</td>
<td>8.9</td>
<td>9.8</td>
<td>1.08 (0.88, 1.32)</td>
<td>0.062</td>
</tr>
<tr>
<td>hSCR &gt;/= 2.0</td>
<td>14.0</td>
<td>12.2</td>
<td>0.87 (0.77, 0.98)</td>
<td>0.066</td>
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<tr>
<td>All cause death*</td>
<td>8.3</td>
<td>9.7</td>
<td>1.16 (0.94, 1.42)</td>
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<td>hSCR &lt; 2.0</td>
<td>14.1</td>
<td>12.6</td>
<td>0.89 (0.79, 1.00)</td>
<td>0.026</td>
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<tr>
<td>hSCR &gt;/= 2.0</td>
<td>7.5</td>
<td>8.7</td>
<td>1.14 (0.91, 1.43)</td>
<td>0.023</td>
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</tbody>
</table>

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