

1173 Unstable Angina: Prognostic Determinants

Tuesday, April 01, 2003, Noon-2:00 p.m.
McCormick Place, Hall A
Presentation Hour: Noon-1:00 p.m.

Helicobacter Pylori Eradication Improves Prognosis of Coronary Artery Disease Through a Mechanism Not Related With Platelet Activation

Alessandro Sincie, Ignasi Elizalde, Magda Heras, Nuria Casanova, Salvador Diaz-Aja, Teresa Martorell, Miguel Lonzo, Sonia Perez, Julain Gonzalez, Ginés Sanz, Josep M. Pique, Hospital Clinic, Barcelona, Spain

Background: Epidemiological association between Helicobacter pylori (HP) infection and coronary artery disease (CAD) has been suggested, but underlying pathophysiological links remain unknown. Platelet activation and aggregation have been reported in animal models. This pilot randomized controlled trial evaluated the impact of HP infection and eradication on platelet activation and inflammatory markers in patients (pts) with CAD as well as the influence of HP status on subsequent adverse coronary events (cardiac death, angina or myocardial infarction).

Methods: Ninety survivors after an acute coronary event not requiring surgical revascularization were included. HP status was assessed with an urea breath test and performed at baseline and at two-months follow-up. HP positive pts were randomized to receive a 7-day course of omeprazole, amoxicillin, and metronidazole or corresponding placebo (randomization ratio 1:2). Pts were followed up for one year or until death or readmission with an acute coronary syndrome.

Results: No baseline differences were observed between HP positive (n = 49) and HP negative (n = 41) pts. Among HP positive pts, 18 received placebo and 31 active medication. HP eradication was assessed with an urea breath test in 21 cases. No differences were observed neither in inflammatory or platelet activation markers nor in anti-Chlamydia serology between pts with persistent or resolved HP infection. However, 15 and 12 months, a recurrent coronary event was observed in 35% and 55% of pts with persisting HP infection vs 10% and 25% of pts in whom HP was either absent or eradicated respectively (log-rank = 0.01, Breslow = 0.01). Only final HP status (RR 2.7 [95% Cl, 1.2-6.3]; p=0.014) and dyslipemia (RR 3.0 [95% Cl, 1.2-6.3]; p=0.046) were selected as independent predictors of recurrent coronary events. HP infection does not induce significant platelet activation in pts with CAD. HP infected pts have an increased probability to suffer a recurrent coronary event which may be reverted by eradicating the infection.

Baseline Creatinine Clearance Provides Additional Prognostic Information in TIMI Risk Score in Non-ST Elevation Acute Coronary Syndromes

Carlos T. Aauiar, Jorge Ferreira, Pedro A. Goncalves, Marisa Trabulo, Ricardo Seabra-Alessandro Sionis, Ignasi Elizalde, Magda Heras, Nuria Casanovas, Salvador D&z-A&z, Teresa Martorell, Miguel Lonzo, Sonia Perez, Julian Gonzalez, Ginés Sanz, Josep M. Pique, Hospital Clinic, Barcelona, Spain

Background: Recent clinical trials and guidelines support invasive risk stratification with early coronary angiography and revascularization for patients with Non-ST-elevation acute coronary syndromes (NSTE-ACS). The optimal timing of angiography from hospital admission however is uncertain; therefore, we retrospectively investigated whether timing of angiography affects mortality in patients with Non-ST-elevation ACS.

Methods: Study population consisted of 836 patients admitted to the coronary care unit between 1997-2000 with Non-ST-elevation acute coronary syndromes who underwent angiography during the same hospital stay. Patients were categorized into three groups based on the time interval between admission and angiography: <24 hours, 24-48 hours, and >48 hours. Six-month incidence of death and/or myocardial infarction was determined and compared between groups.

Results: Six-month outcome based on time to angiography in patients with Non-ST elevation acute coronary syndromes

Outcome Based on Time to Angiography in Patients With Non-ST Elevation Acute Coronary Syndromes

Khaled A. Daiani, Sanjaya Khanal, Mouaz Al-Mallah, Iyengar Hirshikesh, Noel Gutierrez, and Michael P. Hudson, Henry Ford Heart and Vascular Institute, Detroit, MI

Background: Recent clinical trials and guidelines support invasive risk stratification with early coronary angiography and revascularization for patients with Non-ST-elevation acute coronary syndromes (ACS). The optimal timing of angiography from hospital admission however is uncertain; therefore, we retrospectively investigated whether timing of angiography affects mortality in patients with Non-ST-elevation ACS.

Methods: Study population consisted of 836 patients admitted to the coronary care unit between 1997-2000 with Non-ST-elevation ACS who underwent angiography during the same hospital stay. Patients were categorized into three groups based on the time interval between admission and angiography: <24 hours, 24-48 hours, and >48 hours. Six-month incidence of death and/or myocardial infarction was determined and compared between groups.

Results: Table 1 below summarizes baseline characteristics and 6-month outcomes according to angiography group. Following multivariate adjustment, angiography group remained significantly associated with survival with angiography delay >48 hours associated with worse survival.

Conclusions: Younger age, male gender, absence of prior MI were associated with increased likelihood of angiography within 48 hours of hospital admission. Delay of angiography >48 hours was associated with worse survival while angiography delay 24-48 hours yielded optimal survival results.

Baseline Characteristics and Outcome

Table 1 Hazard ratios for anti-huhs60 and anti-myhcp65 in quartiles for the combined endpoint

<table>
<thead>
<tr>
<th>Variable</th>
<th>&lt;24hrs (N=314)</th>
<th>24-48hrs (N=267)</th>
<th>&gt;48hrs (N=255)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (s.d.)</td>
<td>62.4 (12.4)</td>
<td>62.6 (13)</td>
<td>65.1 (11.6)</td>
<td>0.02</td>
</tr>
<tr>
<td>Female (%)</td>
<td>37.3</td>
<td>39</td>
<td>50.6</td>
<td>0.003</td>
</tr>
<tr>
<td>Prior MI (%)</td>
<td>25.8</td>
<td>25.1</td>
<td>38.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Index PCI (%)</td>
<td>50.7</td>
<td>47.2</td>
<td>41.6</td>
<td>0.001</td>
</tr>
<tr>
<td>MI (%)</td>
<td>4.5</td>
<td>6.7</td>
<td>7.8</td>
<td>0.22</td>
</tr>
<tr>
<td>Death or MI (%)</td>
<td>10.2</td>
<td>11.2</td>
<td>16.5</td>
<td>0.067</td>
</tr>
<tr>
<td>Death (%)</td>
<td>7.4</td>
<td>5.6</td>
<td>12.3</td>
<td>0.03</td>
</tr>
</tbody>
</table>

Presentation Hour: Noon-1:00 p.m.

Elevated Titre of Antihuman Heat Shock Protein 60 Predicts an Adverse Medium-Term Prognosis in Patients With Unstable Angina

David H. Birnie, Lewis E. Vickery, Stewart Hills, John Norris, Stuart M. Cobbe, Glasgow University, Glasgow, United Kingdom

Background: There is evidence that the inflammatory state of coronary atherosclerosis is important in determining plaque stability. Increased CRP levels have prognostic implications and interest has turned to other aspects of inflammation including antibodies to heat shock proteins expressed within plaques. Their prognostic importance in coronary disease has not been addressed. Methods: Consecutive emergency admissions with acute chest pain of suspected cardiac origin, but without ST elevation or significant CK-MB elevation were eligible for inclusion. After discharge, they were followed up by review of their hospital case records and directly by telephone and letter. Time to clinical endpoints were CHD death, non-fatal MI, CABG, PTCA, angiogram and readmission with further cardiac ischemic chest pain. Anti-human heat shock protein 60 (anti-huhs60) and anti-mycobacterial heat shock protein 65 (anti-myhcp65) titres were examined on samples drawn on the morning after admission. Results: A total of 586 patients were enrolled from a single centre. During follow-up (mean of 304 days, range 1 to 796 days), 277 patients had an endpoint. Table 1 details hazard ratios for anti-huhs60 and anti-myhcp65 in quartiles for the combined endpoint conditional on age, smoking, log(CRP), hypertension and diabetes in a multivariate Cox model. Conclusions: Patients admitted with acute cardiac chest pain and elevated levels of anti-huhs60 had an adverse medium-term prognosis. Anti-myhcp65 titres were not predictive.