Anemia Worsens Prognosis After Primary Angioplasty

Background: The presence of anemia at the time of primary percutaneous coronary intervention (PCI) has been associated with worse clinical outcomes.

Methods: We performed a propensity score matched analysis of 2352 patients from the CADILLAC trial with complete data, who underwent PCI for ST-elevation myocardial infarction (STEMI) in 2001-2002 (n=1176) or 2003-2004 (n=1176). The presence of anemia was defined as (hematocrit <39% for men and <36% for women).

Results: Patients with anemia were older, more frequently female, and had a higher prevalence of diabetes and hypertension. At discharge patients with anemia were less likely to be treated with aspirin (91.2% vs. 96%, P=0.0012) and beta-blockers (67.7% vs. 79.2%, P<0.001). In both males and females anemia was associated with increased 30-day and 1-year mortality, and women with anemia had an increased rate of stroke (Table). One-year rates of reintervention (2.9% vs. 2.3%) and target vessel revascularization (10.8% vs. 13.7%) were similar in patients with vs. without anemia, respectively. By multivariate analysis lower hematocrit was an independent predictor of 30-day mortality (HR 0.80, P=0.04), one-year mortality (HR 0.83, P=0.02) and disabling stroke (HR 0.93, P=0.004). Conclusions: Anemia at baseline in patients with AMI treated with PCI is common, and is associated with an adverse early and late prognosis. Anemic patients are less likely to be treated with medications proven to improve outcomes after AMI, which adversely affects their survival.

 Endpoint: 30-day and 1-year outcomes

<table>
<thead>
<tr>
<th>Endpoints</th>
<th>Males</th>
<th>P-value</th>
<th>Females</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Without anemia</td>
<td>N=1176</td>
<td></td>
<td>N=833</td>
<td></td>
</tr>
<tr>
<td>With anemia</td>
<td>N=1306</td>
<td></td>
<td>N=461</td>
<td></td>
</tr>
<tr>
<td>Mortality (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>At 1 year</td>
<td>1.2</td>
<td>0.04</td>
<td>1.0</td>
<td>0.0002</td>
</tr>
<tr>
<td>At 1 year</td>
<td>2.7</td>
<td>0.03</td>
<td>17.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Disabling stroke (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>At 30 days</td>
<td>0.0</td>
<td>0.0</td>
<td>2.6</td>
<td>0.01</td>
</tr>
<tr>
<td>At 30 days</td>
<td>1.2</td>
<td>0.16</td>
<td>4.0</td>
<td>0.0005</td>
</tr>
</tbody>
</table>

High Risk Genotypes for Myocardial Infarction Are More Common in African Americans Than European Americans

David E. Lanfear, Sharan Marsh, Sharon Cresci, John A. Spetrus, Howard L. McLeod, Washington University School of Medicine, Saint Louis, MO, Mid-America Heart Institute, Kansas City, MO

Background: African Americans have higher rates of cardiovascular disease than their white counterparts. Whether this greater burden is due to genetic or environmental factors is not known. Recent large case-control studies have identified polymorphisms in common-27 (GJA4), plasminogen activator inhibitor-1 (PAI-1), and stromelysin-1 (MMP3) as risk factors for myocardial infarction (MI). We tested the frequency of these polymorphisms in black and white patients to provide new insights into the frequencies of high-risk genotypes in different racial groups.

Methods: Genomic DNA from population samples of 95 African Americans (AA) and 95 European Americans (EA) was used for genotyping. DNA containing the polymorphism of interest was amplified using the polymerase chain reaction, followed by genotyping using Pyrosequencing.

Results: All three MiR-genotypes were observed in both populations. All were in Hardy-Weinberg equilibrium. The frequencies of the ‘high risk’ genotypes were: GJA4 C1019T T/T: AA-20%, EA-7% (p=0.015); PAI-1 -668delG G/G: AA-56%, EA-16%

Inherent Sex Differences in the Ischemia-Induced Myocardial Inflammatory Response of Proestrus Females and Normal Males

Meiling Wang, Lauren Baker, Ben Tsai, Kirstan K. Meldrum, John W. Brown, Daniel R. Meldrum, Indiana University School of Medicine, Indianapolis, IN, Indiana Center for Vascular Biology and Medicine, Indianapolis, IN

Background: Although it is well established that there are gender differences in the progression of heart disease, little information exists regarding the mechanisms of inherent sex differences in myocardial responses to acute injury. Recent evidence indicates that the female inflammatory response may explain differential survival following sepsis and heart disease; yet, the molecular basis for the cardiac inflammatory response, especially the p38 MAPK activation and IL-1/IL-6 production, is an important component of myocardial injury. We hypothesized that proestrus females would demonstrate: a different myocardial functional recovery following ischemia; and, 2) inherent differences in p38 MAPK activation and IL-1/IL-6 production.

Methods: Langendorff perfused rat hearts (normal age matched males and proestrus females) were subjected to 27 min ischemia and 40 min reperfusion. Developed pressure (DP) was continuously recorded. Myocardium was assessed for p38 activation (phospho-p38, western), and IL-1/IL-6 mRNA production (RT-PCR). STATS: ANOVA with Bonferroni-Dunn (P<0.05=significance).

Results: The recovery of left ventricular developed pressure in female rat after I/R was much higher than that in male rat (female: 74.6%±0.12, n=5; male: 43.1%±0.04, n=5; p<0.05). IL-1 and IL-6 mRNA levels were significantly lower in female heart than that in male heart after I/R (IL-1α, female 0.41±0.018, male 0.6±0.013 p=0.005; IL-1β, female 0.49±0.022, male 0.69±0.026 p=0.001; IL-6, female 0.6±0.02, male 0.76±0.015 p<0.001) . More p38 MAPK activation existed in male rat heart.

Conclusions: 1) Sex differences exist in normal male and female myocardial responses to ischemia; 2) following ischemia, proestrus females demonstrated less p38 MAPK activation than males; 3) proestrus females produce less IL-1 and IL-6 than normal, aged-matched males subjected to the same insult.

High Risk Genotypes for Myocardial Infarction Are More Common in African Americans Than European Americans

David E. Lanfear, Sharan Marsh, Sharon Cresci, John A. Spetrus, Howard L. McLeod, Washington University School of Medicine, Saint Louis, MO, Mid-America Heart Institute, Kansas City, MO

Background: African Americans have higher rates of cardiovascular disease than their white counterparts. Whether this greater burden is due to genetic or environmental factors is not known. Recent large case-control studies have identified polymorphisms in common-27 (GJA4), plasminogen activator inhibitor-1 (PAI-1), and stromelysin-1 (MMP3) as risk factors for myocardial infarction (MI). We tested the frequency of these polymorphisms in black and white patients to provide new insights into the frequencies of high-risk genotypes in different racial groups.

Methods: Genomic DNA from population samples of 95 African Americans (AA) and 95 European Americans (EA) was used for genotyping. DNA containing the polymorphism of interest was amplified using the polymerase chain reaction, followed by genotyping using Pyrosequencing.

Results: All three MiR-genotypes were observed in both populations. All were in Hardy-Weinberg equilibrium. The frequencies of the ‘high risk’ genotypes were: GJA4 C1019T T/T: AA-20%, EA-7% (p=0.015); PAI-1 -668delG G/G: AA-56%, EA-16%
The Impact of Deep Sternal Wound Infection on Long-Term Survival Following Coronary Artery Bypass Grafting

Ioannis K. Toumpoulis, Constantine E. Anagnostopoulos, Joseph J. DeRose, Daniel G. Swistel, St. Luke's-Roosevelt Hospital Center at Columbia University, New York, NY, University Hospital of Ioannina, Ioannina, Greece

Background: To identify the impact of deep sternal wound infection (DSWI) on long-term survival after coronary artery bypass grafting (CABG). Methods: We studied 3760 consecutive patients who underwent isolated CABG between 1992 and 2002. Patients with CABG and no DSWI were compared with those who developed DSWI. Long-term survival data were obtained from the National Death Index. Groups were compared by Cox proportional hazards models. Kaplan-Meier survival plots. The propensity for DSWI was determined by logistic regression analysis and each patient with DSWI was then matched to 10 patients without DSWI.

Results: Forty patients (1.1%) developed DSWI. Multivariate logistic regression analysis found that the independent predictors of DSWI were diabetes mellitus (odds ratio (OR) 5.5; P=0.001), hemodynamic instability (OR 4.0; P=0.026), use of bilateral internal thoracic arteries (OR 2.6; P=0.010), endocarditis and/or sepsis (OR 29.9; P=0.001) and dialysis (OR 3.4; P=0.049). There were no differences in thirty-day mortality for matched groups. Patients with DSWI had longer length of stay (35.0 versus 16.4 days; P=0.001). Kaplan-Meier curves of the two matched groups are shown in figure. After adjustment for pre, intra and postoperative factors, the adjusted hazard ratio of long-term mortality for patients with DSWI was 2.44 (95% CI 1.51-3.92; P=0.001).

Conclusions: We found that DSWI after CABG operations was associated with increased long-term mortality.

Impact of Diabetes on 12-Month Outcomes Following Coronary Artery Bypass Grafting (ROSETTA-CABG) Registry

Melissa Gilman, Karen Oksanec, Hiep Nguyen, Robert Duerr, Michael Del Core, Dominique Fourchy, Thao Huyhn, Ellis Lader, Felix J. Rogers, M. Rashid Chaudry, Louise Pilot, Mark J. Eisenberg, The ROSETTA-CABG Investigators, Jewish General Hospital/ McGill University, Montreal, PQ, Canada.

Background: Little is known about the impact of diabetes mellitus on outcomes during the first 12 months following CABG.

Objectives: The purpose of our study was to examine the relationship between diabetes mellitus and clinical outcomes during the first 12 months following CABG. Methods: The ROSETTA-CABG Registry is a prospective multicenter study examining the use of functional testing after CABG. We examined 363 diabetic and non-diabetic patients enrolled in the registry at 16 clinical centers in 6 countries. Diabetes status was defined by medication use at discharge. Only patients undergoing a first successful isolated CABG in which all ischemic areas were thought to be revascularized were included. Results: Among the 363 patients, 34 (8.9%) were receiving insulin at discharge, 62 (16.2%) were receiving oral hypoglycemic agents, and 267 (69.5%) were not receiving insulin or oral hypoglycemic agents. The patients were predominantly elderly men (mean age 63.2 ± 10.2, 81.0% male). The mean number of arteries bypassed was 3.6 ± 1.3. Insulin-treated patients had a higher incidence of composite clinical events (unstable angina, myocardial infarction, death) compared with patients receiving oral hypoglycemic agents and non-diabetic patients (17.6% versus 3.2% and 6.1%, respectively, insulin vs non-insulin p=0.02). There was also a trend for insulin-treated patients to have a higher incidence of composite procedural events (cardiac catheterization, PCI, repeat CABG) compared with patients receiving oral hypoglycemic agents and non-diabetic patients (18.8% versus 4.9% and 3.8% respectively, insulin vs non-insulin p=0.07). After controlling for baseline clinical characteristics, insulin use at discharge had a persistent association with composite clinical events (OR=3.19, 95% CI 1.07-9.56, p=0.04).

Conclusions: During the 12-month period after a successful CABG, insulin-treated patients have a higher rate of adverse events compared with patients receiving oral hypoglycemic agents and non-diabetic patients. These results suggest that diabetic patients may benefit from more aggressive surveillance following CABG.