year mortality after primary angioplasty for STEMI. Therefore, the use of statins after STEMI is highly recommended.

9:15 a.m.

**833-4**

Undertreatment of Low Serum High-Density Lipoprotein Cholesterol in Patients With Acute Myocardial Infarction

Anil Maroo, W. H. Wilson Tang, Byron Hoogwerf, Stephen G. Ellis, Cleveland Clinic Foundation, Cleveland, OH

Background: The prevalence of low serum high-density lipoprotein cholesterol (HDL-C) in patients with acute myocardial infarction (AMI) is understudied. We examined contemporary patients hospitalized for AMI in order to determine the prevalence, clinical characteristics, and treatment patterns of those with low HDL-C (<40 mg/dL). We compared clinical and laboratory data between patients with low HDL-C and HDL-C > 40 mg/dL by univariate analysis.

Results: In our cohort of 265 patients (mean age 61±14 years, 65% male, 20% diabetic, mean left ventricular ejection fraction 44±12%), 147 patients (55%) had low HDL-C. Compared to those with normal HDL-C, those with low HDL-C were younger and more likely to be male, had lower systolic blood pressure, were more likely to be actively smoking, and had higher serum triglycerides (177 vs 110 mg/dL, p<0.01). There were no statistically significant differences in the number of diabetic patients or in mean LDL-C levels (105 vs 106 mg/dL) in either group. Overall, 49% of the entire cohort, and 29% of those with low HDL-C, had LDL-C levels <100 mg/dL. Only 7 patients (6%) in the low HDL-C group were discharged on niacin or fibrate preparations, even though 86 patients (59%) had no contraindications to pharmacotherapy. In contrast, 90% of the entire cohort (85% of the low HDL-C group) were discharged on statins.

Conclusions: Low HDL-C is highly prevalent (55%) in contemporary patients with AMI, many of whom have LDL-C levels below recommended targets. Although aggressive LDL-C lowering attenuates some of the adverse effects of low HDL-C, secondary prevention models that focus on targeting LDL-C alone do not adequately address current patterns of dyslipidemia. The opportunity for inclusion of HDL-C raising therapies prior to discharge in post-MI patients is clear.

9:30 a.m.

**833-5**

Markers of Inflammation Predict Mortality and Are Reduced by Pexelizumab in Patients With Acute Myocardial Infarction: Insights From the Complement Inhibition in Myocardial Infarction Treated With Angioplasty (COMMA) Trial

Pierre Theroux, Paul W. Armstrong, Kenneth W. Mahaffey, Judith S. Hochman, Scott Rollins, Kevin J. Malloy, Thomas Parish, Jose C. Nicolau, Joel Lavoie, Christopher B. Granger, Montreal Heart Institute, Montreal, PQ, Canada

Background: In the COMMA trial, inhibition of the complement protein C5 with pexelizumab (Pex) paradoxically reduced mortality without influencing infarct size (Circulation 2003; 108). We examined plasma markers to investigate the role of inflammation and the impact of treatment.

Methods: 350 patients in the Pex bolus and infusion and placebo groups were studied at baseline before Pex and PCI, at the end of infusion (24 hours), and 48 hours later. Levels of hsCRP were assessed by nephelometry, and IL-6 and TNFα by ELISA.

Results: All markers increased from baseline through 72 h (p<0.001). IL-6 levels peaked at 24 h, hsCRP and TNFα at 72 h. Older age and elevated CK-MB (AUC) were associated with higher IL-6 and hsCRP. hsCRP was lower in the Pex group at 24 h (p=0.02). Levels of IL-6 and hsCRP were significantly reduced in Pex patients > 65 years at 24 h and 72 h; hsCRP in those with CK-MB > median was similarly reduced. Mortality at 90 days was strongly related to baseline levels of IL-6 (OR per increasing quartile: 3.2, 95% CI: 1.7-5.9), hsCRP (1.6, 1.1-2.5), and TNFα (1.4, 0.9-2.1).

Conclusion: Inflammation was associated with higher mortality after primary PCI. Inhibition of complement by Pex reduced inflammation independently of the primary PCI. These findings support the hypothesis that inflammation has a role in the development of post-MI complications and is a target for therapy in patients with AMI.

9:45 a.m.

**833-6**

An Updated Meta-Analysis of the Effect of Glucose-Insulin-Potassium on Survival in Acute Myocardial Infarction: Impact of Reperfusion Therapy

Ramesh M. Gowda, David L. Brown, Beth Israel Medical Center, New York, NY

Background: Glucose-insulin-potassium (GIK) has been proposed as adjuvant therapy in acute myocardial infarction (AMI) because of its ability to induce multiple favorable metabolic alterations in the cardiac myocyte. However, randomized controlled trials performed over the last 4 decades have yielded conflicting results regarding the impact of GIK on mortality in AMI. We performed an updated meta-analysis to investigate the effect of GIK on mortality in AMI.

Methods: We conducted a comprehensive MEDLINE search that revealed 3 randomized controlled trials of GIK treatment in AMI. The odds ratios of the individual trials were pooled using a random effects model. Separate meta-analyses were performed on the 9 studies in which no reperfusion therapy was provided and the 4 studies in which GIK was administered in addition to reperfusion therapy.

Results: Of the 4149 patients randomized, 2145 received GIK while 2004 were in the control groups. Mortality was reduced from 13% (257/2004) in the control group to 11% (234/2145) in the GIK-treated patients (Odds Ratio (OR), 0.78; 95% confidence interval (CI), 0.58 to 1.05; P=0.106). In the trials performed without reperfusion therapy, the mortality was reduced from 20.5% (192/934) in the control group to 16.6% (149/897) in the GIK-treated group (OR, 0.77; 95% CI, 0.60 to 0.98; P=0.034). In the trials with reperfusion therapy, there was a slight increase in mortality from 6.0% (85/1707) in the control group to 6.8% (85/1248) in the GIK-treated patients (OR, 0.97; 95% CI, 0.46 to 2.0; P=0.845).

Conclusion: These results appear to be a trend toward a reduction in mortality by GIK in AMI. However, the benefit of GIK is restricted to patients who do not receive reperfusion therapy. Patients who receive reperfusion therapy do not benefit from GIK. GIK should be considered for AMI patients who are not eligible for reperfusion therapy.