INFLAMMATORY AND LIPID MARKERS PRE- AND POST-LDL APHERESIS IN PATIENTS WITH FAMILIAL HYPERCHOLESTEROLEMIA (THE INFLAME STUDY)

ACC Moderated Poster Contributions
McCormick Place South, Hall A
Monday, March 26, 2012, 9:30 a.m.-10:30 a.m.

Session Title: Risk Stratification and Repair
Abstract Category: 1. Chronic CAD/Stable Ischemic Heart Disease: Basic
Presentation Number: 1194-31

Authors: Vimal Ramjee, Monica Epperson, Ngoc-Anh Le, Louette Vaughn, Jefferson Baer, Christine Nell-Dybdahl, Peter Wilson, Laurence Sperling, Emory University, Atlanta, GA, USA

Background: LDL apheresis should be considered in patients with familial hypercholesterolemia (FH) who have not achieved lipid goals on maximum pharmacotherapy. The acute dramatic reduction in LDL after apheresis provides a unique perspective on the dynamics of inflammatory and lipid substrates.

Methods: The INFLAME Study is a pilot study focused on the rebound kinetics of lipid, oxidative and inflammatory markers over a 48 hour period following an LDL apheresis procedure. FH patients on maximum pharmacotherapy undergoing LDL apheresis were identified and non-consecutively enrolled. C-reactive protein (CRP), LDL cholesterol (LDL), lipoprotein-associated phospholipase A2 mass (PLAC), lipoprotein a (Lp(a)), and apolipoprotein B (apoB) were measured pre- and post-apheresis at multiple time points during an inpatient diet-controlled stay.

Results: 8 FH patients (7 heterozygous, 1 homozygous; 6 female, 2 male) with a mean age of 48 ± 17 years, and body mass index of 28.4 ± 5.2 kg/m2 were studied. Mean % decrease post-apheresis was 72.1% for LDL, 71.8% for apoB, 60.4% for Lp(a), 43.8% for PLAC, and 61.4% for CRP. Compared to pre-apheresis levels, mean percent recovery at 12, 24 and 48 hours post-apheresis for: LDL was 31.1%, 37.5%, and 49.7%; apoB was 33.0%, 40.7%, and 52.7%; Lp(a) was 43.4%, 51.7%, and 70.4%; PLAC was 57.4%, 73.0%, and 78.4%; and CRP was 90.5%, 134%, and 120%, respectively. LDL had the highest correlation to apoB in rate change acutely (r = 0.88, 24 hours), and at 48 hours (r = 0.94). CRP had the highest correlation to PLAC in rate change acutely (r = 0.82, 24 hours), over 48 hours (r = 0.83), and independent of meals (r = 0.90, 24 vs. 48 hours). Recovery rates for CRP and PLAC did not correlate with those of LDL or apoB.

Conclusions: Following the dramatic acute effect of apheresis, our data indicates a rapid rebound in CRP and PLAC that precedes lipid recovery. In contrast to the linear recovery of LDL, both CRP and PLAC exhibit biphasic rebound with CRP returning to pre-procedure levels at the fastest rate. These data are consistent with distinct regulatory processes for CRP and PLAC as inflammatory markers and the presence of a high inflammatory state in these patients, independent of LDL levels.