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DOES AGGRESSIVE STATIN THERAPY REDUCE CORONARY ATHEROSCLEROTIC PLAQUE LIPID CONTENT? RESULTS FROM: REDUCTION IN YELLOW PLAQUE BY AGGRESSIVE LIPID LOWERING THERAPY (YELLOW) TRIAL

i2 Oral Contributions McCormick Place South, S106b Sunday, March 25, 2012, 8:00 a.m.-8:10 a.m.

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Authors: <u>Annapoorna Subhash Kini</u>, Pedro Moreno, Jason Kovacic, Atul Limaye, Ziad Ali, Joseph Sweeny, Usman Baber, George Dangas, Samin Sharma, Mount Sinai Medical Center, New York City, NY, USA

Background: Although statin therapy reduces coronary atherosclerotic burden, the extent to which statins modulate lipid content and coronary flow physiology is not known. We evaluated the short-term impact of aggressive statin therapy on changes in coronary plaque composition, lipid content, and coronary flow physiology using a novel multimodality imaging approach.

Methods: We randomized 80 consecutive patients with multivessel coronary artery disease (CAD) undergoing elective percutaneous coronary intervention (PCI) to aggressive (rosuvastatin 40 mg daily) vs. conventional statin therapy. Following PCI of the culprit lesion, non-culprit lesions with a fractional flow reserve (FFR) < 0.8 were interrogated using virtual-histology intravascular ultrasound (VH-IVUS) and near-infrared (NIR) spectroscopy. Coronary flow physiology and changes in plaque composition were assessed after 6-12 weeks during follow-up angiography. The primary outcome is change in lipid content using NIR spectroscopy and expressed as lipid core burden index (LCBI). Secondary outcomes included changes in coronary flow physiology using FFR and the correlation between LCBI, serum lipids and IVUS parameters.

Results: The mean age of the study population (n=80, 25% female) was 64.2 ± 9.4 years. Most patients had a history of hypertension (94%) and 20% were smokers. After a mean follow-up of 53.3 ± 10.8 days, total cholesterol ($144.9 \pm 29.7 \text{ mg/dl vs.} 134.9 \pm 33.6 \text{ mg/dl, p=0.02}$) and LDL-C ($81.9 \pm 25.7 \text{ mg/dl vs.} 69.8 \pm 25.4 \text{ mg/dl, p<0.001}$) were significantly reduced compared to baseline values. To date, 64 of 80 patients have returned for follow-up and staged PCI was deferred in 4 (5%) as repeat FFR was >0.8. All angiograms and intravascular imaging are being evaluated by an independent core laboratory blinded to study assignment. Follow-up will be complete by January 1, 2012.

Conclusions: The present study is the first to provide insight on the short-term impact of aggressive statin therapy on coronary plaque composition, lipid content and coronary flow physiology. We will also demonstrate the complementary role of NIR spectroscopy as an adjunctive imaging modality to VH-IVUS and FFR in coronary plaque assessment.