676-6 Lipid Rich Plaques with Thrombus are Common in Unstable Rest Angina: Observations from Atherectomy Tissue Analysis

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Several autopsy studies indicate that disrupted atherosclerotic plaques particularly those rich in lipid with associated thrombus are the cause of acute MI. Other plaques without disruption and with little or no lipid. Although unstable rest angina (URA) shares a common pathogenesis to acute MI, there are little pathological data on plaque composition in UA. Directional Coronary Atherectomy (DCA) provides the opportunity to study plaque composition in UA. Methods: We prospectively analyzed the DCA tissue specimens of 60 pts with de novo culprit lesions for fibrocellular and lipid constituents by hematoxylin & eosin and oil red O stains. A fibrous plaque (FP) was defined as one which had predominantly fibrous and sclerotic tissue with no or minimal lipid (0 or 1+ on scale of 3). A lipid plaque (LP) was defined as one with moderate or high lipid (2 or 3+) content. The presence of inflammatory cells and thrombus were also noted. Histopathology was analyzed independent of clinical presentation.

Coronary thrombus on histologic analysis was present in 23/36 (64%) of LP vs. in 5/24 (21%) of FP (p < 0.01). Coronary thrombus was present in 88% and 82% of LP in rest angina and post MI respectively. Inflammatory cells were noted in 12 LP and 2 FP (p = 0.02) — in 38% of both rest angina and post MI specimens. Conclusions: Lipid rich plaques are very common in rest angina and post MI, moderately common in crescendo or new onset and rare in stable angina. LP are usually associated with thrombus and inflammation particularly in rest angina and post MI pts. These DCA tissue analyses confirm and expanded on prior autopsy studies in acute syndromes and support the pathogenetic link between unstable rest angina and acute MI.

676-2 Progression and Regression of Coronary Atherosclerosis Occur within the Same Patient During Placebo Treatment and During Lipid-Lowering Therapy with Pravastatin

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REGRESS (Regression Growth Evaluation Statin Study) is a placebo controlled multicenter study to assess the effect of 2-y treatment with Pravastatin (PRAV) on progression and regression of angiographically documented coronary atherosclerosis (CA). CA in patients with a serum cholesterol between 4-8 mmol/l (155-310 mg/dl). Analyses of the coronary arteriograms were performed by quantitative computer analysis. The primary endpoints of the study: change in Mean Segment Diameter and Minimum Obstruction Diameter (MOD) averaged per patient, showed significant retardation of mean progression of CA in the PRAV-group as compared to the placebo (PLAC)-group. However, these mean changes per treatment group are hardly informative about individual CA-behavior. Therefore we determined for all 641 patients included in the primary MOD-analysis: 1. a mean progression score (MPS)-cumulative value of all >0.4 mm progressing obstructions divided by number of contributing obstructions and 2. a mean regression score (MRS)-cumulative value of all <0.4 mm regressing obstructions divided by the number of contributing obstructions. Obstructions changing >0.4 mm were considered stable and do not contribute to the scores. Thus, each patient was characterized by a MPS and a MRS. An overview of the patient MPS and MRS is presented in the figure below.

Conclusion: significant progression and regression of CA within the same patient occurred in 41 (13%) of the patients and in 27 (9%) PLAC-patients. Thus, although pravastatin slows major progression of CA, progression and regression of CA within the same patient still occurs in a considerable number of patients during lipid lowering therapy.