Abnormal T-axis was defined as -180 to -15 and 105 to 180 degrees. CRP levels were trocardiographic abnormalities in 8501 subjects (aged 28 to 75, 49.8% male) from the Methods:

Background: The Stuveling, Jan A. Kors, Wiek H. van Gilst, Folkert W. Asselberos, Ad J. van Boven, Gilles F. Diercks, Hans H. Hillege, Erik M.

Conclusions: These results suggest that in a large general population, elevated CRP is associated with ECG abnormalities, suggestive for cardiac ischaemia. Therefore, CRP may be useful in early risk profiling to improve cardiovascular risk assessment and treatment.

9:30 a.m.

8:45 a.m.

8:42-2

Accumulation of a Novel Macrophage-Targeting Photodynamic Compound Within Lipid-Rich Atherosclerotic Plaques

Ahmed Tawekol, Alan Fischman, Tayyaba Hasan, Touqir Zahra, James Muller, Michael Hamblin, Massachusetts General Hospital, Boston, Massachusetts.

Vulnerable atherosclerotic plaques contain an abundance of inflammatory cells. We recently developed a photodynamic therapeutic (PDT) agent, (chlorin e6 conjugated with malated ylamin), that is recognized by scavenger receptors and can target macroph-

ages with high selectivity. We therefore sought to test the hypothesis that the novel mac-

rophage-targeting photodynamic compound accumulates in experimental atherosclerotic lesions in quantities that are sufficient for photodynamic therapy. Four New Zealand rabbits were studied, in which atherosclerotic lesions were induced in 2 by concomitantly instillation of the methylhydroxyloma aceta followed by a 5% peanut oil-2% cholesterol diet for 10 weeks. Thereafter, chlorin e6 conjugated with malated ylamin was administered intravenously to the rabbits. At 24 hours after injection of the PDT compound, the animals were sacrificed, and the aortas examined for uptake of the fluo-

rescent PDT agent.

PDT uptake was significantly higher within the atherosclerotic aortas (5.2 +/- 3.2 vs. 1.0 +/- 1.2, cell fluorescence units/gm tissue x 10^6, atherosclerotic aorta vs control aorta, p<0.03, n=6 segments).

These data indicate that this macrophage-targeted photodynamic compound accumulates within atherosclerotic plaques, and support future efforts to employ PDT compounds to ablative inflammatory foci within atherosclerotic plaques.

9:00 a.m.

Statin Administration In Patients With Acute Coronary Syndromes: Results in Decreased Heat Release of Culprit Atherosclerotic Lesions

Christophori Stainwag, Konstantinos Toutouzas, Eleotzoulias Tsimarl, Ioannis Kallikazaros, Manolis Vavouranakis, Sofia Vaina, Athanasios Nikitas, Christina Chrysochoo, Dimos Panagiotakos, Pavlos Toutouzas, Hipokrepasion Hospital, Athens, Greece.

Background: It is been shown, that there is thermal heterogeneity within human ath-

erosclerotic plaques. Recent studies have suggested, that stiatics may induce mechan-

ical plaque stability by reduction of inflammatory cells within the plaque. This study was designed to examine the effect of statins on atherosclerotic plaque stibalization by meas-

uring the temperature variation of atherosclerotic plaques. Methods: We studied 60 patients (25 patients with unstable angina (UA) and 25 patients with acute myocardial infarction (AMI)). All pts underwent diagnostic catheterization. Thirty-two pts (18 pts with UA and 14 pts with AMI) were under statin treatment for over a month and 28 pts were not receiving statins (18 pts with UA and 10 pts with AMI). Fifty-five pts were under aspirin treatment. Total cholesterol and low-density cholesterol were measured in all pts. A thermography catheter previously validated (Medispes S.W.A.Gzug-Switzerland) was used during the diagnostic catheterization, in order to measure the temperature difference (TD) between the atherosclerotic plaque and the healthy vessel wall.

Results: TD was progressively increased its pts with UA compared to pts with AMI. (0.41 +/- 0.28 vs 0.63 +/- 0.41°C, p< 0.02). When we categorized the study population into pts receiving statins and pts not treated with statins, TD was lower in the treated group (0.41 +/- 0.4 vs 0.65 +/- 0.3°C, p<0.01). Moreover, untreated pts with UA or AMI had greater TD compared to treated pts (UA: 0.45 +/- 0.26 vs 0.29 +/- 0.25°C, p=0.09; AMI: 0.82 +/- 0.58 vs 0.53 +/- 0.34°C, p<0.01). Multivariate analysis showed that treatment with statins was an independent factor in the assessment of temperature variation, adjusted for age, hypercholesterolemia, hypertension, smoking, aspirin intake, and clinical syndrome.

Conclusion: Pts with acute coronary syndromes, under statin treatment have decreased heat production from the culprit atherosclerotic plaques. Recent studies have suggested, that statins may induce mecha-

nisms in atherosclerotic plaques, and support future efforts to employ PDT compounds to ablative inflammatory foci within atherosclerotic plaques.

8:43-2

Statin Administration In Patients With Acute Coronary Syndromes: Results in Decreased Heat Release of Culprit Atherosclerotic Lesions

Christophori Stainwag, Konstantinos Toutouzas, Eleotzoulias Tsimarl, Ioannis Kallikazaros, Manolis Vavouranakis, Sofia Vaina, Athanasios Nikitas, Christina Chrysochoo, Dimos Panagiotakos, Pavlos Toutouzas, Hipokrepasion Hospital, Athens, Greece.

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Conclusion: Pts with acute coronary syndromes, under statin treatment have decreased heat production from the culprit atherosclerotic plaques. Recent studies have suggested, that statins may induce mecha-

nisms in atherosclerotic plaques, and support future efforts to employ PDT compounds to ablative inflammatory foci within atherosclerotic plaques.

8:42-4

Association Between Ischemic Electrocardiographic Abnormalities and C-Reactive Protein in a General Population


Background: The inflammatory marker C-Reactive protein (CRP) and ischemic electrocardiographic (ECG) abnormalities reflect both vascular instability and are easy to stratify for the presence of an increased level of urinary albumin excretion. High sensi-

tive CRP was measured by nephelometry (BN II, Boehringer, Mannurg, Germany).

Abnormal T-axis was defined as -180 to -15 and 105 to 180 degrees. CRP values were divided below and above the upper quartile (CRP>2.60 mg/dl).

8:15 a.m.

8:42-6

PPAR-gAgonist Induces Regression of Atherosclerotic Plaques: In Vivo Study by High Resolution Magnetic Resonance Imaging


Introduction: Nuclear receptor PPAR-g regulates adipogeton and lipid metabolism. It is expressed in macrophages and may have antiatherogenic effects. We reported the ability of MRL to monitor in vivo changes in atherosclerotic (AT) lesions. Our objective was to compare the effects of simvastatin, a PPAR-g agonist-2 (2-[4-(phenyl)oxazolyl]oxazolidine-5-acetic acid) and their combination on pre-established lesions in a rabbit model using MRI and histology.

Methods: Aortic AT lesions were induced in rabbits by double balloon-injury and atherogenic diet. Following lesion induction, animals were MRI imaged and, based on severity of the established AT lesions, assigned to the different groups: Progesterone (no therapy), n=6; simvastatin (5mg/kg/day, n=6), PPAR-g agonist-6 (5mg/Kg/day, n=6) and their combi-

nation (n=7). The atherogenic diet was maintained during the study. After 6 months all animals were sacrificed and processed for histology. The effect of the different treatments on AT lesions was assessed by measuring vessel wall area (VWA), a surrogate of AT burden. VWA measurements by MRI were normalized to the randomiza-

tion value (each rabbit served as its own control).

Results: Good agreement between MRI and histopatology measurements of VWA was found (p=0.001, R=0.88). The mean VWA at the time of randomization was 8.4±0.6 mm².

Conclusions: These data suggest that MRL may be less effective at inhibiting athero-

sclerotic progression when pretreatment atherosclerosis is more advanced.

9:45 a.m.