Neovessel Unit (Total Count) | Diabetic Ruptured Plaques (n=41) | Non-Diabetic Ruptured Plaques (n=34) | P Value
--- | --- | --- | ---
Neovessels in the outer media | 173 ± 61 | 104 ± 42 | 0.0001
Neovessels in the inner media | 124 ± 52 | 48 ± 24 | 0.0001
Neovessels in the intima | 27 ± 20 | 13 ± 10 | 0.0001
Inflammation score (0-2) | 1.8 ± 0.5 | 1.5 ± 0.5 | 0.0001

Diabetic

Non-Diabetic

831-6 Long-Term Secondary Prevention With Folic Acid: No Effects on Clinical Outcomes (the GOES Extension Study)

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Purpose: Folic acid has favourable effects on vascular endothelium and lowers plasma homocysteine levels. In addition, homocysteine appears to be an independent risk factor for atherosclerotic disease. However, the value of folic acid in secondary prevention had seldom been tested. Two yr folic acid treatment in the randomized GOES study showed no reduction in clinical endpoints despite a 18% homocysteine reduction in patients on folic acid. Suggested was that the follow up could have been too short, therefore the study was extended with another 18 months Tx. Here we report results of the extended GOES trial, an open-label trial with folic acid 0.5 mg per day in a patient population with stable coronary artery disease (CAD).

Methods: 593 Patients were included in this study; 300 were randomized to folic acid and 293 served as controls. Mean follow-up time was 42 months. At baseline all patients had been on statin therapy for a mean of 3.2 years.

Results: In patients treated with folic acid plasma homocysteine levels decreased with 18% from 12.0 ± 4.8 to 9.4 ± 3.5 micromol/L, while these levels remained unaffected in the control group (p < 0.001 between groups). The primary endpoint (all-cause mortality and a composite of vascular events) was encountered in 75 (25.6%) patients in the folic acid group and in 75 (25.6%) patients in the control group (RR 0.98; p=NS). Also in the quartile of patients with the highest baseline homocysteine levels (>13.7 micromol/L) no salutary effects of folic acid Tx could be demonstrated. In a multivariable survival model with adjustments for clinical factors the most predictive laboratory parameters were, in order of significance, levels of creatinine clearance, and homocysteine concentration.

Conclusions: Within 3.5 years folic acid does not seem to reduce clinical endpoints in patients with stable CAD while on statin treatment. Homocysteine might therefore merely play an important role in preventing atherosclerosis. EPC counts have been found to be inversely related to traditional coronary artery disease (CAD) risk factors, yet their association to CAD severity remains unknown.

831-1 Ruptured Diabetic Atherosclerotic Plaques Have More Inflammation and Neovascularization Than Ruptured Plaques From Patients Without Diabetes

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Background: Plaque rupture may be asymptomatic or precipitate acute thrombotic events, and patients with diabetes mellitus (DM) are at higher risk for acute events than patients without DM. To evaluate if this difference is related to plaque composition, we quantified inflammation and neovascularization in ruptured aortic plaques from patients with/without DM.

Methods: Neovessels and macrophages/T cells were identified by CD34 (blue) and CD68/CD3 (red) biologic immunohistochemistry (Figure) in 41 DM ruptured and compared to 34 non-DM ruptured plaques.

Results: See table.

Conclusion: Ruptured plaques from DM have increased inflammation and neovascularization supporting plaque composition as a contributor for the increased incidence of atherothrombotic complications among DM population.

831-3 Coronary Lumen Change Is Determined Primarily by Adventitial Remodeling Rather Than Plaque Change During Therapy

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Background: Remodeling of coronary arteries has been demonstrated during plaque regression, but the contribution to lumen change is unclear.

Methods: Intracoronary ultrasound automated pull-backs were recorded on S-VHS tape both before and after twelve months of lipid-lowering therapy in a target coronary artery in 18 subjects with known coronary artery disease. The pullbacks were digitized and calibrated, and identical segments with plaque in the target artery were identified on the pre-and post-therapy images. The lumen and adventitia-media borders were manually traced on all images in the segment that allowed image interpretation, with the longitudinal position noted. The plaque and adventitial volumes were calculated by a numerical integration of the area of the plaque over the longitudinal length. The mean plaque adventitial, and lumen areas in sq. mm were calculated based on plaque and adventitial volume and segment length.

Results: The change in mean lumen area was poorly correlated with the change in mean plaque area (R = 0.256, p = 0.306). Lumen area change was strongly correlated with adventitial area change (R = 0.903, p < 0.001, see figure below) The relationship was Change in mean lumen area = 0.712 x Change in mean adventitial area + 0.347 mm².

Conclusions: The changes in lumen area after one year of lipid-lowering therapy are better correlated with adventitial change than with plaque change, indicating that adventitial remodeling is an important determinant of lumen change.

831-7 Circulating Endothelial Progenitor Cells Predict Coronary Artery Disease Severity

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Background: Circulating endothelial progenitor cell (EPC) counts are hypothesized to play an important role in preventing atherosclerosis. EPC counts have been found to be inversely related to traditional coronary artery disease (CAD) risk factors, yet their association to CAD severity remains unknown.

Methods: We measured EPC counts by quantitative cell culture in 122 patients undergoing diagnostic cardiac catheterization. The association between patients’ EPC count and the presence of multi-vessel CAD and of traditional cardiac risk factors was assessed using logistic regression analysis.

Results: The median age of the study population was 59.3 years, 40% were male, 78% were white, 39% had diabetes mellitus, 24% had hypertension, and 19% had hypercholesterolemia. The median age of the study population was not different between the male and female groups. The median number of EPCs was 150 per ml (range 80-250) in the male group and 160 per ml (range 90-250) in the female group (p = 0.31). Patients with diabetes mellitus had significantly lower EPC counts than patients without diabetes mellitus (140 per ml vs. 160 per ml, p = 0.02). Patients with hypertension had significantly lower EPC counts than patients without hypertension (150 per ml vs. 160 per ml, p = 0.03). Patients with hypercholesterolemia had significantly lower EPC counts than patients without hypercholesterolemia (140 per ml vs. 160 per ml, p = 0.03). In a logistic regression model, EPC count was inversely related to traditional coronary artery disease (CAD) risk factors, yet their association to CAD severity remains unknown.

831-2 Atherosclerotic Plaque, Inflammation, and Oxidative Stress: Clinical Studies

Tuesday, March 09, 2004, 8:30 a.m.-10:00 a.m. Morial Convention Center, Room 265