the presence of HTN (n=217, 52%) if the arterial pressure was >140/90 mm Hg and/or they were being treated for HTN, and tested serum IgG antibodies to cytomegalovirus, hepatitis A virus, C*hlamydia pneumoniae, Helicobacter pylori,* herpes simplex virus type 1 and type 2, as well as C-reactive protein (CRP) levels. Results: The prevalence of antibodies to hepatitis A virus or *Helicobacter pylori,* but not to others, was higher in the HTN than the non-HTN patients. These associations were significant even after adjustment for traditional risk factors. Adjusted OR with 95%CL was 1.59 (1.05-2.40) for hepatitis A virus and 1.75 (1.15-2.68) for *Helicobacter pylori* infection. In addition, increasing number of seropositivities (pathogen burden) was significantly associated with increasing HTN risk. The prevalence of HTN was 58% in the high pathogen burden group (>4 positive antibodies) compared with 45% in the low burden group (P<0.01). The pathogen burden remained a significant predictor of HTN after multivariate analysis (adjusted OR 1.60 with 95% CL 1.05-2.42). Interestingly, we found that elevated CRP levels (>0.5mg/dL) were also associated with HTN (adjusted OR 1.80 with 95% CL 1.12-2.87), and that elevated CRP levels combined with individual infections and pathogen burden increased the risk of HTN: adjusted OR with 95% CL was 3.09 (1.56-6.12) for hepatitis A virus, 3.68 (1.83-7.40) for *Helicobacter pylori*, and 3.15 (1.56-6.37) for pathogen burden. Conclusion: Our data suggest that infection plays a role in the development of HTN, and elevated CRP levels can increase the risk posed by infection in HTN.

Noon

1059-11 Venous Endothelium Dysfunction Is Also Presented in Hypertensive Patients

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Background: Endothelium dysfunction has been demonstrated in the arteries of hypertensive patients. Although veins and arteries produce nitric oxide, it is unknown if the formers are also impaired in the arterial hypertension. The aims of this study were: 1) To evaluate whether or not the hypertensive patients exhibit venous endothelium dysfunction; 2) To determine the relationship between endothelium dependent and independent vasodilation responses in venous and arterial systems in these subjects.
Methods: Sixteen patients with mild hypertension (SBP 145.8 +/- 8.8 mm Hg and DBP 98.3 +/- 4.8 mm Hg), out of medication and without other risk factor, and fifteen matched normotensive volunteers had the venous and arterial endothelial function evaluated, respectively, by the dorsal hand vein (DHV) and high resolution ultrasound (flow mediated dilation - FMD) techniques.
Results: The maximal dilation response (Emax), produced by acetylcholine (ACH) was consistently reproducible, allowing evaluating the venous endothelium dependent response. Hypertensive group had a marked reduction of Emax to ACh (54,9+/-10,8%) when compared to normotensive controls (Emax= was 85,2+/-27,0%). The flow mediated dilation responses were reduced in the hypertensive subjects compared to their controls (6.6+/1.3 versus 12.4+/-1.4 %, respectively). The responses to nitric oxide donors were similar in both groups tested by the DHV and FMD methods. By the analysis of the measurement method comparison data (Bland & Altman), the responses both techniques agreed in normotensive and hypertensive subjects.
Conclusion: Hypertensive patients had an attenuated endothelial-dependent response, indicating that the endothelium dysfunction observed in arteries is also present in the venous system.

Noon

1059-12 Sustained Upregulation of Inflammatory Cytokine and Its Receptor Genes Associated With Proteinase Activation in Abdominal Aortic Aneurysm: Results From Combined Study With cDNA Array and Real-Time Reverse Transcriptase Polymerase Chain Reaction Methods

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Background: In the development of aortic aneurym, inflammatory process plays a pivotal role in the vessel tissue degradation. Under these conditions, cytokines are known to serve as an essential mediator of inflammation, contributing to degenerating local tissue associated with or without aneurysm formation probably through the activation of proteinases. However, few data exist regarding which cytokines are important for proteinase activation associated aneurysm formation. Therefore, we analyzed gene expression levels of cytokines, their receptors and proteinases such as matrix metalloproteinase (MMPs) in abdominal aortic aneurysm (AAA) by cDNA array and real-time RT-PCR methods.
Methods and Results: Aortic samples from the maximally dilated and non-dilated (control) regions were obtained from 22 patients with graft replacement surgery for AAA. The ^{32}P-labeled cDNA probe mixture synthesized from 5 microgram total RNA with gene-specific primers was hybridized with 388 cytokine-related cDNA array. For 4 pairs of AAA and control, the signal intensities for each target cDNA normalized to GAPDH were compared. Overt upregulation in AAA was observed for Interleukin (IL)-8 and its receptor such as CXCR-2, which were further confirmed by real-time RT-PCR method. We also determine gene expression level of MMP-1, 3, and 9. The expression levels for the IL-8 and CXCR-2 genes were significantly upregulated in AAA compared with control as followed: IL-8, 0.53 ± 0.16 Versus 0.11 ± 0.04 (p<0.01); CXCR-2, 2.04 ± 0.75 Versus 0.29 ± 0.10 (p<0.01). Under these conditions, there was significant upregulation of MMP-1 (4.48 ± 2.01 Versus 0.26 ± 0.12, p<0.01) and MMP-3 (5.01 ± 0.97 Versus 1.89 ± 1.00, p<0.05) genes.
Conclusion: Sustained upregulation of IL-8, a CXC-chemokine, and its receptor, CXCR-2, was observed in the AAA associated with overexpression of MMPs, suggesting that inflammatory process with proteinase activation contributes to the development of AAA. This pathway may be an alternative gene or drug target for the treatment of AAA.

Noon

1059-13 Detection and Propagation of Calcified Nanostructures From Human Aneurysms

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Background: Mechanisms leading to vascular calcification remain incompletely understood. Nanometer-sized, mineralized structures recognized by a commercially available monoclonal antibody (8D10, Nanobac OY) are present in calcified human aneurysms. These structures were not detected by TUNEL staining, suggesting they were not apoptotic bodies. The 8D10 antibody is directed against nanobacteria, a controversial, slow-growing, and calcifying microorganism. Therefore, experiments were designed to determine whether structures from aneurysms are viable, nano-sized organisms.
Methods: Aneurysms (n=3) collected as surgical waste were decalcified, sterile filtered (0.22 μm), and cultured in DMEM containing gamma-irradiated calf serum.
Results: In 2 of 3 cultures micron-sized particles visible by light microscopy increased in number over 4-6 weeks. The negative culture came from an aneurysm without stainable nanoparticles. With transmission electron microscopy (EM), cultured particles showed an inner core surrounded by a shell of calcium phosphate (documented via energy dispersive microanalysis). After dissolution of the shell with EDTA, spherical structures of 50-100 nm were seen by scanning EM. These cultured particles incorporated [^{3}H]uridine at a rate 2.3 times greater than control cultures of DMEM containing serum and inorganic hydroxyapatite (HA) crystals (P<0.01). Therefore, these nanostructures appear to synthesize RNA. Particles cultured from aneurysms also stained with the 8D10 antibody, and SDS-PAGE of extracted proteins revealed multiple distinct bands, including one (M_r 47 kDa) recognized by the 8D10 antibody. The pattern of proteins extracted from inorganic HA crystals incubated with DMEM and calf serum did not contain the 47-kDa band recognized by the 8D10 antibody.
Conclusion: In conclusion, these results suggest that viable nano-sized organisms are present within calcified human arterial tissue. A cause and effect relationship between the presence of these organisms and development of arterial calcification remains to be determined.

Noon

1059-14 C825T Polymorphism of the G-Protein Beta(3) Subunit Gene and Atrial Fibrillation: Association of the TT Genotype With a Reduced Risk for Atrial Fibrillation

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Background: A polymorphism consisting of a C825T substitution in the G-protein beta(3) subunit gene (GNB3) has been associated with enhanced signal transduction via heterotrimeric G-proteins. Furthermore, association between enhanced human atrial inward rectifier potassium currents and the TT genotype, but not the CT genotype has been shown. Therefore, we investigated a possible impact of the GNB3 C825T polymorphism on atrial fibrillation by genotyping patients with atrial fibrillation and a control cohort of patients without atrial tachyarrhythmias. Methods: 291 consecutive patients admitted to our center with symptomatic paroxysmal or persistent AF (mean age 58±10 years) and 292 control patients (59±11 years) were genotyped for the C825T polymorphism. Patients with coronary heart disease, valvular heart disease or cardiomyopathy were excluded from the study in order to avoid the influence of disease-related atrial remodeling. Control subjects were 292 consecutive patients admitted to our center in which coronary artery disease was excluded by coronary angiography. The control group had a similar incidence of cardiovascular risk factors (hypertension, hypercholesterolemia, body mass index) as the group with atrial fibrillation. Results: The prevalence of the GNB3 TT genotype was significantly lower in patients with atrial fibrillation (5.8%) than in the control group (12.0%); however, no significant differences in the frequencies of the CT and CC genotypes were found. TT genotype was associated with a 51% decrease in the unadjusted risk (OR: 0.49, 95% CI=0.28-0.85, P=0.01) and a 54% decrease in the adjusted risk (OR from a multivariant model: 0.46, 95% CI=0.24-0.87, P=0.02) for the occurrence of atrial fibrillation. Sinus cycle length, P wave duration, PQ interval and rate-corrected QT interval (QTc) during sinus rhythm were not influenced by the genotype. Conclusion: The present study suggests an association between the GNB3 TT genotype and a reduced risk for the occurrence of atrial fibrillation.

Noon

1059-15 Early Administration of Clopidogrel Is Safe After Off-Pump Coronary Artery Bypass Surgery

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Background: Patients who undergo off-pump coronary artery bypass (OPCAB) may be hypercoagulable with an increased risk of early graft thrombosis due to the lack of platelet dysfunction that accompanies “on-pump” coronary artery bypass. Clopidogrel, a