9:00 a.m.

881-3 Role of Acute Phase Reactants and Infection as Predictors for Major Adverse Clinical Events and Angina Pectoris After Percutaneous Coronary Intervention

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Background: Inflammation may play an important role in the development of restenosis after percutaneous coronary intervention (PCI). Involvement of previous infection with Cytomegalovirus (CMV) and Chlamydia pneumoniae has been postulated. We investigated the value of acute-phase reactants and infection with CMV and Chlamydia pneumoniae in predicting major adverse clinical events (MACE) and angina pectoris (AP) after PCI.

Methods: A total of 600 patients were enrolled (mean age 61.6±10.5 yr, 69.9% male, 64.2% stented). At baseline CMV-IgG, Chlamydia IgG, and IgA were measured using ELISA methods. Also the pro-inflammatory parameters C-reactive protein (CRP), IL-6 and Lipoprotein(a) were measured. All patients were followed for 8 months. Cardiac death, target lesion revascularization (PCI, bypass surgery) and myocardial infarction were recorded as MACE.

Results: In Chlamydia pneumoniae IgA seropositive patients the incidence of MACE was 12.4% versus 6.9% in seronegative patients (p=0.028). Lipoprotein(a) and CRP levels were significantly higher in patients with MACE (280 mg/L versus 192 mg/L, p=0.034 respectively 4800 mg/L versus 7230 mg/L, p=0.045). Chlamydia pneumoniae IgG, CMV-IgG, and IL-6 showed no statistically significant difference.

CRP levels were also higher in patients with repeat AP (5165 mg/L versus 4071 mg/L, p=0.026). Chlamydia and CMV seropositivity, IL-6 and Lipoprotein(a) were no predictors of repeat AP. Correction for stenting and age did not influence the outcome of the various parameters.

Conclusion: The present study indicates that Chlamydia pneumoniae IgA seropositivity predicts MACE after PCI. Patients with MACE also showed higher levels of Lipoprotein(a) and CRP suggesting that these acute-phase reactants are correlated with clinical events after PCI. CRP levels are also correlated with repeat AP after PCI.

9:15 a.m.

881-4 No Impact of Statin Treatment on Integrin Expression on Leucocytes in Patients With Stable Coronary Artery Disease

Joerg Friedrich, Karl M. Siegler, Christoph Anders, Rahime Gök, Bärbel Klein, Ralf Winkler, Stefan Weise, Ariane Kunkler, Gunnar Taubert, Jochen Senges, *Herzentrum Ludwigshafen, 67063 Ludwigshafen, Germany, Institut für Hämatologie und Transfusionsmedizin, 67063 Ludwigshafen, Germany.*

Background: activated leucocytes are discussed to be involved in progression of coronary artery disease (CAD). Studies have shown that statin treatment reduces CAD progression and the number of acute coronary events.

Objective: aim of this study was to compare leucocyte expression of the integrins CD11b and CD18 and the adhesion molecule CD31, all involved in transepithelial migration of monocytes and neutrophils, in consecutive patients with or without statin treatment.

Methods: 1876 participants of the **LURIC** (**Lu**dwigshafen **R**isk and **C**ardiovascular health) study , an ongoing prospective study of environmental and genetic risk factors in cardiovascular disease in patients with angiographically documented coronary status, were analysed. All patients had coronary stenosis of at least 20% in one vessel, unstable and patients with acute ST- or Non-ST infarction < 2 weeks were excluded. Flowcytometry was performed to determine expression of CD11b, CD18 and CD31 on monocytes (M) and neutrophils (N).

Results: Given as mean fluorescence intensity (MFI)/Median values

	No Statins	Statins	P- value
CD11b (M)	6.0	5.9	n.s.
CD18 (M)	8.5	9.3	0.02
CD31 (M)	20.9	22.4	<0.001
CD11b (N)	4.2	4.0	n.s.
CD18 (N)	3.7	4.0	0.04
CD31 (N)	5.8	6.2	0.03

Data were adjusted for age, gender, smoking, hypertension, diabetes, cholesterol, C-reactive protein

Conclusions: 1) In the patients studied a slightly but significantly higher expression of the integrin CD18 and of CD31 was observed in the group of patients treated with statins 2) The antiinflammatory effect of statins in CAD was not related to suppresion of CD11b, CD18 or CD31 expression on monocytes and neutrophils.

9:30 a.m.

881-5 Increased C-Reactive Protein Levels in Women at Increased Cardiovascular Risk Predict One-Year Events Only When Associated With Increased Interleukin-6 Levels

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Elevated CRP levels suggestive of heightened inflammatory state in vascular conditions are often associated with elevated Interleukin-6 (IL-6) levels. It has been suggested that CRP and IL-6 may be a predictors of unfavorable outcome in postmenopausal women (PMW) receiving hormone replacement therapy. Because of the possible metabolic effect of HRT on CRP the relative predictive importance of CRP and IL-6 levels in PMW receiving HRT remains to be elucidated. Therefore we studied 346 consecutive PMW (mean age 66±9 years) with cardiovascular risk >20 in 10 years and receiving HRT for at least one year, by measuring CRP and IL-6 before, and 3 months and 1 year after initiation of HRT.

Overall HRT increased CRP levels by 76% while decreased IL-6 levels by 8%. During 1 year follow up 1 patient died (non cardiac), and 4 had a major cardiovascular event.

PMW with events had elevated CRP levels compared with baseline but within the mean±1SD compared to levels obtained in all women after initiation of HRT. IL-6 levels in PMW with events were significantly higher in PMW with events than in those without events. IL-6 were predictor of future events while elevated CRP levels were associated with an unfavorable outcome only when IL-6 levels were also elevated. In a stepwise multivariate analysis IL-6 levels were a stronger predictor of outcome than CRP. CRP levels were predictors of future events only after removal of IL-6 levels and presence of cardiovascular symptoms from the analysis.

In conclusion CRP levels are increased in PMW receiving HRT. Elevated IL-6 levels may identify those PMW at increased one year risk. CRP levels predict events only when they are coupled with IL-6 levels.

9:45 a.m.

881-6 Endothelial Microparticles Correlate With High-Risk Angiographic Lesions in Acute Coronary Syndromes

Leon Bernal-Mizrachi, C. Fierro, E. McDonough, J. Purow, W. Jy, H. S. Velasquez, J. J. Jimenez, L. L. Horstman, Y. S. Ahn, E. Marchena, *University of Miami, Miami, Florida.*

Background We previously reported high EMP in patients with acute coronary syndromes (ACS). Here, we investigated the relation between EMP and morphology of coronary lesions. **Methods** Three cardiologists blinded to EMP levels independently reviewed angiograms of 43 pts (14 MI and 20 UA) using Ambrose classification. α-CD31-PE and α-CD42-FITC were added to platelet-poor plasma from ACD blood and $CD31^+$/$CD42^-$ microparticles <1.5 μm were assayed as EMP by flow cytometry. Mann-Whitney rank sum test was used for statistics. **Results** Angiographic findings were grouped as high risk *vs* low risk. Mean ±SE of EMP per μL are shown in the table. **[A1]** EMP in eccentric type II or multiple irregular lesions were 2.5-fold higher than in type I or concentric lesions in ACS. **[A2]** Lesions with thrombi had 3-fold higher EMP than those without. **[B1]** Milder stenosis <45% had 3-fold higher EMP than those with stenosis >45% and 5-fold higher EMP than those without stenosis (3135 ±1102; p<0.01). **[B2]** Among patients with type 2 lesions, those with first ACS had 4-fold EMP compared to those with recurrent ACS.

High risk	EMP	Low risk	EMP	p Value
[A1] Eccentric type II or multiple irregular lesions N=23	10706±2787	Eccentric type I or concentric lesions N=12 N=12	4248±1339	<0.05
[A2] ACS with thrombi N=9	17187±5864	ACS w/o thrombi N=22	4952 ±827	=0.05
[B1] Stenosis, >45% N=21	5834 ±5085	Stenosis >20% <45% N=9	15212±5462	=0.05
[B2] Recurrent ACS & eccentric type II lesion N=12	4425 ±985	First ACS & eccentric type II lesion N=11	16464±4762	<0.01

Conclusion **[A]** EMP levels correlate with high risk angiographic lesions. EMP were higher in eccentric type II, multiple irregular as well as lesions with thrombi in ACS patients. **[B]** In contrast, EMP were lower in >45% stenosis and recurrent ACS. This suggest that damage from severe stenosis or recurrent ischemia reduces capacity of endothelium to release EMP. EMP appear to be a useful marker in detecting higher risk angiographic lesions in ACS.