1123-110 **Long-Term Prognosis of Patients With Myocardial Infarction and Normal Coronary Angiography: Impact of Inherited Coagulation Disorders**

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Prevalence of inherited coagulation disorders including factor V Leiden is higher in pts with myocardial infarction (MI) and normal coronary angiography (NCA) than in pts with MI and coronary artery stenosis. Despite a good long-term prognosis of pts with NCA, the prognosis of those with inherited coagulation disorders is unknown. Objectives. The purpose of this study was to compare the clinical thrombosis outcome of pts with (GpI) or without (GpII) an inherited coagulation disorders who suffered from an acute MI with NCA. Methods. From September 1994 to November 2000, 82 consecutive pts (mean age 49±15 years; 29 females) with MI but NCA were recruited. Results. Twelve pts (15%) had an inherited coagulation disorder: APC resistance in 8 pts, factor XII deficiency in 3 pts and protein C deficiency in 1 pt. GpI and GpII were statistically similar regarding age (45±11 vs 50±16 years-old), gender (33 vs 36% female), tobacco (50 vs 53%), diabetes mellitus (8 vs 10%), hypertension (25 vs 17%), obesity (8.3 vs 14%), coronary heart disease family history (33 vs 19%), hypercholesterolemia (50 vs 21%; p=.08), and left ventricular ejection fraction (58±13 vs 61±13%). Prevalence of coronary spasm did not differ significantly (8.3% vs 17%) between the two gps. All patients were initially treated with antiplatelet agents with the exception of one (8%) in GpI and 6 (9%) in GpII who were under oral anticoagulant therapy (ns). The mean follow-up was 57±26 (range from 2-91 months). Four patients were lost of follow-up, 0% in GpI and 5.7% in GpII (ns). During the outcome, 12/78 (15.4%) thrombotic events occurred, including venous thrombosis or pulmonary embolism (1/12 vs 1/66), reinfarction (2/12 vs 4/66), and stroke (2/12 vs 2/66), two events in one patient (GpI). Kaplan-Meier event-free survival, with combined end-point defined as venous thrombo-embolic event, reinfarction, or stroke differed between the two groups: 4/12 (33.3%) in GpI and 7/66 (10.6%) in Gp II (p <.02). Conclusions. Patients with MI, NCA and congenital coagulation disorder present a high risk of thombotic recurrence under antiplatelet agent. This new finding supports the hypothesis that anticoagulation therapy should be recommended in this selected situation.

1123-111 **Interleukin-18 Levels Suggest Different Inflammatory Pathways in Unstable Angina**

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Background. Interleukin (IL)-18 is a cytokine that plays a central role in the inflammatory cascade, inducing IFN-gamma production by Th1 lympochytes, acting in sinergy with IL-12. IL-18 has been recently associated with increased risk of cardiac event. We aimed to investigate if IL-18 has a role in the acute phase response of unstable angina (UA) and if there is a correlation between IL-18 and other inflammatory markers. Methods. We studied 38 UA (Braunwald class IIIB) patients, 26 stable angina patients (SA) and 5 healthy controls (C). In all patients we measured plasma levels of IL-18, C-reactive protein (CRP) and IL-1Ra, a reliable indicator of IL-1 family cytokine production. UA patients were subgrouped according to CRP levels in UAH (25 patients with CRP >3 mg/L) and in UAL (13 patients with CRP <3 mg/L). Results. IL-18 levels (ng/mL; median, range) were significantly higher in UAH and SA vs UAL and C (respectively: 0.098, 0.003-2.122; 0.178, 0.003-0.904; 0.012, 0.003-0.78; 0.003, 0.003-0.03; p<0.01). CRP levels (mg/L; median, range) were significantly higher in UAH vs UAL, SA and C (respectively: 9.8, 3.1-43.4; 0.8, 0.8-2.9; 1.65,0.8-29.7; 1.2, 0.8-4.2; p<0.01). IL-1Ra levels (pg/mL; median, range) were significantly higher in UAH vs UAL, SA and C (respectively: 3444, 200-12000; 973, 200-9920; 1746, 200-12000; 325, 200-1984; p<0.01). A positive linear correlation was observed between plasma levels of IL-18 and IL-1Ra (r=0.36, p<0.01), but not between CRP and IL-18. Correlation between CRP and IL-1Ra levels was of borderline significance (p=0.054). Conclusions. Our data confirm that IL-18 is associated with ischemic heart disease, but also indicate that different pathogenetic mechanisms may be involved in UA. In fact UA patients with low CRP also have low IL-18 and IL-1Ra levels, suggesting a different pathogenetic component, possibly unrelated to acute inflammatory response.

1123-112 **Comparison of Repolarization and Inflammatory Activity in Patients With and Without Ventricular Fibrillation During Acute Myocardial Infarction of a Similar Size (CK<1000 U/l)**

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Duration of repolarization and QT-dispersion during acute myocardial infarction (AMI) predict a risk for major cardiac events, if they do not normalize after catheter intervention or thrombolysis. The aim of the present study was to assess the differences in QTc-interval and QT-dispersion during the acute myocardial infarction and assess the potential correlation between the QT-dispersion, QTc and factors as infarction size, C-reactive protein serum level and serum potassium in patients with and without ventricular fibrillation during AMI. Patients and Methods: In 43 patients (CK <1000 U/l) with (VF) and 77 patients without ventricular fibrillation (Non-VF) during the acute phase of myocardial infarction QTc, QT-dispersion, serum potassium, C-reactive protein and CK max were documented and analysed. Results: While there was a similar distribution of gender, age and CK-level we found a significantly higher baseline heart rate in patients with VF (87+/- 2,85 bpm) vs Non-VF (71.11 +/- 2.06 bpm). No significant differences were found in QTmax. (0.43+/-0.09 seconds(s). vs 0.44 +/- 0.08 s) , QTcmax. (0.5 +/- 0.06 s vs 0.5 +/- 0.05 s) and in serum potassium-levels (4.02+/- 0.6 vs 4.29 +/- 0.6 mmol/l). A significant difference was found for C-reactive protein levels (44.08 +/- 6.9 vs 12.25 +/- 2.7 mg/l, p= 0.025).Conclusion:: 1. The comparison of infarct-size-matched patients with and without VF during an AMI reveals a significantly higher inflammatory activity in patients with VF. 2. The duration of repolarization but not the dispersion correlates positively with C-reactive protein levels in patients with complicating VF versus patients without VF. 3. Inflammatory activity may play a triggering role for the development of ventricular fibrillation in AMI.

1123-113 **Direct Physiological Evidence of Damage to Collaterals During Primary Coronary Angioplasty for Acute Myocardial Infarction**

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Background: Distal microembolisation of thrombotic debris during primary percutaneous coronary intervention(PCI) for acute myocardial infarction (AMI) may result in microcirculatory dysfunction. We tested the hypothesis that microembolisation during balloon angioplasty could result in damage to collaterals.
Methods: we measured Doppler-derived collateral flow index (CFI) in 11 consecutive patients (age 64±11, 9 male, 2 female) who underwent successful primary PCI for AMI (LAD 6, RCA 5). All patients received abciximab.CFI was compared during 1st and 2nd balloon inflations. CK and troponin I measurements were obtained from femoral venous plasma. Area at risk was determined off-line by coronary arteriography.
Results: There was a significant reduction in mean CFI between 1st and 2nd balloon inflation (0.35±0.1 versus 0.22±0.1, p=0.01, 37% mean reduction). 24 hour CK and troponin I results were compared between patients with >30% reduction in CFI and those with <30% reduction in CFI. CFI reduction >30% was associated with higher CK and troponin levels at 24 hours independent of time to reperfusion or area at risk. The percentage reduction in CFI between 1st and 2nd inflation correlated positively with 24 hour troponin levels (r=0.6, p=0.04).
Conclusion: this study demonstrates an abrogation of collateral flow in response to sequential balloon inflations during primary PCI for AMI associated with a greater degree of myonecrosis. We suggest that this phenomenon is a result of damage to collaterals.

	CFI Reduction >30%	CFI Reduction <30%	p
n	5	6	
CK (U/L)	1214±604	521±273	0.03
Troponin I (u/L)	205±107	86±50	0.04
Reperfusion (hrs)	8±5	11±8	NS
Area at risk (%)	25±2	24±9	NS

POSTER SESSION

1124 Platelets and Platelet Inhibition Among Patients With Acute Coronary Syndromes

Monday, March 31, 2003, 3:00 p.m.-5:00 p.m.
McCormick Place, Hall A
Presentation Hour: 3:00 p.m.-4:00 p.m.

1124-89 **Pharmacodynamics of Eptifibatide and Abciximab in Patients With Non-ST Segment Elevation Acute Coronary Syndromes (TAM2)**

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Background: The importance of the relationship between clinical outcome and degree of platelet aggregation inhibition (PAI) induced by GPIIb-IIIa inhibitors used in large trials in patients with non-ST segment elevation (NSTE) acute coronary syndromes (ACS) is increasingly appreciated. In the PURSUIT trial, eptifibatide treatment targeted to achieve ≥80% platelet aggregation inhibition was associated with clinical benefit at 30 days and 6 months. The GUSTO IV ACS trial, however, failed to show any statistically significant benefit of abciximab treatment on 30 day outcomes. This difference might be linked to variability of antiplatelet effects of these agents. **Methods:** We conducted a prospective study in 40 patients with NSTE ACS prior to catheterization or coronary intervention at 3 centers using the PURSUIT dose of eptifibatide (180/0.2) and the GUSTO IV dose of abciximab (0.25/0.125). Blood samples were collected at baseline, and during the infusion at 10 min, 1 hr, 6 hr, 8 hr, 12 hr, 18 hr, and 24 hr. Measurements of ex vivo light transmission aggregometry (LTA) were performed using PPACK anticoagulant and 20 μM ADP agonist. Receptor Occupancy (RO) using the D3 monoclonal antibody assay was also determined in a subset of patients. **Results:** Eptifibatide achieves higher PAI during the entire infusion period than abciximab ($p < 0.01$). At 10 min, average PAI with eptifibatide and abciximab was 88% and 80%, respectively, 95% and 79% at 6 hours, and 97% and 79% at 24 hours. There was also more variability in individual patient response to abciximab. Although average RO for eptifibatide was similar to that of abciximab at 10 min, average RO in the eptifibatide treatment arm was higher at all subse-