**1052-11**
Serum Uric Acid Level Is an Independent Predictor of Restenosis After Successful Percutaneous Transluminal Coronary Stent Implantation

Kazuki Kumagai, Masatake Fukumai, Tatsusho Shimogomara, Takahisa Yamada, Akio Hirata, Masatoshi Asal, Norihiko Makino, Hitohata Kioka, Hitohata Kiko, Osaka Prefectural General Hospital, Osaka, Japan.

**Background:** Inflammatory activities have been shown to be associated with an increased risk for ischemic coronary events after percutaneous transluminal coronary stent implantation (PTCS). Serum uric acid (UA), reflecting oxidative radical activity through xanthine oxidase, might be also a potent marker for chronic inflammation. However, it is not elucidated whether elevated UA levels would be related to target lesion restenosis (TLR) following PTCS. Therefore, we studied whether UA level might be one of predictors of TLR. This study included 217 consecutive patients following successful PTCS without renal dysfunction and without serum UA-lowering drugs. Results: During the follow-up period of 2 years, TLR occurred in 46 patients (21%). In patients with TLR, serum UA level just before PTCS were significantly higher than in patients without TLR (5.5±1.5 vs. 5.8±1.5mg/dl, p=0.0003). Patients with UA of >7.0mg/dl had a significantly higher TLR rate (43%) at 2 years after PTCS than in patients with UA of 7.0mg/dl or less (χ2=0.0001, Kaplan-Meier analysis and log-rank test). Univariate analysis revealed that UA level (p=0.0003), sex (p=0.09), the diameter of stent (p=0.19), sex (p=0.09), the diameter of stent (p=0.19), serum C-reactive protein level (p=0.093) and DM (p=0.044) were predictive of TLR, but that eGFR, smoking, hypertension, hyperlipidemia, acute coronary syndrome, prior revascularization or medications were not. Furthermore, Cox hazard model showed that UA level (hazard ratio: 3.02, 95% CI: 1.34-6.95; p=0.004); serum C-reactive protein level (hazard ratio: 1.45, 95% CI: 1.02-1.95; p=0.044) and the diameter of stents (hazard ratio: 0.95, 95%CI: 0.57-1.64; p=0.408) are independent predictors of TLR. Conclusion: This study indicates that elevated serum uric acid level just before PTCS would be a most powerful independent predictor of TLR following successful PTCS.

**1052-12**
Does Stent Design Affect Platelet Activation? Results of the Platelet Activation in Stenting (PAST) Study

Paul A. GurbeL, Kevin P. Callahan, Michele J. Buczkowski, Victor L. Serebruany, Sinai Hospital, Baltimore, Maryland.

**BACKGROUND:** Platelet activation induced by coronary artery stenting affects clinical outcomes and may be related to stent design. However, little is known about how platelet activation may be affected by the specific design of a stent. METHODS: Patients (n=53) were randomly assigned to stenting with a closed-cell (UNIP, Boston Scientific) or open-cell (TETRA, Guidant) stent. Patients were treated with aspirin and were loaded with 300 mg of clopidogrel in the cath lab and received 75 mg daily thereafter. All stents were deployed at >12 atm. GPllblllla inhibitors were not used. Platelet aggregation (5 mol ADP and 1 pg/ml collagen); and flow cytometry (mean fluorescence intensity (MFI)) to determine platelet activation were less following NIR implantation: 30d ADP aggregation (%) (32.3±9.9, 121±26, p<0.0001); 30d CD31 (136f 46, 91±33, p=0.016); 60d CD31 (226f 105, 134±39, p=0.026); 90d CD31 (239f 121, 146±46, p=0.003); 180d CD31 (195f 106, 137±42, p=0.017); 360d CD31 (174f 96, 115±36, p=0.043); and 30d CD107a (24f 13 vs 16f 5, p=.045) and 24 h CD 107a (22f 13 vs 16f 5, p=.008) were independent predictors of TLR. Conclusion: These results suggest that elevated serum uric acid level just before PTCS would be a most powerful independent predictor of TLR following successful PTCS.

**POSTER SESSION**

**1053**
Adjuvant Drug Therapy in Percutaneous Interventions

Sunday, March 17, 2002, 3:00 p.m.-5:00 p.m.
Georgia World Congress Center, Hall G Presentation Hour: 3:00 p.m.-4:00 p.m.

**1053-1**
Early and Sustained Survival Benefit Associated With Statin Therapy at the Time of Percutaneous Coronary Intervention

Albert W. Chen, Deepak L. Bhatt, Derek P. Cheit, Martin J. Quinn, David J. Miletone, Eric J. Topol.

**Background:** Long-term administration of statin therapy has been shown to reduce major coronary events and cardiac mortality in clinical trials. Beyond lipid lowering, statins favorably affect platelet aggregation, thrombosis, endothelial function, inflammation, and plaque stability, which may potentially improve outcome after PCI. Therefore, we hypothesized that statin therapy is an early beneficial effect in patients undergoing PCI.

**Methods:** Each year from 1993 through 1999, we prospectively collected data and followed our first 1,000 patients undergoing PCI. Baseline, procedural, and 6-month data of statin-treated and non-statin-treated patients were compared. Propensity score and multivariate survival analysis were used to adjust for heterogeneity between the 2 groups. Results: Of 6,647 patients with complete follow-up, 23.5% were treated with statin at the time of the procedure. Statin therapy was associated with a 60% lower mortality at 30 days (1.0% vs 2.5%; p = 0.0007) and a 37% reduction at 6 months (3.1% vs 4.9%; p = 0.003). After adjusting for the propensity to receive statin therapy prior to the procedure and for other confounders, statin therapy remained an independent predictor for survival at 6 months following PCI (HR 0.58; 95% CI: 0.46-0.93, p = 0.017). The mortality benefit was independent of peri-procedural MI, recurrent MI, and target vessel revascularization.

**Conclusion:** Statin therapy among PCI patients was associated with a significant mortality lowering at early and intermediate term follow-up. The mechanism of survival benefit in statin beyond lipid-lowering needs further elucidation.

**1053-2**
A Blinded Multicenter Dose Escalation Phase IIa Study of the Factor Vila/Tissue Factor Inhibitor Recombinant Nematode Anticoagulant Protein C2 In Patients Undergoing Ejective Percutaneous Coronary Intervention


**Introduction:** A multicentered, randomized, double-blind, placebo controlled, dose escalation study in patients undergoing elective percutaneous coronary intervention (PCI) evaluated the safety of recombinant Nematode Anticoagulant Protein C2 (rNAPc2), a potent inhibitor of the factor Vila/Tissue factor (FVila/TF) complex. Methods: Patients received either placebo or rNAPc2 from one of 4 dose groups as a single SC administration 2-4 hours before PCI, as well as aspirin and IV unfractionated heparin (activated clotting time > 250 sec). The primary endpoints were femoral compression time (FCT) after sheath removal, and incidence of major and minor bleeding. Secondary endpoints included the plasma concentration of protrombin activation fragment 1+2 (F1+2) over time. Results: 1052-11 Serum Uric Acid Level Is an Independent Predictor of Restenosis After Successful Percutaneous Transluminal Coronary Stent Implantation

Kazuki Kumagai, Masatake Fukumai, Tatsusho Shimogomara, Takahisa Yamada, Akio Hirata, Masatoshi Asal, Norihiko Makino, Hitohata Kioka, Hitohata Kiko, Osaka Prefectural General Hospital, Osaka, Japan. **Background:** Inflammatory activities have been shown to be associated with an increased risk for ischemic coronary events after percutaneous transluminal coronary stent implantation (PTCS). Serum uric acid (UA), reflecting oxidative radical activity through xanthine oxidase, might be also a potent marker for chronic inflammation. However, it is not elucidated whether elevated UA levels would be related to target lesion restenosis (TLR) following PTCS. Therefore, we studied whether UA level might be one of predictors of TLR. This study included 217 consecutive patients following successful PTCS without renal dysfunction and without serum UA-lowering drugs. Results: During the follow-up period of 2 years, TLR occurred in 46 patients (21%). In patients with TLR, serum UA level just before PTCS were significantly higher than in patients without TLR (5.5±1.5 vs. 5.8±1.5mg/dl, p=0.0003). Patients with UA of >7.0mg/dl had a significantly higher TLR rate (43%) at 2 years after PTCS than in patients with UA of 7.0mg/dl or less (χ2=0.0001, Kaplan-Meier analysis and log-rank test). Univariate analysis revealed that UA level (p=0.0003), sex (p=0.09), the diameter of stent (p=0.19), sex (p=0.09), the diameter of stent (p=0.19), serum C-reactive protein level (p=0.093) and DM (p=0.044) were predictive of TLR, but that eGFR, smoking, hypertension, hyperlipidemia, acute coronary syndrome, prior revascularization or medications were not. Furthermore, Cox hazard model showed that UA level (hazard ratio: 3.02, 95% CI: 1.34-6.95; p=0.004); serum C-reactive protein level (hazard ratio: 1.45, 95% CI: 1.02-1.95; p=0.044) and the diameter of stents (hazard ratio: 0.95, 95%CI: 0.57-1.64; p=0.408) are independent predictors of TLR. Conclusion: This study indicates that elevated serum uric acid level just before PTCS would be a most powerful independent predictor of TLR following successful PTCS.
Conclusions: In this large pilot experience, rates of ischemic and bleeding events among patients undergoing percutaneous coronary intervention (PCI). Background: Female gender is a known potent risk factor for post-procedural bleeding regardless of gender. However, this effect was most striking among female patients (Table). Correlating a concomitant safety and efficacy endpoint of death, MI, revascularization, and major hemorrhage, bivalirudin was associated with a 50% relative risk reduction in adverse events in women (14/890 [20.4%] vs 69/897 [9.9%] (p<0.0001), and a 35% relative risk reduction in men: 171/1461 (11.7%) vs 111/1462 (7.6%) (p=0.0001)

Conclusions: Bivalirudin is associated with a substantial reduction in bleeding and ischemic events in both men and women, but the improved safety profile is especially prominent among women. Such patients with an increased risk of bleeding during PCI may benefit if bivalirudin is used in place of unfractigrated heparin.

**Major Hemorrhage**

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<tr>
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<th>Heparin</th>
<th>Bivalirudin</th>
<th>p Value</th>
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<tbody>
<tr>
<td>Women</td>
<td>(107/390) 15.5%</td>
<td>(37/267) 5.3%</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Men</td>
<td>(92/281) 6.9%</td>
<td>(94/164) 2.7%</td>
<td>&lt;0.0001</td>
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**A New Rapid Ecarin Clotting Time Assay but Not Activated Clotting Time Strongly Correlates With Bivalirudin Concentration: A Percutaneous Coronary Intervention Study**


Background: Recently, direct thrombin inhibitors (DTI) have been approved for anticoagulation in percutaneous coronary interventions (PCI). The accuracy of the activated clotting time (ACT), used for monitoring heparin, is uncertain for patients receiving DTI. A new point-of-care assay has been developed based on the ecarin clotting time (ECT), a direct measurement of anti-Factor IIa clotting time (ACT), used for monitoring heparin. We enrolled 64 consecutive PCI patients receiving the DTI bivalirudin at The Cleveland Clinic. We compared point of care ECT and ACT to the direct measurement of anti-Factor IIa activity during PCI. Methods: To test the accuracy of available ACT assays (Hemochron and Pro-DM) and the rapid ECT assay (Pharmacentis), we enrolled 64 consecutive PCI patients receiving the DTI bivalirudin at The Cleveland Clinic. We compared point of care ACT and ECT to central laboratory anti-Factor IIa activity levels. The anti-Factor IIa activity levels were determined using Ecarin Clotting Time (ECT) and Hemochron ACT (Hematime, Procton & Gamble) and rapid ECT (Pharmacentis). Results: Bivalirudin concentration correlated well with ECT sample (r=0.76, p<0.0001) and ACT sample (r=0.73, p<0.0001). There was also poor correlation between ECT and Hemochron ACT (r=0.27, p=0.04) and ECT and pro-DM ACT (r=0.52, p=0.001). There was poor correlation between pro-DM ACT and Hemochron ACT (r=0.40, p=0.002). Conclusion: ECT is a reliable measurement of anti-coagulation in patients receiving bivalirudin, while ACT has poor correlation with bivalirudin concentration. Assays not in poor correlation between ACT and ECT in such patients. These findings have important implications for guiding the extent of anticoagulation during PCI with DTI.