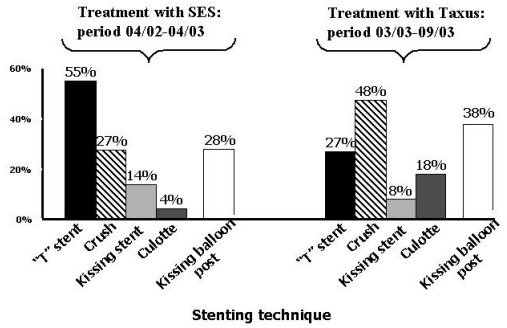


POSTER SESSION

1025

Percutaneous Interventions: Pharmacologic and Biologic Adjuncts

Sunday, March 07, 2004, Noon-2:00 p.m.
Morial Convention Center, Hall G
Presentation Hour: 1:00 p.m.-2:00 p.m.

1025-41 Insulin Sensitizers Are Associated With Improved Outcomes in Diabetic Patients Undergoing Brachytherapy

John A. Kao, Mark Grise, Peter Castarella, Huan Giap, Aniradha Koka, Gerard Huppe, Kathleen Sirkin, Prabhakar Tripuraneni, Paul S. Teirstein, Scripps Clinic, La Jolla, CA

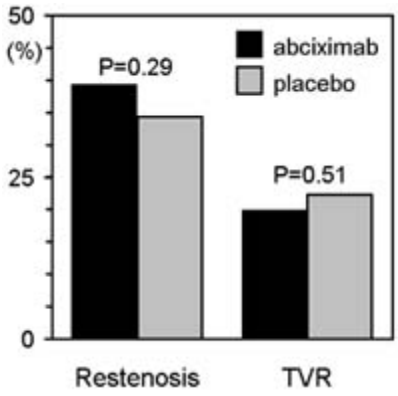
BACKGROUND: Diabetics have worse baseline disease and higher restenosis rates than non-diabetics. Insulin sensitizers may decrease cardiovascular events in diabetics. Our goal was to determine the effect of insulin sensitizers on clinical events of diabetics undergoing brachytherapy for in-stent restenosis.
METHODS: Diabetics receiving brachytherapy in SCRIPPS I, II, III, and IV at Scripps Clinic were divided into two groups; insulin sensitizers (biguanides or thiazolidinediones, n=50), and non-sensitizers (insulin, sulfonylureas or diet, n=67). Clinical events were defined as target vessel revascularization (TVR), MI (STEMI and NSTEMI), non-TVR, and death.
RESULTS: By 12 months a significant reduction in the composite endpoint was observed in the insulin sensitizer group with a significant decrease in the individual endpoint of Death. Treatment with an insulin sensitizer was the strongest predictor of clinical events (OR=3.47, p=0.0035). This effect was independent of adjunctive medical therapy for coronary artery disease. Patients treated with insulin alone had equivalent outcomes compared to patients treated with sulfonylureas or sulfonylureas with insulin (p>0.05 for all comparisons).
CONCLUSIONS: Insulin sensitizers improve clinical outcomes and convey a mortality benefit in diabetics undergoing brachytherapy for in-stent restenosis. The increased events in the non-sensitizer group is not driven by by patients receiving insulin therapy.

	Sensitizers N=50	Non-Sensitizer Therapy N=67	P Value
6-Month Combined Event Rate (%)	27.3%	31.9%	0.72
Death	0	7%	0.13
MI	6%	9%	0.81
TVR	18%	18%	0.33
Non-TVR	15%	10%	0.33
12 Month Combined Event Rate (%)	37%	57.4%	**0.041**
Death	0	10%	**0.049**
MI	6%	12%	0.44
TVR	30%	30%	0.61
Non-TVR	10%	15%	0.61

1025-42 Abciximab Administration for the Prevention of Angiographic Restenosis in Small Coronary Arteries: Results From the Randomized ISAR-SMART-2 Trial

Jörg Hausleiter, Adnan Kastrati, Julinda Mehilli, Helmut Schühlen, Sonja Siebert, Franz Dotzer, Josef Dirschinger, Albert Schömig, Deutsches Herzzentrum München, Munich, Germany, 1.Medizinische Klinik, Klinikum rechts der Isar, Munich, Germany

On the basis of non-dedicated studies it is believed that abciximab reduces the risk of restenosis after percutaneous coronary interventions. In the current multi-center trial, patients with symptomatic coronary artery disease and lesions in small coronary arteries (vessel size < 2.8mm) were randomized to receive stenting (phosphorylcholine-coated stent) or PTCA as well as abciximab or placebo by a 2x2 factorial design. The objective of the pharmacological aspect of the ISAR-SMART-2 trial was to assess whether abciximab administration is associated with a reduction in angiographic restenosis in small coronary arteries.
Methods: From July 2000 through May 2002, a total of 502 patients were randomly assigned to abciximab administration (251 pts) or placebo (251 pts). The primary endpoint of the study was the incidence of angiographic restenosis (>= 50% diameter stenosis) at repeat angiography (available in 82% of patients). All patients received a loading dose of 600mg clopidogrel at least 2 hours before the intervention.
Results: The incidence of major adverse cardiac events did not differ at 30 days after intervention. The restenosis rate and the rate of target vessel revascularization at 1 year are shown in the Figure.
Conclusion: These results show that abciximab administration on top of an high-dose clopidogrel loading does not provide protection against restenosis after percutaneous coronary interventions in small coronary arteries.



1025-43 Bivalirudin Confers Antiplatelet Effects in Patients Undergoing Percutaneous Coronary Intervention

Sunil X. Anand, Michael C. Kim, Mazullah Kamran, Samin K. Sharma, Frank Carbon, Erdal Cavusoglu, Juan F. Viles-Gonzalez, Juan J. Badimon, Jonathan D. Marmur, The Zena and Michael A. Weiner Cardiovascular Institute, Mount Sinai School of Medicine, New York, NY, SUNY Health Science Center at Brooklyn, Brooklyn, NY

Background: Thrombin activity is paramount in the formation of platelet-mediated acute thrombosis precipitated by atherosclerotic plaque rupture or endothelium disruption resulting from mechanical intervention. The objective of this study was to investigate and compare the effects of a bivalent direct thrombin inhibitor, bivalirudin, with unfractionated heparin (UFH) on platelet function in the setting of percutaneous coronary intervention (PCI).
Methods: Patients (mean age 61.7 ± 11.1) were administered an intravenous 0.75 mg/kg bolus followed by a 1.75 mg/kg/hr infusion of bivalirudin (n=12) or an intravenous 40-50 U/kg bolus of UFH (n=15) during PCI. No patient received a pre-loading dose of clopidogrel. Blood samples were collected from patients at baseline and 5 minutes after drug administration from the femoral artery access sheath. To determine platelet function (by assessing platelet surface coverage), the cone and platelet analyzer assay was performed on all samples at a high shear rate (1875 s^{-1}). Computerized planimetry measurements were performed, and 4 to 16 images per timepoint were quantified for platelet surface coverage (PSC) in a blinded fashion.
Results: Direct thrombin inhibition by bivalirudin resulted in a marked 34 ± 6 % reduction in PSC as compared to baseline values (*p < 0.001). On the other hand, UFH demonstrated a remarkable 29 ± 8% increase in PSC as compared to baseline values (*p < 0.05). No clinical sequalae were experienced by any patient.
Conclusion: Our data suggests that direct thrombin inhibition by bivalirudin administration during PCI confers a profound anti-platelet effect as compared to UFH. Interestingly, UFH administration leads to significant platelet activation and aggregation during PCI.