	Balloon	Filters	p-value
	n=57	*n=57*	
TIMI 3 flow			
Baseline	82.5%	86.0%	ns
Final	100%	100%	ns
Blush 3			
Bseline	30.4%	28.6%	ns
Final	66.1%	46.4%	0.05
TFCg			
Baseline	24.1	29.2	ns
Final	14.3	21.3	<0.01
Change	9.8	7.9	ns
TFCn			
Baseline	17.4	13.0	ns
Final	10.9	11.5	ns
Change	6.5	1.5	<0.05
TFC			
Baseline	43.5	40.6	ns
Final	26.0	34.0	0.05
Change	17.4	6.6	<0.05

1063-66 **Arterial Closure Device Decreases Vascular Complications After Percutaneous Coronary Interventions**

Michael C. Kim, Annapoorna S. Kini, Paul C. Lee, Marybeth Duffy, Thomas Joseph, Warren Sherman, Samin K. Sharma, Mount Sinai School of Medicine, New York, NY

Background: Vascular complication is an important determinant of patient satisfaction after Percutaneous Coronary Interevntion (PCI). Many studies have shown that use of closure devices (CD) had lower vascular complications after PCI , but some studies have not.
Methods: We analyzed patients who had PCI from July 1999 to March 2003: 2,033 patients had attempted closure devices (61% Perclose, 27% Angioseal, 6% Vasoseal, 3% Duett, 2% unsuccessful), and 4,419 patients had manual compression.
Results: Using intention to treat analysis, the use of closure device compared to manual compression (MC) resulted in decrease in overall vascular complication (1.4% vs. 2.6%, p=0.0008): specifically, in moderate to large hematomas, or retroperitoneal bleeding (p=0.0062), pseudoaneurysm (p=0.003), but not in vascular surgery (p=0.7)). Successful deployment of the device had a very low vascular complication rate (1.2%). The predictors for CD deployment failure(2% of all devices used) were: Month of July (8.4% vs. 1.7% p<0.0001) and increasing body weight (p=0.03). After successful deployment of CD, the predictors for significant vascular complications included July month, the use of Duett or Vasoseal (7.7% vs. 1.1% in Perclose or Angioseal, p<0.0001), female sex (2.2% vs. 0.8%, p<0.0001), CHF NYHA class (p=0.0034), age (p<0.001), and the use of post-procedural heparin (3.4% vs. 1.1% p=0.04), but not the use of glycoprotein IIb/IIIa (p=0.8). Weight and ACTmax were not predictors for vascular complication after successful deployment of device. If the device fails to deploy, the risk of vascular complication was much higher (18% vs. 1.2%, p<0.0001), especially if glycoprotein IIb/IIIa had been used (21% vs. 0%, p=0.04), mostly moderate or large hematoma. Vascular complication was a significant predictor for in-hospital death (p<0.0001), though the successful or unsuccessful use of CD had no significant statistical impact on survival. Length of stay was significantly lower in CD (1.9±3.1 day vs. 2.6±5.0 days) p<0.0001).
Conclusion: Use of vascular closure devices after PCI resulted in significantly lower vascular complications and a significantly lower length of stay.

ORAL CONTRIBUTIONS

803

Restenosis: Basic Research I

Monday, March 08, 2004, 9:15 a.m.-10:30 a.m.
Morial Convention Center, Hall E-2

9:15 a.m.

803-1 **The Number and Adhesive Properties of Circulating Endothelial Progenitor Cells in Patients With In-Stent Restenosis**

Hylton I. Miller, Jacob George, Itzhak Herz, Emil Goldstein, Solicu Abshizada, Arik Finkelstein, Yoav Michowitz, Gad Keren, Tel-Aviv Medical Center, Tel-Aviv, Israel

Background: Endothelialization is essential to facilitate vessel healing after stent deployment to prevent restenosis. Circulating endothelial progenitor cells (EPC) are present in the peripheral blood and display endothelial functional properties along with ability to home to damaged vasculature. Prolific in-stent intimal growth may be related to impaired endothelialization resulting from a reduced number or dysfunction of circulating EPC.
Methods: 27 patients (pts) after stent deployment had recurrent unstable angina and underwent coronary angiography. In 16 pts in-stent restenosis was demonstrated (group A), in 6 focal and in 10 diffuse. 11 had patent stents (group B). Both groups were similar with respect to drug usage and risk factors. Circulating EPC numbers were determined by the colony-forming unit assay and their phenotype was characterized by endothelial-cell markers. Adhesiveness of EPC from both groups to extra-cellular matrix and to endothelial cells was also assayed.
Results: Pts in both groups with in-stent restenosis and with patent stents displayed a similar number of circulating EPC (26.5±2.6 vs. 25.3±4.8). Pts with diffuse in-stent restenosis exhibited reduced numbers of EPC (24.0±3.9) as compared with patients with focal restenosis (30.7±1.7; p<0.05). Fibronectin-binding was compromised in pts in group A as compared to pts of group B (9.2±2.5 vs. 15.3±3.2 cells/field; p<0.01
Conclusions: Reduced numbers of circulating EPC in pts with diffuse in-stent restenosis and impaired adhesion of EPC from patients with restenosis provides a potential mechanism mediating the exuberant proliferative process of restenosis. These markers, if further validated, could provide means of risk stratifying patients for the likelihood of developing in-stent restenosis.

9:30 a.m.

803-2 **Genetic Predictive Factors for Restenosis After Percutaneous Transluminal Coronary Angioplasty**

Pascalle S. Monraats, Willem R.P Agema, Aeilko H. Zwinderman, Robbert J. de Winter, Rene A. Tio, Pieter A.F.M Doevendans, Harry J.G.M Crijns, Moniek P.M de Maat, Douwe E. Atsma, Arnoud van der Laarse, Ernst E. van der Wall, J.Wouter Jukema, Leiden University Medical Center, Leiden, The Netherlands

Background: Percutaneous transluminal coronary angioplasty (PTCA) is still limited by the recurrence of luminal stenosis. The underlying mechanisms are not fully understood. Stratifying patients at risk based upon clinical factors has proven difficult so far. However evidence exists that gene polymorphisms may play a role in the restenotic process. The aim of this study was to evaluate if various gene polymorphisms can predict clinically important restenosis after PTCAin an unselected patient population and thereby influence the choice of therapy.
Methods: The GEnetic DEterminants of Restenosis (GENDER) project was a multicenter prospective cohort study, which included 3,146 patients after successful PTCA. Patients with acute myocardial infarction (MI) were excluded. Genotyping was performed for different polymorphisms in several candidate genes.
Results: A total of 3,146 patients (age 62.1±10.7 yrs) were followed for 10±3 months. Of the patients 2,250 (71.5%) were male, 459 (14.6%) had diabetes and 1,459 (46.4%) had multivessel disease. The majority was treated for stable angina. Stenting was performed in 2,340 (74.4%) patients. Target vessel revascularisation (TVR) after the first month of follow-up by either CABG or PTCA was necessary in 304 patients (9.7%). So far we identified an association between three polymorphism and TVR. The adrenergic beta-2 receptor and stromal cell derived factor (p=0.022, p=0.038 respectively) were associated with increased risk of TVR. Small inducible cytokine subfamily A (eotaxin) (p=0.057) showed a protective association with TVR.
Conclusion: So far in this unselected large group of patients treated with PTCA, we already identified three polymorphisms in different candidate genes that were related to restenosis after PTCA. More genotyping is being performed. Our results can contribute to the unravelling of the restenotic process and might provide a better risk stratification and lead to a more tailored therapy for patients with identified increased risk of restenosis after PTCA.