1061-63 Laser Angioplasty for Within Stent Restenosis – Final Results of the LARS Surveillance Study

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Coronary stenting is associated with a restenosis rates of 10 to 20%. When treated with repeat PTCA the recurrence rate can be as high as 80%. The long term result may be more favourable by debulking tissue like with eximer laser coronary angioplasty (ELA).

Methods: In this prospecitve, multicenter study we investigated safety and efficacy of ELA with adjunctive PTCA in restenotic (>70% diameter stenosis) or occluded stents in 414 vessels (88% native, 12% vein grafts) with 494 stents implanted. The lesion length was 19.0 ± 13.1 mm with 22 different types of stents (25% AVE, 19% Palmaz-Schatz) implanted 5.8 months prior.

Results: Stenosis diameter was reduced by ELA to 42 ± 16% and after adjunctive PTCA (12.6 ± 4.0 atm) to 7 ± 12%. Procedural success was achieved in 92%. Complications after ELA were registered in 57 patients (13.8%) including 2 minor perforations and 20 dissections (4.8%). After adjunctive PTCA in 411 lesions there were 2 more perforations and 45 more dissections (10.9%) which required additional stents in 67 pts (16%). Stent damage (n = 2) and pericardial tamponade (n = 2) were rare. During hospitalization there were 6 deaths (n = 3 cerebral hemorrhages), 2 myocardial infarctions and 10 non-Q-wave infarctions.

Conclusion: ELA was safe and effective in treatment of restenoses within stents. Long term results are pending.

1061-64 First Clinical Experience With Intravascular Low Power red Laser Light Therapy for Prevention of Restenosis Following Coronary Stenting

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Background: Low power red laser light therapy (LPRLT) has been shown to reduce restenosis following balloon angioplasty in animal models.

Methods: The purpose of this study was to determine the safety and efficacy of LPRLT to prevent restenosis following coronary stenting. Three groups of patients received LPRLT using the cold laser illuminator™ (Global Therapeutics, Boulder, CO) with a laser power of 1.1 J/cm². Group I and II included patients with either a suboptimal angioplasty result (n = 26), or recurrent restenosis after balloon angioplasty (n = 24). Group III included 20 patients with restenosis following coronary stenting.

Results: The primary success rate was 100%. No major in-hospital complications occurred during this study. Thirteen patients underwent a control angiogram because of recurrence of angina pectoris. A significant stent stenosis was found in only 5 patients. Elective coronarogram at 6 months was performed in another 49 pts. Restenosis (DS > 50%) was found in 9 pts (18.4%). Quantitative coronary analysis per lesion treated: minimal luminal diameter (MLD) pre: 0.8 ± 0.5 mm, post: 2.9 ± 0.6 mm, follow-up: 1.9 ± 0.6 mm. Restenosis rate was 20.6%.

Conclusion: These preliminary results suggest that LPRLT after coronary stenting is feasible and safe and potentially has a beneficial effect on stent restenosis in this high risk patient population. A multicenter randomized trial to confirm these results is pending.

1062 Restenosis: New Insights

Monday, March 30, 1998, 3:00 p.m.–5:00 p.m.
Georgia World Congress Center, West Exhibit Hall Level
Presentation Hour: 3:00 p.m.–4:00 p.m.

1062-98 Neointimal and Not Adventitial Proliferation Plays a Role in Restenosis After Percutaneous Transluminal Coronary Angioplasty in Humans – A Histomorphometric Analysis

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Background: Late vascular recoil, considered to result from adventitial proliferation, rather than neointimal formation is presently promoted as the cause of restenosis (RS) after percutaneous transluminal coronary angioplasty (PTCA) in humans.

Material and Methods: Of an autopsy series of 92 post-PTCA patients and based on follow-up angiographic criteria, we performed histomorphometric surface area analysis of 18 coronary arteries with RS (interval PTCA/death = 2 mos ~ 2 yrs) and of 5 coronary arteries with luminal narrowing but not meeting the criteria of RS (interval PTCA/death = 8 mos ~ 2 yrs). The proximal non-dilated sites served as reference.

Result: All PTCA sites showed neointimal tissue, albeit to varying degrees. The mean luminal area at PTCA sites was expressed as a ratio of the mean luminal area at reference sites. This ratio was significantly ($p < 0.0001$) lower at RS sites (0.246 ± 0.104) than at non-RS sites (0.692 ± 0.240). Area quantification of the adventitia showed no statistically significant difference between RS and non-RS lesions. The mean neointimal area was expressed as a ratio of the mean neointimal area + luminal area. This ratio was significantly ($p < 0.01$) higher at RS sites (0.779 ± 0.108) than at non-RS sites (0.545 ± 0.064). The mean total vessel area was significantly ($p < 0.01$) higher at RS sites (10.916 ± 2.849 mm²) than at reference sites (9.250 ± 2.072 mm²).

Conclusions: These observations contradict a key role for adventitial proliferation as a cause of restenosis after PTCA in humans, but rather emphasizes the role of the neointima.

1062-99 Plasma Urokinase Antigen Levels Predict Angiographic Restenosis

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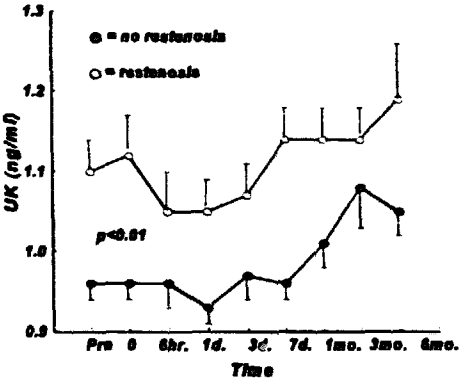
Purpose: To determine the relationship between restenosis and plasma levels of urokinase antigen (UK), urokinase activity (uPA), tissue plasminogen activator (tPA), or plasminogen activator inhibitor (PAI).

Methods: In a prospective study enrolling 159 patients (pts), plasma levels of UK, uPA, tPA, PAI were drawn immediately before and serially after elective PTCA/stenting of a de novo lesion in a native coronary artery. Restenosis, defined as >50% residual lumen diameter stenosis by quantitative coronary angiography was determined at 6 months.

Results: To date, 116 of 126 (92%) of eligible pts. have had angiographic follow-up. In the overall group, uPA levels showed a significant increase at and beyond 3 days postprocedure. The restenosis rate was 24%. The "restenosis" group had higher UK levels overall (including pre-PTCA) than the "no restenosis" group (Figure). No significant differences were observed in the tPA and PAI levels between the two groups.



Conclusions: Plasma urokinase antigen levels identify a group of patients at risk for restenosis post-PTCA. Plasma urokinase activity levels elevate significantly 3 days post-PTCA.

1062-100 Atherosclerotic Progression in the Proximal Non-treated Segment in Patients Undergoing Coronary Interventions Evaluated by Intravascular Ultrasound

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Background: Injury of the endothelial cells may result in plaque formation. Purpose of the study was to evaluate the atherosclerotic changes in the proximal non-treated segments in patients who underwent PTCA.

Methods: Intravascular ultrasound (IVUS) was performed in 59 patients (51 males, aged 55 ± 9 yrs) immediately post successful PTCA and 6 months follow-up using a 3.5 F, 20/30 MHz IVUS catheter (Sonicath, Boston Scientific Co). The vessel and plaque areas in the proximal and distal non-treated reference segments 2 cm proximal and distal to the intervention segment were evaluated.

Results: No significant differences were found concerning the vessel areas between initial examination and at 6-month follow-up in the proximal

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