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4:30

COLCHICINE FOR THE PREVENTION OF RESTENOSIS AFTER CORONARY ANGIOPLASTY

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Colchicine, an antimitogenic agent, has shown promise in the prevention of restenosis after coronary angioplasty (PTCA) in experimental animal models. We randomized 197 pts 2:1 to colchicine 0.6 mg po bid (130 pts) vs placebo (67 pts) for 6 months following elective PTCA. Treatment was begun within 24 hours of PTCA. 96% of all prescribed pills were ingested. The mean age of the study population was 60 years; 169 pts (86%) were men. Demographic characteristics were similar in the two groups. A mean of 2.7 lesions were dilated per pt. Side-effects (diarrhea, dyspepsia) resulted in a 6.6% drop-out rate in colchicine pts. Complete quantitative angiographic follow-up was obtained in 145 pts (74%). Quantitative measurements were obtained in 2 orthogonal views on the 393 lesions dilated in the 145 pts with baseline (pre-PTCA) immediate post-PTCA (post-PTCA) and 6 months follow-up (6 mos) angiographic films.

	Quantitative Luminal Diameter Stenosis			
	Pre-PTCA	Post-PTCA	6 mos	
placebo (152 lesions)	67%	24%	47%	p=NS
colchicine (241 lesions)	67%	24%	46%	

The lesion restenosis rate (defined as a return to ≥70% stenosis and loss of ≥50% of the initial gain) was 22% in placebo pts and 22% in colchicine pts (p=NS). The patient restenosis rates were also not different in the 2 groups **CONCLUSION:** Colchicine is ineffective for the prevention of restenosis after PTCA.

5:00

EFFECTS OF DELAYED ANGIOPEPTIN TREATMENT ON MYOINTIMAL HYPERPLASIA FOLLOWING ANGIOPLASTY

Marcus Howell, Robert Trowbridge, Marie Foegh, Georgetown University Medical Center, Washington D.C., U.S.A.

The synthetic octapeptide Angiopeptin (AP) has been shown to significantly inhibit myointimal hyperplasia. The precise mechanism of AP is not known nor is the initiating factors causing myointimal hyperplasia. This study investigates the effect of delaying AP administration. Angioplasty was performed in the aorta, the common and external iliac arteries of 20 New Zealand White rabbits with a Fogarty embolectomy catheter. The rabbits were randomized into 5 groups of 4 animals each. The control group received saline while the remaining 4 groups received AP 10 ug/kg bid s.c. until sacrifice 22-24 days following angioplasty. One of the treatment groups received the first dose during angioplasty (0 h) while the remaining 3 groups received their first dose 8, 18, and 27 h following angioplasty, respectively. Intimal hyperplasia was determined by morphological analysis of elastin stained sections and expressed as area of intimal hyperplasia / total vessel area x 100%.

	Control	0 h	8 h	18 h	27 h
Aorta	9.68	2.81*	6.48	8.56	12.34
Common Iliac	17.31	6.97*	11.98	14.46	22.26
External Iliac	24.06	10.67*	15.21	20.68	28.28

*P≤0.01

AP significantly inhibits myointimal hyperplasia only when given prior or during angioplasty. Thus Angiopeptin works on early mechanisms that cause restenosis following balloon angioplasty, and these early mechanisms initiate the process leading to intimal hyperplasia.

4:45

PARADOXICAL INCREASE IN RESTENOSIS RATE WITH CHRONIC HEPARIN USE: *FINAL RESULTS OF A RANDOMIZED TRIAL*

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We hypothesized that long-term administration of heparin may decrease restenosis after coronary angioplasty by its favorable effects on thrombosis, smooth muscle proliferation, and lipid metabolism. A prospective, controlled trial was therefore initiated, with patients randomized to receive either 10,000 units subcutaneous heparin daily, or usual care. Of the 23 patients who completed the protocol at the time of study termination, 14 out of 17 receiving heparin experienced restenosis documented by quantitative angiographic analysis (82% *vs* 33% for controls, p<0.05). Restenosis also appeared higher when examined on a per-lesion basis (60% *vs* 22%, p=0.06). Mean loss of initial diameter improvement was 53 ± 16% [SE] *vs* -20 ± 46% for controls. Secondary endpoints were also higher in the heparin group, with 13 patients with recurrent angina (76% *vs* 17%), 3 with MI (18% *vs* 0%), and 1 with cardiac death (6% *vs* 0%). Complications associated with the study were limited to the heparin group, with 7 patients experiencing abnormal bleeding (41% *vs* 0%), leading to surgery in 1 individual. The study was terminated prior to achievement of targeted enrollment on ethical grounds.

Thus, chronic heparin use after successful coronary angioplasty paradoxically appears to increase the likelihood of both angiographic restenosis and adverse clinical outcome.

5:15

PREVENTION OF RESTENOSIS AFTER PTCA BY ANGIOTENSIN CONVERTING ENZYME INHIBITORS

Neil B. Gottlieb, Ronald S. Gottlieb, Joel Morganroth and Frank V. Brozovich. Graduate Hospital, Philadelphia, PA.

To determine whether angiotensin converting enzyme (ACE) inhibitors reduce the occurrence of restenosis after percutaneous transluminal coronary angioplasty (PTCA), we retrospectively analyzed records from June, 1988, to December, 1989, of 322 successfully angioplastied patients. No patients developed chest pain, elevation of ST segments on EKGs, positive cardiac enzymes or other evidence of abrupt vessel closure following the PTCA. After PTCA, all patients received IV heparin for 12-24h, until sheath removal, bolus heparin for an additional 24-48h and were discharged on an aspirin per day. Patients were separated into two groups; those at hospital discharge incidentally treated for hypertension or heart failure with ACE inhibitors (n=36), and those treated with a drug regimen which did not include ACE inhibitors (n=286). The two groups were similar with respect to age (61±13.5 vs 60±12.5, p>0.05) and other demographic characteristics. Restenosis, defined as the presentation to a physician with symptoms of angina within 6 months of the PTCA and the finding on repeat catherization of a significant stenosis at the site of the PTCA, occurred in 30% of the patient who were discharged on a drug regimen which did not include ACE inhibitors. For the patients treated with an ACE inhibitor, the incidence of restenosis was 3%. Using Chi squared analysis, for the patients treated with an ACE inhibitor the occurrence of restenosis was significantly reduced (p<0.05). Thus, it appears that inhibition of angiotensin converting enzyme may significantly reduce the incidence of restenosis after successful PTCA.