ABSTRACTS – POSTER

990 Therapy of Unstable Angina

Wednesday, March 27, 1996, 9:00 a.m.–11:00 a.m.
Orange County Convention Center, Hall E
Presentation Hour: 9:00 a.m.–10:00 a.m.

990-37 Failure of Present Clinical Algorithms of Intravenous Heparin Adjustment to Maintain Stable Therapeutic Anticoagulation in a Coronary Care Unit Setting

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Intravenous heparin anticoagulation in the coronary care unit is very common. Although a wide variety of anticoagulation algorithms, with and without weight adjustment, have been described, their efficacy is in question. To determine the effectiveness of presently existing clinical heparinization protocols, we studied the results of 3,745 consecutive patients (mean age: 64 yrs, males: 72%, average weight: 82 kg, admitted with acute MI: 63%) receiving IV heparin in the LDS Hospital CCU. Time from initiation of heparin to the first therapeutic PTT (defined as 60–90 sec) (TFTPTT), % of PTT's < 60, % of PTT's > 90, and the total % of PTT's outside the therapeutic range (%OTR) were evaluated for the entire group and for subsets of patients in which the initial weight adjusted dosing was within three ranges of < 12 U/kg/hr, 12–17 U/kg/hr, and > 17 U/kg/hr. Results are:

Initial Heparin Dose	TFTPTT	%PTT's < 60	%PTT's > 90	%OTR
< 12 U/kg/hr (n = 1348)	10.6 ± 11.8hr	48 ± 28%	21 ± 23%	69 ± 24%
12–17 U/kg/hr (n = 2060)	7.8 ± 9.0hr	35 ± 24%	31 ± 25%	66 ± 24%
> 17 U/kg/hr (n = 337)	7.1 ± 7.5hr	31 ± 24%	36 ± 26%	67 ± 22%
Entire Group (n = 3745)	9.5 ± 9.9hr	39 ± 26%	28 ± 24%	67 ± 24%

Conclusion: Two thirds or more of PTT's obtained in the CCU in all groups of patients on IV heparin were found to be out of range. Although patients treated in the more aggressive weight adjustment ranges tended to reach a therapeutic PTT sooner than those treated in the lower range, this difference is likely not large enough to show clinical relevance. This study underscores the difficulty of maintaining stable adequate anticoagulation in clinical practice using IV heparin, regardless of whether weight adjustment is used, and the need to evaluate alternative more stable anticoagulation strategies.

990-38 Treatment of Intracoronary Thrombus: Successful Lysis With Intra-Coronary t-PA

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In order to assess the efficacy of intracoronary (IC) t-PA we reviewed in a core laboratory the coronary angiograms at baseline, 20, 40, 60 minutes (min) and 24 hours after treatment of 120 patients (pts) with acute coronary syndromes and IC thrombus. Thrombus was scored as none (0), possible (1) and definite (2–4) with discrete intra-luminal filling defects of increasing size (small < 1/2, moderate ≥ 1/2 + < 2, and large ≥ 2 vessel diameters respectively). Five IC t-PA infusion strategies were assessed (20 mg bolus, 20 mg over 20 min, 20 mg over 40 min, 40 mg over 20 min and 40 mg over 40 min). For the entire cohort, percent stenosis (84 ± 15 baseline vs 75 ± 20 at 24 hr) and TIMI flow (58% with grade 3 baseline vs 69% at 24 hr) improved with therapy.

Angiographic Outcome	Baseline	60 min	24 hr
No clot (%)	7	15	26
Definite clot (%)	79	63	47
Large clot (%)	33	21	11

Procedural and angiographic variables were analyzed to predict successful lysis. A decrease of ≥ 2 thrombus scores at 24 hr was noted in 55% of pts with TIMI 3 flow at baseline compared to 33% of pts with TIMI 0–2 flow (p = 0.05). A decrease of ≥ 2 thrombus scores at 24 hr was noted in 56% of pts treated with high dose (40 mg) t-PA compared to 40% of pts treated with low dose (20 mg) t-PA (p = 0.14). We conclude that IC t-PA relieves coronary narrowing, improves flow, successfully lyses IC clot at low dose with some added benefit from high dose and that its effectiveness is dependent on pre-treatment flow.

990-39 Regression of Culprit Coronary Lesions Demonstrated by Repeat Quantitative Coronary Angiography Within Three Months After Thrombolysis for Acute Myocardial Infarction

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Little is known about the natural history of coronary lesions after successful thrombolysis (T) for acute myocardial infarction (MI). In the APRICOT-study we performed coronary angiography within 48 hours after T and patients (pts) with a patent infarct-related artery underwent a second angiography after 3 months. Initial visual assessment showed high-grade stenosis to be an independent predictor of reocclusion. We also found that 15% of complex lesions showed remodelling to smooth and less severe lesions. Quantitative coronary angiography (QCA) was performed to obtain accurate data concerning reocclusion and regression of culprit lesions.

In 230 of 248 pts the angiograms were suitable for QCA. In this group 66 pts showed reocclusion. The mean diameter stenosis of the culprit lesions in pts with reocclusion was 61.4 ± 10.7% compared to 54.4 ± 11.7% in pts without reocclusion (p < 0.0001). The table shows data of 161 pts without reocclusion. Three subgroups were studied according to changes in morphology. (Only three lesions changed from smooth to complex and were not considered in this analysis).

Morphol. change	Mean diameter stenosis (%)		p
	Angio 1	Anglo 2	
CO → CO (N = 41)	55.8 ± 11.2	50.5 ± 10.9	0.002
CO → SM (N = 38)	52.6 ± 12.1	40.3 ± 16.3	0.00001
SM → SM (N = 82)	54.4 ± 12.0	51.2 ± 14.0	0.01

SM = smooth; CO = complex

Conclusion: QCA confirms that stenosis severity measured early after successful T for acute MI is associated with reocclusion. In pts without reocclusion reduction in stenosis severity occurs in most lesions. However, the largest reduction is seen in lesions changing from complex to smooth, implicating that these lesions are especially subject to ongoing thrombus resolution and plaque repair.

990-40 Identification of High-Risk Patients With Unstable Angina

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Early revascularization has been proposed in patients with unstable angina (UA) who have in-hospital recurrence of symptomatic ischemia despite bed rest and aggressive medical therapy. In our ward, mean waiting times for revascularization of over 2 weeks allowed for assessment of: 1) the risk in delaying revascularization for more than 72 to 96 hrs, and 2) whether patient risk for recurrent ischemia could be defined. We graded recurrent angina as *refractory* (Group 1) when pts had ≥ one ischemic episode/day over consecutive 48 hours and ≥ five episodes total, and as *occasional* (Group 2) when pts had ≥ one ischemic episode without the previous features. Of 335 consecutive pts with UA who were admitted to our ward, 109 (32.6%) had recurrent ischemia (33 pts in Group 1, 76 in Group 2). Significant differences (p < 0.05) between pts in Groups 1 and 2 were found for females (49% vs 29%), insulin-dependent diabetes (9% vs 1%), triple/left main coronary disease (43% vs 23%), mean ejection fraction (57 ± 17% vs 64 ± 12%), and episodes of ischemia: lasting more than 20 min (94% vs 40%), with arrythmias (27% vs 11%) or requiring inotropic drug (18% vs 4%). The waiting times for revascularization were similar (22 ± 13 vs 17 ± 7 days). In Group 1, 11 of 17 pts treated medically were angina-free after 16 ± 8 days, but six (18.1%) had intercurrent events (two deaths within 96 hrs despite rescue revascularization); the remaining 16 pts underwent revascularization safely. In Group 2, 62 of 67 pts treated medically had resolution of ischemia after 5 ± 5 days, but five patients (6.6%) had events (two within 96 hrs of admission); the 10 remaining pts had successful revascularization. Conclusions: 1) patients with recurrent ischemia after 48 hrs of medical therapy should be referred for immediate angiography; 2) delaying revascularization in these patients more than 96 hrs increases the risk of cardiac events; 3) refractory UA may be defined by the persistence of ischemic episodes despite 4 to 7 days of medical therapy.