ABSTRACTS – POSTER

1033 Anticoagulant and Antiplatelet Therapy on Unstable Angina

Wednesday, March 19, 1997, 9:00 a.m.–11:00 a.m.
Anaheim Convention Center, Hall E
Presentation Hour: 9:00 a.m.–10:00 a.m.

1033-31 Delaying and Preventing Ischemic Events in Patients with Acute Coronary Syndromes Using the Platelet Glycoprotein IIb/IIIa Inhibitor Lamifiban

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Potent antiplatelet therapy in patients with acute coronary syndromes (ACS) may effectively attenuate ischemic events. PARAGON A randomized 2282 patients with unstable angina/non-Q MI to treatment with low or high dose lamifiban (LL and HL) with or without heparin (H) or standard therapy (ST), heparin alone. Incidence and timing (after study drug termination) of 30-day (re)infarction (RI) and in-hospital refractory ischemia (ISC) are displayed:

	LL, H (n = 377)	LL, noH (n = 378)	HL, H (n = 373)	HL, noH (n = 396)	H (n = 758)
RI	7.7%	7.7%	9.9%	8.1%	8.2%
ISC	7.5%	7.7%	8%	9.3	9.1%
RI	40 hrs	59 hrs	68 hrs	68 hrs	53 hrs
Timing	12,90	24,180	33,180	17,255	28,236
ISC	90 hrs	93 hrs	175 hrs	97 hrs	65 hrs
Timing	22,322	20,208	49,316	43,316	20,326

LL with and without H has a favorable effect on delaying and preventing ischemic events in ACS patients. The effects on death and (re)MI will require a larger definitive trial.

1033-32 Accelerated *Primary* Platelet Aggregation in Acute Myocardial Infarction: Use of a New Aggregometry Employing Laser-Light Scattering System

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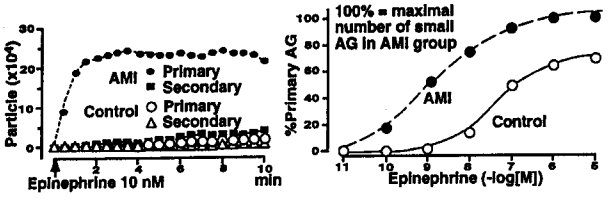
Background: Platelet aggregation (AG) plays an important role in the pathogenesis of acute myocardial infarction (AMI). Since the conventional aggregometry cannot identify small aggregates, there have been considerable limitations in detecting the early phase of AG. Aggregometry AG-10, which uses laser-light scattering methods, is capable of identifying the temporal changes of both the size and number of aggregates. Using this new aggregometry, we evaluated the primary and secondary AG processes in AMI. *Methods:* Peripheral blood samples were obtained from 15 AMI patients (pts) within 3 hrs from onset, and were immediately assayed for platelet AG using AG-10 (no pts had received either anticoagulant or antiplatelet regimens prior to sampling). Blood samples from 10 healthy volunteers were used as controls. *Results:* Within the first 5 mins of AG, a marked increase in the number of small aggregates suggested an accelerated primary AG in AMI. In the following 5 mins, a modest increase in the number of large aggregates suggested the formation of the large aggregates (i.e. the secondary AG). As compared to controls, AMI pts had increases in the responses of primary AG to both epinephrine and ADP that were up to 100 times higher (ED_{50} of epinephrine: 0.9 vs. 102 nM, $p < 0.001$; ED_{50} of ADP: 14 vs. 560 nM, $p < 0.01$).



Conclusions: These data document, for the first time, systemically enhanced primary AG in pts with AMI and may provide new insights into understanding of the pathogenesis of AMI.

1033-33 Low Molecular Weight Heparin vs Regular Heparin in the Treatment of Patients with Unstable Angina

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The role of regular heparin [RH] as an important agent in therapy of unstable angina [UA] is well documented but the efficacy of low molecular weight heparin [LMWH] in these patients is still unclear. The aim of our study was to assess results of LMWH therapy in comparison to RH treatment in patients [pts] with UA. Study population consisted of 176 pts with UA defined as episodes of chest pain lasting < 20 min and/or with ST changes during 12 hours before start of treatment. Pts were divided into two groups [GR]: GR I – 74 pts received LMWH (Fraxiparine) in dose 250/kg/day/i.v.; GR II – 102 pts received RH in APTT adjusted. Pts were also treated with nitrates, aspirin, beta-blockers and/or calcium channel. Baseline clinical data of groups were comparable. During hospitalisation end points: death, myocardial infarction [MI] were assessed. Also frequency of: positive results of treatment [PRT], emergency angiography [EA] and emergency PTCA or CABG [EPT/CABG] and bleeding complication [BC] during therapy were estimated. As PRT considered stabilisation of symptoms and ecg without CK elevation during 48 hrs after start of therapy. *Results:*

	Group I-LMWH	Group II-RH	p
Death [% of pts]	1.4	1.9	NS
MI [% of pts]	13.5	17.5	< 0.01
PRT [% of pts]	74.3	72.7	NS
EA [% of pts]	20.2	24.5	< 0.01
EPT/CABG [% of pts]	13.5	15.6	NS
BC [% of pts]	1 .2	4.9	< 0.01

Conclusions: LMWH seems to be useful in the therapy pts with UA. In pts receiving LMWH the ratio of subsequent myocardial infarction and urgent coronary angiography was lower and bleeding complications were less frequent.

1033-34 Relationship of Outcomes to Treatment with Lamifiban in Patients Undergoing PTCA: Analysis of PARAGON A

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Previous studies of GP IIb/IIIa inhibitors in patients with acute coronary syndromes undergoing PTCA demonstrated a reduction in death and MI. In PARAGON A 306 patients (13.6%) underwent PTCA. We assessed the relationship between death/MI and a safety composite (SFCOMP = stroke/≥ moderate bleeding/thrombocytopenia) and treatment with the non-peptide GP IIb/IIIa blocker, lamifiban at high (HL) or low (LL) dose with or without heparin (H), or standard therapy (ST = heparin and ASA). Results are shown below.

	LL,H (n = 373)	LL,–H) (n = 375)	HL,H (n = 370)	HL,–H (n = 390)	ST (n = 749)
PTCA	11.8%	13.6%	11.4%	12.6%	16.0%
Time (h)*	48.1	41.5	48.8	67.8	35.0
Death/MI	6.8%	11.8%	16.7%	14.3%	15.8%
SFCOMP	9.5%	4.1%	14.3%	12.8%	8.8%

*Time = median time from end of infusion to PTCA

Conclusion: Treatment with lamifiban may delay and reduce the use of PTCA in patients with unstable angina/nonQ MI. Outcomes in patients undergoing PTCA who receive potent platelet inhibition compare favorably to those receiving standard therapy. Use of HL confers no additional benefit over LL but carries additional risk.