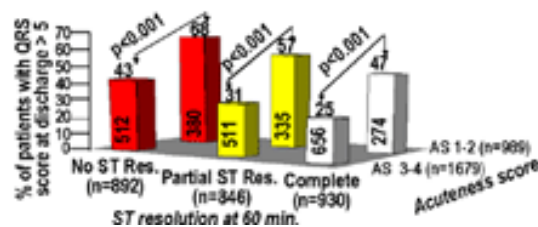


Results: At 60 min, 850/2192 pts with AS ≥ 3 had an 11% greater absolute frequency of complete STR (>70%) than 344/1239 with low AS (<3) $p < 0.001$. Patients surviving to discharge (fig.), showed that within each subgroup of STR, patients with high AS were statistically more likely to have a small QRS infarct at discharge as compared to those with low AS $p < 0.001$. Moreover in a multivariate logistic regression model including baseline characteristics, Σ ST, STR, time to treatment, the AS ≥ 3 proved the strongest predictor of small infarct size (OR=0.374, CI= 0.315-0.445).

Conclusion: Even after adjusting for time to treatment the AS provides novel insight into the likelihood of STR 60 min after fibrinolysis and the probability of a smaller infarct size regardless of the extent of STR.



1117-90

Establishing a New Therapeutic Activated Partial Thromboplastin Time Range for Unfractionated Heparin for Acute Coronary Syndromes

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Introduction: The therapeutic aPTT range of 50 to 70 sec recommended by the ACC/AHA for patients receiving unfractionated heparin (UFH) for acute coronary syndromes (ACS) is vulnerable to variation in test reagents. Rather than use the standard therapeutic aPTT range, it has been recommended that each institution establish its own therapeutic aPTT range based upon anti-factor Xa levels. We evaluated our institution's therapeutic aPTT range by examining the correlation between aPTTs and anti-factor Xa levels and established a new therapeutic aPTT range with a new thromboplastin reagent based upon the therapeutic anti-factor Xa levels.

Methods: 62 plasma samples were collected from 26 consecutive patients receiving UFH for ACS. aPTTs measured with a thromboplastin reagent and a new thromboplastin reagent and anti-factor Xa levels were obtained on each plasma sample. Linear regression analysis was performed to establish a new therapeutic aPTT range from corresponding therapeutic anti-factor Xa levels.

Results: 32% of patients with target range aPTTs had anti-factor Xa levels below the accepted level of 0.35-0.7 U/mL for ACS while 68% had therapeutic anti-factor Xa levels. When the same blood was tested with a new thromboplastin reagent, only 9% of patients with target range aPTTs had anti-factor Xa levels <0.35 U/mL while 91% had therapeutic anti-factor Xa levels. The Pearson correlation coefficient (r) for the new thromboplastin reagent was 0.93. The slope of the regression line was 221.3. The therapeutic aPTT range established with the new thromboplastin reagent was 61-100 sec.

Conclusion: Monitoring aPTTs without standardizing the thromboplastin reagent may not adequately reflect therapeutic heparin levels. Despite apparently therapeutic aPTTs, patients treated with UFH may be under-anticoagulated. Our new anti-Xa-adjusted therapeutic aPTT range shows an increase in the positive predictive value of aPTTs. Large-scale clinical studies are needed to determine the optimal anti-factor Xa range for ACS patients treated with UFH, with further refinements if GP IIb/IIIa antagonists are concomitantly used and to show a clinical benefit for monitoring heparin levels with anti-factor Xa levels.

1117-91

Invasive Therapy Along With Glycoprotein IIb/IIIa Inhibitors and Intracoronary Stents Improves Survival in Non-ST-Segment Elevation Acute Coronary Syndromes

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Background: Although several clinical trials have shown that early invasive therapy in UA/NSTEMI patients reduces the risk of composite endpoints inclusive of death, MI or angina, it is unclear whether such an approach improves survival.

Methods: We conducted a meta-analysis on five studies in a total of 6,766 UA/NSTEMI patients who were randomized to either routine invasive (n=3,371) versus conservative therapy (n=3,395) in the era of GP IIb/IIIa inhibitors and intra-coronary stents.

Results: As compared with conservative therapy, an invasive approach was associated with a reduction in the risk of all-cause mortality at 6-12 months (3.3% vs. 4.2%, relative risk [RR] 0.80, 95% confidence interval [CI] 0.63 to 1.03) and at 24 months (5.0% vs. 6.5%, RR 0.77, 95% CI 0.60 to 0.99) of follow-up. Although invasive therapy reduced the risk of the composite endpoint of death or MI at 6-12 months in men (RR 0.68, 95% CI 0.57 to 0.81) and troponin positive patients (RR 0.74, 95% CI 0.59 to 0.94), the results for women (RR=1.07, 0.82 to 1.41) and troponin negative patients (RR=0.82, 0.59 to 1.14) were equivocal.

Conclusions: Invasive therapy in UA/NSTEMI patients with adjunctive use of GP IIb/IIIa inhibitors and intra-coronary stents improves survival. Enhanced risk stratification is needed in women and troponin negative patients so that invasive therapy may be more effectively recommended in these groups.

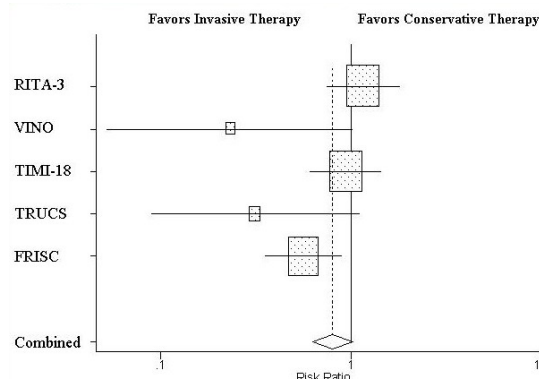


Figure 1. Forest plot of death at 6-12 months for invasive versus conservative strategies. Summary estimate RR = 0.80 (95% CI; 0.63, 1.03).

POSTER SESSION

1118

New Observations From Acute Myocardial Intervention Trials III

Tuesday, March 09, 2004, 9:00 a.m.-11:00 a.m.
Morial Convention Center, Hall G
Presentation Hour: 9:00 a.m.-10:00 a.m.

1118-77

Effects of Carvedilol Compared to Atenolol on Ejection Fraction and Clinical Endpoints After Myocardial Infarction

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Background: Beta-blockers have been found to reduce mortality and morbidity in post-myocardial infarction (MI) patients. However, it is not fully understood, whether all beta-blockers have similar favourable cardiovascular effects. The aim of this study was to compare the effects of carvedilol and atenolol on global and regional ejection fraction (EF), and on predefined cardiovascular endpoints.

Methods: In a randomized, prospective, open and endpoint-blinded single center study, 118 patients received carvedilol (mean dosage 36.2 ± 15.1 mg) and 114 patients atenolol (mean dosage 72.1 ± 30.6 mg). No difference in baseline data, such as age, gender, race, smoking, hypertension, diabetes mellitus or lipid lowering treatment, was observed. There was no difference in baseline characteristics regarding infarct localization, reperfusion therapy and acute treatment between the groups. Treatment with aspirin (96%), statin (98%), and ACE-inhibitors was equal in the 2 groups. The global and regional left ventricular (LV) EF were evaluated with gated blood pool scintigraphy.

Results: In the carvedilol group 90 cardiovascular endpoints were observed compared to 104 endpoints in the atenolol group, with a hazard ratio (RR) 0.84 with 95% confidence interval (0.74-0.94) (p=0.002). This occurred despite similar global LVEF was 55 ± 11% at baseline, 57 ± 10% at 3 months and 57 ± 10% at 12 months in the carvedilol group and 53 ± 9%, 56 ± 10% and 56 ± 9% in the atenolol group respectively. No difference in regional EF was observed in the two groups. The main contributor to the difference in cardiovascular endpoints was a composite of cardiovascular deaths, non-fatal MI, and congestive heart failure (worsening and hospitalization).

Conclusion: In patients following an acute MI, carvedilol treatment compared to atenolol was associated with a significant reduction in combined cardiovascular endpoints despite that no difference in global or regional ejection fractions was found. Thus other mechanisms than remodelling might explain this difference in clinical effect between the two drugs.

1118-78

Primary Angioplasty Versus Facilitated Intervention (Tenecteplase Plus Stenting) in Patients With ST Elevated Acute Myocardial Infarction: Final Results of the GRACIA-2 Trial

Francisco Fernandez-Aviles, Joaquin J Alonso, Alfonso Castro-Beiras, Javier Goicolea, Jesus Blanco, Juan Alonso, Juan Lopez-Mesa, Luis Diaz-Liera, Nicolas Vazquez, Rosa Hernandez, Armando Perez, Javier Moreu, The GRACIA-2 Investigators, ICICOR, Hospital Clinico Valladolid, Valladolid, Spain

Background: Combined reperfusion therapies widely applicable could benefit the still high proportion of patients (pts) with ST-elevated acute myocardial infarction (STEMI) for whom primary PCI is not available due to logistic reasons.

Methods: We compared the evolution of 212 pts with STEMI (<12 hs from onset) randomised to 2 strategies: 1) primary PCI optimally performed (stenting of culprit artery under protection of abciximab within 3 hs from onset); or 2) a combined (facilitated) strategy: immediate thrombolysis (T) with tenecteplase (full bolus dose adjusted by weight)