POSTER SESSION

1173   Unstable Angina: Prognostic Determinants

Tuesday, April 01, 2003, Noon-2:00 p.m.
McCormick Place, Hall A
Presentation Hour: Noon-1:00 p.m.

1173-89   *Helicobacter Pylori* Eradication Improves Prognosis of Coronary Artery Disease Through a Mechanism Not Related With Platelet Activation

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Background: Epidemiological association between *Helicobacter pylori* (HP) infection and coronary artery disease (CAD) has been suggested, but underlying pathophysiological links remain unknown. Platelet activation and aggregation have been reported in animal models. This pilot randomized controlled trial evaluates the impact of HP infection and eradication on platelet activation and inflammatory markers in patients (pts) with CAD as well as the influence of HP status on subsequent adverse coronary events (cardiac death, angina or myocardial infarction).
Methods: Ninety survivors after an acute coronary event not requiring surgical revascularization were included. HP status was assessed with an urea breath test and fibrinogen, platelet surface expression of CD62P, CD63 and CD41/61 (flow cytometry), soluble P selectin (ELISA), high-sensitivity C reactive protein, and *C.pneumoniae* serology were measured at baseline and at two-months follow-up. HP positive pts were randomized to receive a 7-day course of omeprazole, amoxycillin and metronidazole or corresponding placebos (randomization ratio 3:2). Pts were followed-up for one year or until death or readmission with an acute coronary syndrome.
Results: No baseline differences were observed between HP positive (n = 49) and HP negative (n = 41) pts. Among HP positive pts, 18 received placebo and 31 active medication with HP eradication in 21 cases. No differences were observed neither in inflammatory or platelet activation markers nor in anti-Chlamydial serology between pts with persistent or resolved HP infection. However at 6 and 12 months, a recurrent coronary event was observed in 35% and 55% of pts with persisting HP infection vs 10% and 25% of pts in whom HP was either absent or eradicated respectively (log-rank ≈ 0.01, Breslow = 0.01). Only final HP status (RR 2.7 [95% CI 1.2-6.3]; p=0.014) and dyslipemia (RR 3.0 [95% CI 1.0-9.0]; p=0.046) were selected as independent predictors of recurrent coronary events. **Conclusions**: HP infection does not induce significant platelet activation in pts with CAD. HP infected pts have an increased probability to suffer a recurrent coronary event which may be reverted by eradicating the infection.

1173-90   Baseline Creatinine Clearance Provides Additional Prognostic Information to TIMI Risk Score in Non-ST Elevation Acute Coronary Syndromes

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Background: The TIMI risk score is a simple tool that is established for risk stratification and therapeutic decision making in patients (pts) with non-ST-elevation acute coronary syndromes (NSTE-ACS). We investigated whether baseline renal function provides prognostic information in addition to this score.
Methods: We studied 433 pts admitted due to NSTE-ACS. Creatinine clearance (CrCl) was determined using the Cockroft-Gault equation (using baseline Cr level). Study endpoint was death or nonfatal myocardial infarction (MI) by 30 days follow-up. ROC curve analysis was used to determine the best discriminatory value for CrCl. Additionally, survival status at 1 year follow-up was determined for all pts.
Results: TIMI risk score was >3 in 257 (59.4%) pts, and CrCl was <55 ml/min in 126 (29.1%). In multivariable analyses, both TIMI risk score >3 (OR 4.7; 95% CI, 1.4-16.2) and CrCl <55 ml/min (OR 3.6; 95% CI, 1.5-8.2) independently predicted 30-day prognosis. Similarly, both TIMI risk score >3 (OR 4.0; 95% CI, 1.1-13.7) and CrCl <55 ml/min (OR 4.2; 95% CI, 1.7-10.3) were independent predictors of 1-year mortality. The combined use of TIMI risk score and baseline CrCl identified pts at low, intermediate and high risk for both the 30-day endpoint and 1-year mortality (see table).
Conclusions: In pts with NSTE-ACS, baseline renal function provides short and long term prognostic information, additional to the TIMI risk score. The combined use of these risk indicators may help guide more cost-effective treatment.

Endpoint	TIMI ≤ 3 and CrCl ≥ 55 (n=136)	TIMI> 3 or CrCl< 55 (n=211)	TIMI> 3 and CrCl< 55 (n=86)
Death/MI: 30 days	1.5%	4.3%	16.3%
Death: 1 year	1.5%	3.3%	15.1%

1173-91   Elevated Titre of Antihuman Heat Shock Protein 60 Predicts an Adverse Medium-Term Prognosis in Patients With Unstable Angina

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Background There is evidence that the inflammatory state of coronary atherosclerosis is important in determining plaque stability. Increased CRP levels have prognostic implications and interest has turned to other aspects of inflammation including antibodies to heat shock proteins expressed within plaques. Their prognostic importance in coronary disease has not been addressed. **Methods** Consecutive emergency admissions with acute chest pain of suspected cardiac origin but without ST elevation or significant CK-MB elevation were eligible for inclusion. After discharge, they were followed-up by review of their hospital case records and directly by telephone and letter. The clinical endpoints were CHD death, non-fatal MI, CABG, PTCA, angiogram and readmission with further cardiac ischaemic chest pain. Anti-human heat shock protein 60 (anti-huhsp 60) and anti-mycobacterial heat shock protein 65 (anti-myhsp65) titres were assayed on samples drawn on the morning after admission. **Results** A total of 588 patients were enrolled from a single centre. During follow-up (mean of 304 days, range 1 to 788 days), 277 patients had an endpoint. Table 1 details hazard ratios for anti-huhspP60 and anti-myhsp65 in quartiles for the combined endpoint conditional on age, smoking, log(CRP), hypertension and diabetes in a multivariate Cox model. **Conclusions** Patients admitted with acute cardiac chest pain and elevated levels of anti-huhsp 60 had an adverse medium term prognosis. Anti-myhsp65 titres were not predictive.

Table 1 Hazard ratios for anti-huhspP60 and anti-myhsp65 in quartiles for the combined endpoint

Anti-huhsp 60 titre (Au/ml)	Hazard Ratio	P Value	Anti-myhsp 65 titre (AU/ml)	Hazard Ratio	P Value
<16	1.0		<9	1.0	
16 to <24	1.65	0.005	9 to < 17	1.02	0.89
24 to <36	1.41	0.066	17 to <31	0.75	0.12
>36	1.55	0.016	>31	0.94	0.75
		OVERALL P=0.031			OVERALL P=0.33

1173-114   Outcomes Based on Time to Angiography in Patients With Non-ST Elevation Acute Coronary Syndromes

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Background: Recent clinical trials and guidelines support invasive risk stratification with early coronary angiography and revascularization for patients with Non-ST-elevation acute coronary syndromes (ACS). The optimal timing of angiography from hospital admission however is uncertain;therefore,we retrospectively investigated whether timing of angiography affects outcomes in patients with Non-ST-elevation ACS.
Methods: Study population consisted of 836 patients admitted to the coronary care unit between 1997- 2000 with Non-ST-elevation ACS who underwent angiography during the same hospital stay. Patients were categorized into three groups based on the time interval between admission and angiography: <24 hours, 24-48hours, and >48hours. Six-month incidence of death and/or myocardial infarction was determined and compared between groups.
Results: Table below summarizes baseline characteristics and 6-month outcomes according to angiography group. Following multivariate adjustment, angiography group remained signifantly associated with survival with angiography delays >48hours associated with worse survival.
Conclusions: Younger age, male gender, absence of prior MI were associated with increased likelihood of angiography within 48 hours of hospital admission. Delay of angiography >48 hours was associated with worse survival while angiography delay 24-48 hours yielded optimal survival results.

Baseline Characteristics and Outcomes

Variable	<24hrs (N=314)	24-48hrs (N=267)	>48hrs (N=255)	p-value
Age (s.d.)	62.4 (12.4)	62.6 (13)	65.1 (11.6)	0.02
Female (%)	37.3	39	50.6	0.003
Prior MI (%)	25.8	25.1	38.4	< 0.001
Index PCI (%)	56.7	47.20	41.6	0.001
MI,(%)	4.5	6.7	7.8	0.22
Death or MI, (%)	10.2	11.2	16.5	0.067
Death, (%)	7.4	5.6	12.3	0.03