B-Type Natriuretic Peptide Levels on Admission Predict Short-Term Mortality and Angiographic Success of Procedure in Patients With Acute ST Elevation Myocardial Infarction Treated With Primary Angioplasty

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Background: B-type natriuretic peptide (BNP) levels in the first days after the onset of symptoms are predictive of short-term mortality in patients with acute coronary syndromes. Few data are available for BNP levels obtained on admission in patients (pts) with acute ST elevation myocardial infarction (STEMI). Methods: Blood samples for BNP determination were obtained on admission in 117 pts (mean age 58.4±10.7 years old) with STEMI. In a 15-minute period, BNP was measured by using simple bedside test for rapid quantification of BNP, before primary percutaneous coronary intervention (PCI). 30 days follow-up was performed. PCI was performed in all (100%) pts. Results: Mean for BNP was 171.8±218.2 pg/ml. Baseline level of BNP was higher among pts who died than among those who were alive at 30 days (median, 541.9±247.2 pg/ml vs. 140.9±185.9 pg/ml; p<0.001). Baseline BNP in subgroups by median level showed a significant increase in mortality: 1 (1%) in inframedian group (IMG) vs. 8 (13%) in supramedian group (SMG) (p<0.05). Baseline level of BNP in subgroups by Killip class on admission was higher among pts who died than among those who were alive at 30 days (Killip class I: median, 475.8±280.6 pg/ml vs. 123.6±138.1 pg/ml; p<0.05; Killip class II-IV: 257.1±362.5 pg/ml vs. 624.5±203.9 pg/ml, p<0.01). After adjustment for independent predictors of risk of death, the odds ratio for death at 30 days in SMG was 13.6 (95% confidence interval, 1.1 to 182.7). There was no difference in subgroups by median BNP in TIMI 3 flow grade before PCI (7% vs. 7%; p=NS). TIMI 3 after PCI was more often seen in pts in IMG vs. SMG (80% vs. 72%; p=0.01). BNP was higher among pts with TIMI 0, 1 or 2 after PCI than among pts with TIMI 3 after PCI (328.3±323.8 pg/ml vs. 151.3±196.7 pg/ml; p<0.01). BNP remained independent predictor for TIMI 0, 1 or 2 after PCI (odds ratio in SMG was 3.5 (95% confidence interval 1.2 to 10.8). Conclusion: BNP levels obtained on admission are powerful, independent indicator of short-term mortality and angiographic success after PCI in pts with STEMI. Rapid tests for BNP assay seem to be new tool in risk stratification of pts with STEMI.

Results: At baseline, expression of 5-lipoxygenase, MCP-1 and IL-6 was higher in chronic ischemic myocardium than in normally perfused myocardium. In response to acute ischemia and reperfusion in the normal myocardium, there was a clear trend towards increased expression of pro-inflammatory factors (e.g. 5-lipoxygenase, COX-2, CysLT2 receptor, MCP-1, IL-6). These responses were less pronounced or detectably absent in the chronic ischemic myocardium.

Conclusion: These findings suggest a discrete increase in several pro-inflammatory factors that may contribute to myocardial pathophysiology in chronic myocardial ischemia. The blunted inflammatory response to acute ischemia in the chronic ischemic myocardium may suggest altered sensitivity in signaling pathways mediating the inflammatory response.

5:15 p.m.

Hyponatremia: A Useful Marker for Early Risk Assessment in Acute ST Elevation Myocardial Infarction

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Background: Hyponatremia (HNa) is common in hospitalized patients and is associated with adverse prognosis, especially in heart failure. Data on the prevalence and prognostic significance of HNa in the setting of acute STElevation myocardial infarction (STEMI) is sparse.

Methods: We studied 1047 consecutive patients (pts) (age 60 ± 12) presenting with STEMI. Plasma sodium concentrations (PNa) were obtained on admission and at 24-h, 48-h, and 72-h. Multiple logistic regression was performed to determine the relation between HNa and 30-day mortality adjusting for age, sex, diabetes, hypertension, smoking, Killip class, peak CK, LVEF, anterior infarction, use of diuretics and reperfusion therapy.

Results: HNa (PNa < 135 mmol/L) was present on admission in 131 pts (12.5%) and developed in 208 (19.9%) during the first 72-h. PNa decreased to ≤130 mmol/L, in 75 (7.2%) of pts. Pts receiving diuretics developed HNa more commonly compared to pts who were not (46% vs 28%, p<0.0001), but the majority of hyponatremic pts (66%) were not receiving diuretics. Kaplan-Meyer curves indicated that pts with HNa were at increased risk of mortality (Figure), HNa on admission (OR 2.2, 95% CI 1.2-4.2, p=0.015) and HNa developing early after admission (OR 2.7, 95% CI 1.6-4.6, p<0.0001) were independent predictors of 30-days mortality.

Conclusion: HNa on admission, or early development of HNa in patients with STEMI is a strong independent predictor of 30-day mortality. PNa may serve as a simple marker to identify patients at high risk.

4:45 p.m.

Featured Oral Session...Acute Coronary Syndromes: Influence of Diabetes on Prognosis

Monday, March 08, 2004, 4:00 p.m.-5:30 p.m.
Morial Convention Center, Room 217

Featured Oral Session...Acute Coronary Syndromes: Influence of Diabetes on Prognosis

Fasting Glucose Is an Independent Predictor of 30-Day Mortality in Nondiabetic Patients With Acute Myocardial Infarction: A Prospective Study

Mahmoud Saleman, Walter Markiewicz, Michael Kapeliovich, Haim Hammerman, Doron Aronson, Rambam Medical Center, Haifa, Israel

Background: Studies have suggested that hyperglycemia may increase mortality after acute myocardial infarction (AMI). However, these studies have not adjusted for important determinants of prognosis and hyperglycemia was defined arbitrarily as a dichotomous threshold.

Results: At baseline, expression of 5-lipoxygenase, MCP-1 and IL-6 was higher in chronic ischemic myocardium than in normally perfused myocardium. In response to acute ischemia and reperfusion in the normal myocardium, there was a clear trend towards increased expression of pro-inflammatory factors (e.g. 5-lipoxygenase, COX-2, CysLT2 receptor, MCP-1, IL-6). These responses were less pronounced or detectably absent in the chronic ischemic myocardium.

Conclusion: These findings suggest a discrete increase in several pro-inflammatory factors that may contribute to myocardial pathophysiology in chronic myocardial ischemia. The blunted inflammatory response to acute ischemia in the chronic ischemic myocardium may suggest altered sensitivity in signaling pathways mediating the inflammatory response.

5:15 p.m.