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

Marcin Gabrowski, Krzysztof J. Filipiak, Grzegorz Karpinski, Adam Rdzanek, Arkadiusz Pietrasik, Zenon Huczek, Grzegorz Honszczak, Janusz Kochman, Grzegorz Opolski, Medical University of Warsaw, Warsaw, Poland

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±182.2 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). The odds ratio for TIMI 3 flow grade after PCI (328.3±332.8 pg/ml vs. 151.3±199.7 pg/ml; p<0.01) was reduced in SMG patients. After adjustment for baseline BNP, the increase in mortality was independent of reperfusion therapy (p=NS). Conclusion: Baseline BNP levels on admission are powerful, independent predictor 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.

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Profiling Inflammation in the Ischemic Human Heart In Vivo

Anders Gabrielsen, Patrick Lawer, Jesper Haeggestrom, Gabrielle Berne Paulsson, Wang Yongzhong, Daniel Steinbruchl, Jens Kastrup, Goran Hansson, Karolinska Institute, Stockholm, Sweden, Copenhagen University Hospital, Copenhagen, Denmark

Background: The present understanding of myocardial inflammation and its potential role in myocardial pathophysiology is incomplete. Therefore, we used Affymetrix® GeneChip analysis on normal and chronic ischemic myocardium before and during acute ischemia and reperfusion stress to profile expression of inflammation-related transcripts. Methods: Biopsies were sampled at baseline, after 45 min of acute total ischemia, and after 30 min of reperfusion recovery from normally perfused and chronic reversibly ischemic myocardium in 6 patients undergoing coronary artery bypass grafting. RNA was isolated, amplified with established techniques, and hybridized to HG_U133A Affymetrix® Genechip arrays. A total of 33 arrays were performed, normalized with the RMA algorithm, and analyzed for expression patterns of pro- and anti-inflammatory factors.

827-6

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

Alexander Goldberg, Haim Hammarberg, Sirouch Petcherski, Sergey Yalontsky, Alexander Zdorovyak, Michael Kapelowich, Walter Markiewicz, Doron Aronson, Rambam Medical Center, Haifa, Israel, Rappaport Medical School, Haifa, Israel

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 ST-elevation 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%) pts during the first 72-h. PNa decreased to ≤130 mmol/L in 75 (7.2%) 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<0.001) 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.
Women With Diabetes Mellitus Have the Greatest Reduction in Myocardial Infarction Mortality Over the Past Decade: Evaluation of 1,428,596 Patients Enrolled in the National Registry of Myocardial Infarction 2, 3, and 4 From 1994-2002


Background: Analyses from NHANES showed a decline in cardiovascular (CV) mortality in the 1970s-80s, but among patients with diabetes mellitus (DM), the rate of decline was attenuated among men (less improvement) and women (no improvement). We sought to examine the relation between sex, DM, and hospital mortality in the setting of contemporary care for myocardial infarction (MI).

Methods: We analyzed data from 1,428,596 patients with MI enrolled in NRMII 2, 3, & 4 from 1994-2000. DM was present in 410,223 patients (29%), increased in prevalence over the study period (26% to 31%), and was associated with higher adjusted hospital mortality (13.8% vs. 10.9%; adjusted OR=1.3 [95% CI 1.3-1.33]. Use of ASA, thienopyridines, statins, and ACE inhibitors increased over the study period (26% to 31%), and was associated with lower adjusted hospital mortality (13.8% vs. 10.9%; adjusted OR=1.3 [95% CI 1.3-1.33]).

Results: DM was associated with lower 30-day mortality among patients with placebo (13.8% vs. 10.9%; adjusted OR=1.3 [95% CI 1.3-1.33]. Use of ASA, thienopyridines, statins, and ACE inhibitors increased over the study period (26% to 31%), and was associated with lower adjusted hospital mortality (13.8% vs. 10.9%; adjusted OR=1.3 [95% CI 1.3-1.33]. Use of ASA, thienopyridines, statins, and ACE inhibitors increased over the study period (26% to 31%), and was associated with lower adjusted hospital mortality (13.8% vs. 10.9%; adjusted OR=1.3 [95% CI 1.3-1.33]. Use of ASA, thienopyridines, statins, and ACE inhibitors increased over the study period (26% to 31%), and was associated with lower adjusted hospital mortality (13.8% vs. 10.9%; adjusted OR=1.3 [95% CI 1.3-1.33]. Use of ASA, thienopyridines, statins, and ACE inhibitors increased over the study period (26% to 31%), and was associated with lower adjusted hospital mortality (13.8% vs. 10.9%; adjusted OR=1.3 [95% CI 1.3-1.33]. Use of ASA, thienopyridines, statins, and ACE inhibitors increased over the study period (26% to 31%), and was associated with lower adjusted hospital mortality (13.8% vs. 10.9%; adjusted OR=1.3 [95% CI 1.3-1.33]. Use of ASA, thienopyridines, statins, and ACE inhibitors increased over the study period (26% to 31%), and was associated with lower adjusted hospital mortality (13.8% vs. 10.9%; adjusted OR=1.3 [95% CI 1.3-1.33]. Use of ASA, thienopyridines, statins, and ACE inhibitors increased over the study period (26% to 31%), and was associated with lower adjusted hospital mortality (13.8% vs. 10.9%; adjusted OR=1.3 [95% CI 1.3-1.33]. Use of ASA, thienopyridines, statins, and ACE inhibitors increased over the study period (26% to 31%), and was associated with lower adjusted hospital mortality (13.8% vs. 10.9%; adjusted OR=1.3 [95% CI 1.3-1.33]. Use of ASA, thienopyridines, statins, and ACE inhibitors increased over the study period (26% to 31%), and was associated with lower adjusted hospital mortality (13.8% vs. 10.9%; adjusted OR=1.3 [95% CI 1.3-1.33]. Use of ASA, thienopyridines, statins, and ACE inhibitors increased over the study period (26% to 31%), and was associated with lower adjusted hospital mortality (13.8% vs. 10.9%; adjusted OR=1.3 [95% CI 1.3-1.33]. Use of ASA, thienopyridines, statins, and ACE inhibitors increased over the study period (26% to 31%), and was associated with lower adjusted hospital mortality (13.8% vs. 10.9%; adjusted OR=1.3 [95% CI 1.3-1.33]. Use of ASA, thienopyridines, statins, and ACE inhibitors increased over the study period (26% to 31%), and was associated with lower adjusted hospital mortality (13.8% vs. 10.9%; adjusted OR=1.3 [95% CI 1.3-1.33]. Use of ASA, thienopyridines, statins, and ACE inhibitors increased over the study period (26% to 31%), and was associated with lower adjusted hospital mortality (13.8% vs. 10.9%; adjusted OR=1.3 [95% CI 1.3-1.33]. Use of ASA, thienopyridines, statins, and ACE inhibitors increased over the study period (26% to 31%), and was associated with lower adjusted hospital mortality (13.8% vs. 10.9%; adjusted OR=1.3 [95% CI 1.3-1.33]).

Conclusion: Between 1994-2000, MI hospital mortality declined among patients with and without DM, associated with increasing use of evidence-based CV therapies. Patients with DM had the greatest mortality improvement, and contrary to the earlier NHANES findings, women with DM have recently experienced the greatest mortality improvement.