

Acute Coronary Syndromes

MONOCYTOSIS AFTER MYOCARDIAL INFARCTION ACCELERATES ATHEROSCLEROSIS: RESULTS OF A FIVE-YEAR FOLLOW-UP STUDY

Poster Contributions

Poster Sessions, Expo North

Sunday, March 10, 2013, 3:45 p.m.-4:30 p.m.

Session Title: The Blood Tells a Story: Coeptin, Fatty Acid Binding Protein, NT-Pro BNP and More

Abstract Category: 1. Acute Coronary Syndromes: Clinical

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Background: The risk of cardiovascular complications, including recurrent myocardial infarction, sudden cardiac death, and stroke, for those who survived acute myocardial infarction is substantial even in the era of optimal reperfusion strategies. Recent experimental studies have reported myocardial infarction (MI) itself accelerates atherosclerosis via extramedullary monocytosis. Previous studies including ours have shown that peripheral monocytosis is associated with plaque progression after MI. We sought to investigate the impact of peak monocyte count on atherosclerotic events after MI.

Methods and Results: 5-year clinical follow-up data were collected from 255 consecutive patients (210 male, aged 62 ± 11 years) with ST-elevation MI who underwent primary percutaneous coronary intervention. The major adverse cardiac and cerebrovascular events (MACCE) were defined as cardiac death, sudden death, non-fatal MI, unstable angina, and stroke. MACCE occurred in 34 patients during 5-year follow-up, including 2 cardiac deaths, 2 sudden deaths, 8 non-fatal MI, 14 unstable angina, and 8 stroke. Patients with MACCE had a higher prevalence of diabetes (47% vs. 24%, $p=0.004$) but other characteristics including age, sex, and peak monocyte level (888 ± 211 vs. 922 ± 803 IU/L) were similar between the groups. Multivariate Cox proportional hazards analysis revealed that diabetes (Hazard ratio(HR) 2.99, 95% confidence interval (CI) 1.48 to 5.86, $p=0.002$) and higher peak monocyte count (defined as $800/\text{mm}^3$, HR 2.63, 95% CI 1.26 to 5.52, $p=0.01$) were significantly predictive of MACCE independent of age, sex, peak C-reactive protein level after MI, and infarct size assessed by peak creatin kinase. Kaplan-Meier survival curves showed that MACCE free survival in the high monocyte group ($n=131$) was significantly worse than low monocyte group ($n=124$) (82% vs. 91%, $p=0.046$ by log-rank test).

Conclusions: High peak monocyte count after ST-elevation MI was associated with adverse atherosclerotic cardiovascular events. Monocytosis after MI may accelerate atherosclerosis independent of infarct size and established cardiovascular risk factors.