ACC.11
60th Annual Scientific Session & Expo

E1078
JACC April 5, 2011
Volume 57, Issue 14

MYOCARDIAL ISCHEMIA AND INFARCTION

HIGHER MORTALITY OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE PATIENTS AFTER PCI IS ONLY PARTLY EXPLAINED BY PULMONARY HYPERTENSION OR LEFT VENTRICULAR DYSFUNCTION

ACC Poster Contributions
Ernest N. Morial Convention Center, Hall F
Monday, April 04, 2011, 3:30 p.m.-4:45 p.m.

Session Title: ACS Comorbidities and Outcome
Abstract Category: 4. Unstable Ischemic Syndrome/Long-Term Outcome
Session-Poster Board Number: 1106-365

Authors: *Tomas Konecny, Radim Spacek, Lujza Galkova, Krishen Somers, Marek Orban, Vladimir Konecny, Paul D. Scanlon, Charanjit S. Rihal, Mayo Clinic, Rochester, MN*

Background: Chronic obstructive pulmonary disease (COPD) is associated with increased mortality after percutaneous coronary interventions (PCI). Whether pulmonary hypertension or left ventricular dysfunction (both of which exist in COPD) could explain this worse prognosis after PCI is not known.

Methods: We identified consecutive patients who underwent PCI at Mayo Clinic Rochester between 1995 and 2005, with a baseline (pre-PCI) Doppler estimated right ventricular systolic pressure (RVSP) and left ventricular ejection fraction (LVEF). Post PCI follow up data was collected prospectively at 6 months, 1 year, and then annually.

Results: This study included 3876 patients (71 ± 11 years, 63% male, BMI 29 ± 6 kg/m2, LVEF 54 ± 15%), of which 468 (12%) had a diagnosis of COPD. Patients with COPD had modestly higher RVSP (42 vs 38 mmHg, p<0.0001), and lower LVEF (51 vs 54%, p<0.0001). Mean follow up was 5 years. Diagnosis of COPD, higher RVSP, and lower LVEF were highly significant (p<0.0001) predictors of mortality both in univariate and multivariate models. COPD was strongly associated with lower survival even after adjustment for all confounders including RVSP and LVEF (Figure, p<0.0001).

Conclusion: Our results suggest that neither pulmonary hypertension nor left ventricular dysfunction can fully explain the markedly increased mortality of COPD patients after PCI, and that additional mechanisms (inflammation, hypoxemia, etc.) could be playing a role.