were randomized 3-14 days after the index AMI to EPL (25 mg titrated to 50 mg QD; n=3319) or placebo (PBO; n=3313). Patients were followed for up to 2.5 years (mean=16 months). End point analyses were performed with a Cox proportional hazards regression model with treatment group as the only factor, stratified by geographic region. Treatment-by-HR ($\leq$90/>90 bpm) factor interaction was based on the Wald's test of the interaction term in the Cox model.

The rate of all-cause mortality in PBO-treated patients was 25.2% in those with baseline HR >90 bpm and 15.9% with baseline HR $\leq$90 bpm. Reductions in risk with EPL relative to PBO according to baseline HR are in Figure. Although most of the treatment-by-factor interaction P-values were not significant, the reduction in risk for SCD in patients with HR >90 bpm was greater compared to patients with HR <90 bpm (treatment-by-factor interaction P-value=0.04).
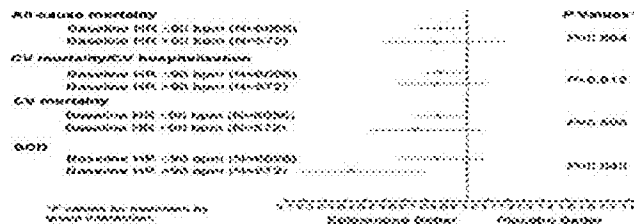

Figure 1. Relative Risk of End points by Baseline HR.

At an average of 16 months of follow-up, EPL treatment demonstrated a consistent effect on the risk of ACM, CVM/H, and CVM in EPHESUS patients with baseline HR $\leq$90 and >90 bpm. While EPL treatment reduced the risk of SCD in patients with HR $\leq$90 and >90 bpm, patients with a baseline HR >90 bpm may derive a larger treatment effect for this end point.

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The impact of statin therapy on the efficacy of eplerenone

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In EPHESUS, treatment with eplerenone (EPL) significantly reduced mortality and morbidity in patients with acute myocardial infarction (AMI) and LVSD with signs of heart failure (HF) when used with standard therapies. Because post-AMI HF patients often receive statin therapy, this retrospective analysis evaluated the treatment effect of EPL used concurrently with standard HF treatment in EPHESUS patients w/ and w/out concomitant statin therapy.

Patients w/ post-AMI HF and LVEF </= 40% on standard therapy were randomized 3-14 days after index AMI to EPL (25 mg titrated to 50 mg QD; N=3319) or placebo (PBO; N=3313) and followed for up to 2.5 years. The comparative analyses of treatment effects in patients who received statins from baseline (from index AMI up to 14 days post-AMI) and those who did not were performed using a Cox proportional hazards regression analysis stratified by the geographical region. Treatment-by-statin interaction was tested using Cox model.

Patients not on statin therapy appeared to have increased risk for end points compared with those on statins; the rate of all-cause mortality in PBO-treated patients was 19.5% and not on statins and 13.6% in those on statin therapy. Risk reductions with EPL relative to PBO for patients w/ and w/out concomitant statin therapy were similar (P >/=0.12) for

each endpoint (Figure 1). There was no evidence of toxicity related to the coadministration of EPL and statins, and the incidence of adverse events with EPL was similar to PBO.
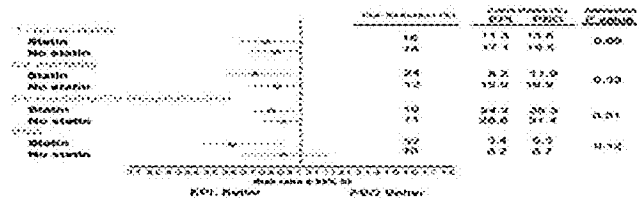

Figure 1. Relative Risk of Endpoints in EPHESUS by Baseline Statin Treatment.

EPL treatment provided mortality and morbidity benefits in post-AMI HF patients w/ and w/out concomitant statin therapy, with greater benefits in those receiving concomitant statin therapy. For each end point, event rates were lower with EPL than PBO, independent of statin treatment.

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Clinical parameters on admission: can they predict in-hospital heart failure after acute coronary syndromes?

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Introduction: heart failure (HF) is a frequent complication of acute coronary syndromes (ACS) and is associated with poor prognosis. Our goal was to find clinical parameters on admission that could predict HF, thereby helping select the most suitable management strategy.

Methods: we studied 1045 patients (61.4 ± 12.5 years-old, 71.7% males) consecutively admitted in our cardiology department with ACS. Clinical and laboratory parameters were obtained during the first 48 hours. The outcome was development of clinical signs of HF during hospitalisation.

Results: cardiovascular risk factors were found in a significant proportion of our sample (hypertension in 55.9%, diabetes mellitus in 29.1%, current or previous smoking in 50%, dyslipidaemia in 62.2%, body mass index > 30 Kg/m^2 in 20%). Forty-three percent of the patients had ACS with ST segment elevation (STEMI) and 40% had left ventricular systolic dysfunction (LVSD). Multivessel and/or left main coronary artery disease were found in 52.3%. Revascularisation was performed in 60.7%. Heart failure occurred in 15.9%. Variables associated with development of HF in univariate analysis were: male sex (OR 0.446, 95%CI 0.317-0.629), age (OR 1.061, 95%CI 1.045-1.068), smoking (OR 0.463, 95%CI 0.328-0.655), hypertension (OR 1.528, 95%CI 1.083-2.157), diabetes (OR 2.639, 95%CI 1.878-3.709), dyslipidaemia (OR 0.531, 95%CI 0.380-0.742), LVSD (OR 8.042, 95%CI 5.378-12.025), STEMI (OR 2.130, 95%CI 1.519-2.987), revascularisation (OR 0.513, 95%CI 0.358-0.736), serum creatinine (OR 1.173, 95%CI 1.044-1.318), hematocrit (OR 0.9, 95%CI 0.871-0.929), glycaemia (OR 1.07, 95%CI 1.05-1.091) and troponin I (OR 1.004, 95%CI 1.002-1.006). Multivariate analysis, using a stepwise binary logistic regression method, revealed that the only ones with independent predictive value were: age (OR 1.056, 95%CI 1.036-1.076), LVSD (OR 6.381, 95%CI 4.009-10.156), STEMI (OR 1.628, 95%CI 1.056-2.510), hematocrit (OR 0.916, 95%CI 0.882-0.952) and glycaemia (OR 1.064, 95%CI 1.039-1.090, for each 10 mg/dl increment). Using the predicted probabilities of this model we constructed a ROC curve. Its AUC was 0.85 (p<0.001 vs ND). Using the best cut-off of predicted probabilities this model showed a sensitivity of 88.3%, specificity of 64.5%, positive predictive value of 33% and negative predictive value of 96.7%.

Conclusion: in-hospital post-ACS heart failure might be predicted on admission using common practice clinical and laboratory data.