AMI causing cardiogenic shock in patients with severely depressed left ventricular function

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Introduction: Left ventricular function is assumed to be the main predictor of cardiogenic shock (CS), however trials and registries show that in average left ventricular function is only moderately depressed in CS after acute myocardial infarction.

Purpose: Characterize population of patients (Pts) with CS after acute myocardial infarction (AMI) and with severe left ventricular dysfunction (defined as ejection fraction (EF) <30%).

Methods: From a national multicenter registry, we evaluated 729ptswith CS after AMI.We considered 2 groups: Group 1 – pts with CS and EF <30% and Group 2 – pts with CS and EF >30%. We registered age, gender, cardiovascular and non-cardiovascular comorbidities, electrocardiographic presentation, vital signs at admission, reperfusion strategy and coronary anatomy. We also evaluated in-hospital complications, such as re-infarction, mechanical complications, high-grade atrial ventricular block, sustained ventricular tachycardia (VT), atrial fibrillation (AF) and stroke. We compared in-hospital mortality and multivariate analysis was performed to assess the impact of EF in in-hospital mortality and to identify predictors of severe left ventricular function.

Results: Severe dysfunction in Cardiogenic shock due to AMI was present in 28.9% (n=211) of pts (68% male, mean age of 72±12 years old). Group 1 had higher incidence of previous heart disease, such as AMI, previous PCI and congestive heart failure (27% vs 14%, p<0.001; 17.7% vs 9.6%

p=0.002 and 16% vs 10%, p=0.022, respectively). STEMI pts were 71% (n=149), and timing from symptoms until first contact was longer (185 min (90; 437) vs 123 (60; 300), p<0.001). Undetermined location AMI was more often in group 1 (8% vs 2%, p<0.001), particularly due to left or right bundle brunch block (13% vs 4.7%, p<0.001, and 15% vs 10%, p=0.041 respectively). Anterior STEMI was also more prevalent in this groups (81% vs 46%, p<0.001). No differences were observed on coronariography rate, rate or type of reperfusion nor multivessel disease. Group 1 pts presented more with left main (LM) (25% vs 12%, p<0.001) and anterior descending (AD) (9.4% vs 2.4%, p<0.001) arteries lesions (88% vs 72.4%, p<0.001) or occlusion (65.5% vs 33.7%, p<0.001). Group 1 presented more with inhospital VT (16% vs 10.8%, p=0.048). In-hospital mortality was also higher (56.5% vs 29.5%, p<0.001). After multivariate analysis we found that severe left ventricular dysfunction was a mortality predictor (OR 3.37; 95% CI 2.05-5.54, p<0.001). LM (OR 3.41; 95% CI 1.86-6.26, p<0.001) and AD (OR 2.74; 95% CI 1.51-4.96, p=0.001) arteries disease and previous AMI (OR 2.36; 95% CI 1.28-4.37, p=0.006) were predictors of severe LV dysfunction.

Conclusions: Severely depressed EF is a predictor of in-hospital mortality. Left main and anterior descending artery disease and previous AMI were identified as predictors of an EF <30%.