Letter to the Editor

Esmolol for tight heart rate control in patients with STEMI: Design and rationale of the beta-blocker in acute myocardial infarction (BEAT-AMI) trial☆

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Long term benefit of beta-blocker treatment is well established for secondary prevention after acute MI [1]. The impact of beta-blockade in the acute phase of a myocardial infarction is a matter of discussion. Oral beta-blockade is recommended for all patients with myocardial infarction during hospital stay and thereafter [2]. Routine use of early intravenous beta-blockade is not recommended.

The concerns linked to intravenous beta-blocker treatment are mainly based on historical or pre-PCI trials. During the pre-reperfusion era intensified usage of intravenous beta-blockade demonstrated mortality reduction in patients with acute MI [3,4]. Contrary, studies with thrombolysis therapy in patients with acute MI uncovered more cardiogenic shocks in patients receiving intravenous beta-blocker therapy. The elevated risk for cardiogenic shocks was explained by MI intensity, reflected by higher Killip classes [5]. The impact of intravenous beta-blocker for patients undergoing PCI for acute MI suggests beneficial effects [6,7]. Most recently the METOCARD trial demonstrated protective effects of intravenous metoprolol administration before PCI in patients with STEMI [8].

Due to emotional stress and increased endogenous catecholamines subjects with acute MI reveal high sympathetic activity reflected by elevated heart rate [9,10] which aggravate cardiac damage in ischemia. Controlling the heart rate with beta-blocker may limit sympathetic influence on cardiac regeneration and therefore be protective.

We are conducting a randomized single-blinded clinical trial comparing tight heart rate control with intravenous esmolol versus standard therapy in patients with acute KILLIP I or II STEMI and successful PCI (Fig. 1). The BEAT-AMI is the first clinical study evaluating the impact of beta-blocker induced tight heart rate control in patients with acute myocardial infarction for the first 24 h after successful PCI.

The primary objective of the BEAT-AMI trial is to evaluate the efficacy of esmolol-induced heart rate control as compared with placebo when used in addition to standard medical therapy in patients with acute STEMI in reducing final infarct size reflected by Troponin T release (peak and AUC). Assuming a heart rate reduction of 10 bpm with esmolol compared to placebo we expect a reduction in mean troponin T max of about 3 ± 0.5 ± 5.87 μg/l, assuming a coefficient of variation of 1, such a troponin T max reduction may be detected with 92% power, obtained by simulation, using the Welch-modified t-test with 50 patients per treatment group (at 5% two-sided significance level). Allocated patients will receive weight-adjusted continuous esmolol-infusion for 24 h targeting a heart rate of 60 bpm compared to placebo-infusion with no heart rate control. The study was funded by Baxter Healthcare (New Jersey, USA).

The BEAT-AMI trial is the first, randomized, single-blind, placebo-controlled, trial designed to assess the efficacy of esmolol-induced tight heart rate control versus placebo for reduction of troponin T release as a surrogate marker of necrosis expansion in subjects with acute STEMI.

Conflict of interest

None.

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Fig. 1. Flow chart of the study.

References