RESULTS In the meta-analysis there was no significant difference in all-cause mortality (RR: 0.89, 95% CI: 0.78 - 1.04, P = 0.579) between AT and PPCI arm at 1-month (RR: 0.89, 95% CI: 0.76 - 1.04, P = 0.155), 6-12 months (RR: 0.87, 95% CI: 0.38 - 1.98, P = 0.620) and >12 months (RR: 0.73, 95% CI: 0.49 - 1.09, P = 0.12) follow-up. The 2 groups were similar with regards to target vessel revascularization (RR: 0.79, 95% CI: 0.55 - 1.04, P = 0.146) and stroke (RR: 1.05 - 1.08, 95% CI: 0.84 - 2.50, P = 0.620) however there was a significant reduction of recurrent MI (RR: 0.67, 95% CI: 0.46 - 0.97, P = 0.032) and MACE (Death + MI + target vessel revascularization) rate (RR: 0.81, 95% CI: 0.66 - 0.99, P = 0.031) in AT arm when compared to PPCI. The net clinical benefit (MACE + stroke) was similar in the two groups (RR: 0.97, 95% CI: 0.87 - 1.07, P = 0.710).

CONCLUSIONS In this largest till date meta-analysis, including all the available randomized controlled trial, we show that AT prior to PPCI in STEMI is associated with similar mortality, stroke and target revascularization rates however there is a significant reduction of MACE rate in the AT arm primarily driven by reduced rates of recurrent MI. There was no difference between the two treatment arms in terms of net clinical benefit.

CATEGORIES CORONARY: Thrombus / Thrombectomy and Embolic Protection

KEYWORDS Major adverse cardiac events, ST-segment elevation myocardial infarction, Thrombus aspiration

TCT-241

Endovascular hypothermia treatment dose-modulates cardioprotection in favor of 32°C target temperature before reperfusion in porcine myocardial infarction

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BACKGROUND Previous evaluations of endovascular cooling during primary PCI for acute myocardial infarction (MI) in human and animal studies suggest a treatment effect on infarct size (IS) related to core temperature at the time of reperfusion. These observations have not translated into results of recent randomized trials. We investigated a comparative dose response relationship between myocardial salvage and depth of cooling.

METHODS Twenty-four pigs were randomly assigned to 3 groups: normothermia 38°C, mild hypothermia 35°C and moderate hypothermia 32°C. MI was induced by 1-hour balloon occlusion of the mid LAD followed by reperfusion. A heat-exchange balloon catheter placed in the inferior vena cava controlled core temperature. Cooling was initiated 30 minutes after occlusion and maintained for 1 hour followed by rewarming to normothermia. IS was assessed at day 6 with magnetic resonance imaging (MRI) and pathology (TTC).

RESULTS Target temperature was reached in 9±5 and 29±8 minutes for 35°C and 32°C, respectively. Area-at-risk (AAR) from MRI staining was equivalent in all groups (30±6, 28±7, 26±6, ANOVA p = 0.217). Both the 32°C and 35°C showed significant IS reduction per AAR (45±12, 17±11, 4±3; 62% and 91% reduction, respectively, p<0.001) and a similar reduction per LV mass (14±5, 5±3, 1±1, p<0.001). Further, 32°C showed a significant 76% IS per AAR reduction relative to 35°C (p<0.012) suggesting additional tissue salvage from deeper cooling. In the linear regression model, target temperature was a significant predictor of IS reduction both per AAR and LV (R² = 0.746 and 0.685, respectively, p<0.001). The predicted infarct reduction is 7% of AAR and 2% of LV per 1°C drop at reperfusion. Additionally, 32°C had less variability compared to 35°C in reducing IS (standard deviation 3±1 vs 10±4; p<0.001). Cardiac output relative to baseline was significantly preserved in 32°C only (–30%±19%, -17±32, -12±42, 9±46, p<0.044). Both 35°C and 32°C exhibited similar IS and LV reductions in T2 edema suggesting reduced injury with hypothermia (p<0.05).

CONCLUSIONS Pre-reperfusion rapid therapeutic hypothermia shows a dose-response infarct size reduction as well as favorable hemodynamic outcomes for moderate hypothermia more consistently than mild hypothermia.

CATEGORIES CORONARY: Acute Myocardial Infarction

KEYWORDS Acute myocardial infarction, Cardioprotection, Hypothermia

TCT-242

Remnant risk of secondary cardiovascular events in stable post-myocardial infarction 1-year survivors

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BACKGROUND State-of-art therapeutics for acute myocardial infarction (AMI) have dramatically improved clinical outcome during the first year after index MI. However, few data exist on the remnant risk of recurrent cardiovascular (CV) events in the stable post-MI 1-year survivors.

METHODS We consecutively enrolled MI patients who underwent percutaneous coronary intervention (PCI) in the COREA-AMI (CardioVascular Risk and identiFicAtion of potential high-risk population in AMI) registry including nine major university hospitals throughout South Korea from January 2004 to December 2009. Patients who were alive and did not experience a recurrent MI or stroke during the first 365 days post-index MI were analyzed in this study. We defined “high-risk” as patients with at least one of the following risk factors: age ≥ 65 years, diabetes mellitus, prior MI, multi-vessel disease, or renal dysfunction. The primary endpoint was a composite of CV death, non-fatal MI, or stroke from Day 366 to study completion.

RESULTS Of 4,748 AMI patients, 4,200 patients were alive at 1 year after index MI stably without non-fatal MI or stroke. Median follow-up duration was 43.8 months (interquartile range 29.8 to 60.5 months). Within the first year, combined endpoints of CV events were 3.0% at low risk and 8.4% at high risk [HR (95% CI): 2.87 (1.91 - 4.31), p<0.001]. From 1 year to 4 year after index MI, cumulative incidence of CV events were 3.9% at low risk and 8.8% at high risk [2.25 (1.49-3.40), p<0.001]. In multivariable Cox proportional regression analysis, additional risk factors of stable post-MI patients were left ventricular dysfunction [1.50 (1.15-1.95), p<0.002] and anemia [1.40 (1.08-1.83), p<0.011]. Patients without taking dual antiplatelet agent at 1 year have borderline remnant risk [1.31 (0.99-1.73), p = 0.059].