OBJECTIVES Cellular repressor of E1A-stimulated genes (Creg) is a secreted glycoprotein that regulates tissue and cell homeostasis and has been shown to antagonize heart injury; however, little is known about the role of CREG in ischemia/reperfusion injury (IRI). In this study, we aimed to investigate the role of CREG in cardiac IRI and clarify the mechanism.

METHODS Myocardial ischemia/reperfusion (MIR) was established by ligation of left descending coronary artery for 30 min in wild type C57 mice ($Creg^{+/+}$) and heterozygous Creg mice ($Creg^{+/-}$). Expression of CREG was determined by Western Blot. Infarction size was determined by TTC and Evan's Blue staining 24 hours after MIR. Cardiac function was evaluated by echocardiography at day 28. Recombinant CREG protein (0.3mg/kg·d) was supplemented through micro-pump embedded subcutaneously in $Creg^{+/+}$ mice. Cardiac function and infarction size were assessed as mentioned above. Apoptosis, lysosome formation and function were detected in both heart tissue and cardiomyocyte cell line H9C2.

RESULTS Level of CREG protein in mouse hearts was downregulated after mice were subjected to MIR, especially in $Creg^{+/-}$ mice. $Creg^{+/-}$ mice had larger infarction sizes after 24 hours of MIR treatment and worse cardiac function at day 28 compared with $Creg^{+/+}$ mice. However, cardiac function was significantly improved in $Creg^{+/+}$ mice treated with recombinant CREG protein. In $Creg^{+/-}$ mice, the number of cardiomyocytes undergoing apoptosis was increased, and LC3A and p62 accumulated, suggesting that autophagy was dysfunctional. Upregulation of CREG decreased apoptosis and activated autophagy of cardiomyocytes. Furthermore, we demonstrated that CREG protects cardiomyocytes against apoptosis by activating autophagy both *in vivo* and *in vitro*.

CONCLUSIONS CREG overexpression protects cardiac function against IRI by preventing cardiomyocyte apoptosis. The protective effects of CREG are partly mediated by activation of lysosomal autophagy.

CARDIOVASCULAR SURGERY

GW28-e0026

Incidence and Risk Factors of postoperative AKI in Patients with Type A Aortic Dissection and the Impact of the acute kidney injury (AKI) on Short-term Outcomes: A Updated Meta-Analysis and Meta-Regression

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OBJECTIVES Background: This is the first meta-analysis investigating the incidence and risk factors of postoperative acute kidney injury (AKI) in patients with type A aortic dissection (TAAD) and the impact of the AKI on short-term outcomes.

METHODS This systematic review and meta-analysis were conducted and reported in adherence to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA). Multiple electronic literature databases were searched using predefined criteria. Articles that were published between January 1, 2011 and November 30, 2016 were screened; 7 eligible articles were included in the meta-analysis. Differences are expressed as odds risk (OR) with 95% confidential interval (CI). Study heterogeneity was tested using the I2 statistic. Study heterogeneity was considered significant when P values < 0.10 or $I2 > 50\%$. A fixed-effects model was used when study heterogeneity was not significant; a random effects model was used when study heterogeneity was significant. Egger's linear regression test was used to estimate publication bias. Meta-regression analysis was performed to examine whether covariates, which could be potential modifiers, can affect the end results of the meta-analysis.

RESULTS The odds risk of actual total incidence of AKI events from the 7 studies was 0.46 (95% CI: 0.43-0.48). The meta-analysis revealed that postoperative AKI was associated with a 247% increase in 30-day postoperative mortality (OR: 3.47, 95% CI: 2.18-5.51, P<0.0001) without significant study heterogeneity and publication bias. Subgroup analysis found that the patients with stage 2 or 3 AKI showed a 445% increase in the mortality compared with the patients without the complication (OR: 5.45, 95% CI: 2.87-10.36, P<0.0001). Meta-regression showed that the potential modifiers, such as publication year, AKI definition, male sex, mean age, preoperative creatinine levels, and body mass index (BMI) had no significant effects on the association between AKI and 30-day mortality. Postoperative AKI also increased the incidence of 30-day postoperative stroke and bleeding by 143% (OR: 2.43, 95% CI: 1.52-3.87, P<0.0001) and 432% (OR: 5.32, 95% CI: 2.11-13.42, P<0.0001), respectively. Multiple logistic regression analysis revealed that patients' advancing age (OR: 1.26, 95% CI: 1.11-1.43, P<0.0001), high BMI (OR: 1.26, 95% CI: 1.02-1.55, P=0.175), and perioperative sepsis (OR: 1.91, 95% CI: 1.13-3.29, P=0.017) independently correlated with postoperative AKI in patients with TAAD.

CONCLUSIONS Patients with TAAD appear to a have high risk of postoperative AKI, which may increase 30-day postoperative mortality and the incidences of stroke and bleeding. Patients' advancing age, high BMI, and perioperative sepsis independently correlate with postoperative AKI.

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The Management Manner and Reasons Analysis for Stress-induced Hypertension in Adults with Aortic Dissection (Stanford type A) after Bentall or Improved-Cabrol of Cardiac Surgery

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OBJECTIVES To discuss the management manner and reasons analysis for stress-induced hypertension in adults with aortic dissection (Stanford type A) after Bentall or Improved-Cabrol of cardiac surgery while aortic and/or aortic valve replaced with trunk stent implantation meanwhile.

METHODS A total of 108 cases with stress-induced hypertension were retrospectively analyzed, the pressure higher than 160/90 mmHg during early period of post-operation, in adults with aortic dissection (Stanford type A) after Bentall or Improved-Cabrol of cardiac surgery in our hospital from 2014.1 to 2016.12 were retrospectively analyzed. The pre-operative basic clinical information, in-operative condition, operative manners and post-operative hemodynamic were summarized in all ones. Cardiac Index (CI), systolic and diastolic blood pressure (SBP/DBP), mean pressure (MAP), heart rate (HR), systemic vascular resistance (SVR), central venous pressure (CVP) and some else were monitored and analyzed. Therapy: 1. Removal motivation; 2.Parameters of ventilator adjusted; 3. Using vasodilator substance, β-receptor inhibitor, calcium channel blocker; 4. Keep internal environment homeostasis; 5. Sedation and analgesia drugs applied according to guidelines.

RESULTS The morbidity of stress-induced hypertension in Bentall or improved-Cabrol cardiac surgery higher than other manners like congenital heart disease, valves replacements and else(P<0.05). However, little change in Bentall and improved-Cabrol(P>0.05) as well as CI and CVP compared to pre-post stress-induced hypertension(P>0.05). Positive correlation appears to stress-induced hypertension of post-operation in patients compare to hypertension of pre-operation(r=0.691, P<0.05). Furthermore, SBP, DBP, MAP and SVR decreased obviously when the pressure controlled(P<0.01). Importantly, a significant positive correlation associated to metabolic acidosis(r=0.532, P<0.05), hyperlactacidemia(r=0.614, P<0.05), no sedative and/or analgesia drugs applied(r=0.596, P<0.05), ventilator uncoordinated(r=0.573, P<0.05) and else.

CONCLUSIONS 1.It is contributed to improving myocardial function, decrease the morbidity of postoperative complications, lower the mortality and shorten the length of ICU stay after inhibit the stress-induced hypertension of post Bentall or Improved-Cabrol cardiac surgery efficiently. 2. Multiple factors endangered the cardiac out break the stress-induced hypertension, such as pre-operation hypertension, sympathetic nervous system excitation cause of cardio-pulmonary bypass, inhibitory effect of central nervous system due to sedative and/or analgesia drugs applied in-operation, inadequate myocardial protection during intra-cardiac manipulation, myocardial ischemia reperfusion injury, acid-base balance and/or electrolyte balance disorder and some else factors.