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Acute Coronary Syndromes

TREATMENT OF REPERFUSION INJURY WITH RECOMBINANT ADAMTS13 IN A PORCINE MODEL OF ACUTE MYOCARDIAL INFARCTION

Poster Contributions
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Background: Angiographic no reflow and decreased microvascular perfusion after percutaneous coronary intervention increase morbidity and mortality in ST-elevation myocardial infarction patients. ADAMTS13 is a metalloprotease that cleaves von Willebrand factor, thereby reducing its prohemostatic properties. There is considerable evidence that ADAMTS-13 levels decrease and von Willebrand factor levels increase in STEMI patients. Recombinant ADAMTS13 has been effective in reducing cerebral infarct size in a murine model of stroke. In this study recombinant ADAMTS13 was tested as a potential treatment of no reflow in a porcine model of cardiac ischemia and reperfusion.

Methods: In 23 female swine (median age 83 days, median weight 30 kg) a balloon was inflated in the the circumflex coronary artery for 75 minutes. Fifteen minutes after reperfusion, an intracoronary bolus of either recombinant ADAMTS13 (400 U/kg, Baxter Innovations Vienna, Austria) or vehicle was given.

Results: ADAMTS13 activity significantly increased in treated pigs (median 18%, IQR 14.5-24.0 to median 324%, IQR 117.0-384.0, p= 0.003) whereas no change was observed in the control group. Animals were sacrificed seven days later for histopathology. There was no difference in the size of myocardial necrosis as assessed with plasma Troponin T measurements, continuous 12 leads ECG, macroscopical infarct analysis, and histopathology using phosphotungstic acid-hematoxylin staining. Microvascular obstruction as estimated by staining with anti-CD31/Hematoxylin and counting of vessels and microthrombi was similar for both groups.

Conclusions: Intracoronary treatment with recombinant ADAMTS13 did not prevent formation of microthrombi or decrease infarct size in this porcine coronary model of ischemia and reperfusion.