generic enoxaparin group. Hence, be careful to use generic enoxaparin in patients who had percutaneous coronary intervention.

Basic Science, Animal Models and Preclinical Studies (TCTAP A-146 to TCTAP A-149)

TCTAP A-146

MicroRNA-21 Expression and its Association with Leukocytes Infiltration on the Early Phase of Coronary Microembolization

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Background: MicroRNAs are small non-coding RNAs that regulate gene expression at the post-transcriptional level by either degradation or translational repression of a target mRNA. It has been demonstrated that miR-21 plays a critical role on inflammatory response after acute myocardial infarction. However, it is unknown about myocardial expression of miR-21 after coronary microembolization (CME) in mini-pigs.
Methods: Ten mini-pigs were enroll in this study, including sham-operation group (n=4) and CME group (n=6). Troponin T and interleukin-6 (IL-6) were detected on baseline, 6 hour and 7 days after CME. Myocardial expressions of miR-21 were detected by Realtime-PCR method. Myocardium specimens were embedded in paraffin for hematoxylin and eosin (HE) staining, while number of leukocytes infiltration were analyzed by Leica DFC 320 digital soft.
Results: Compared with sham-operation group, serum level of troponin T and IL-6 were increased significantly after 6 hours in CME group (Troponin T: 0.642±0.239 vs. 0.02±0.01 ng/ml, P<0.05; IL-6: 140.7±30.8 vs. 35.4±21.4 pg/ml, P<0.05). Leukocyte infiltration on microembolization associated myocardium was also increased markedly after CME. We found that myocardial expression of miR-21 was increased significantly at 7-day after CME (CME vs. sham: 3.72±1.51, P=0.033), which also had a positive relation (r=0.636, P=0.046) with average number of leukocytes infiltration on microembolization area.
Conclusion: Myocardial miR-21 expression was increased after coronary microembolization, which could be involved in the process of leukocytes infiltration.

TCTAP A-147

TongXinLuo Protects Human Cardiac Microvascular Endothelial Cells from Hypoxia/Reoxygenation Injury by Inducing Autophagy via the MEK/ERK Pathway

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Background: In contrast to cardiomyocytes, autophagy in cardiac microvascular endothelial cells (CMECs) during ischemia/reperfusion (I/R) injury has not been fully investigated. Tongxinluo (TXL), which is a traditional Chinese medicine formulation consists of extractions or powders from *Radix ginseng, Buthus martensi*, *Hirudo*, *Eupolyphaga seu steleophaga*, *Scolopendra subspinipes*, *Periostracum cicadae*, *Radix paeoniae rubra*, *Semen ziziphi spinosae*, *Lignum dalbergiae odoriferae*, *Lignum santali albi*, and *Borneolum syntheticum*, was previously demonstrated to be vascular protective.This study was designed to elucidate the role of autophagy and its regulatory mechanisms by TXL in CMECs subjected to I/R injury.
Methods: CMECs were exposed to varying concentrations of TXL solution for 30 min and subjected to hypoxia/reoxygenation (H/R) each for 2 h to determine the optimal working concentration. The autophagy inhibitor 3-methyladenine (3-MA), the autophagy promoter rapamycin, and the MEK inhibitor PD98059 were used to further investigate the role and the modulatory mechanism of autophagy in CMECs.
Results: The results indicated that H/R significantly induced autophagy, as identified by an increased number of monodansylcadaverine (MDC)-positive CMECs, increased autophagosome formation, and a higher type II/type I of light chain 3 (LC3-II/LC3-I) ratio ($p < 0.05$), but not Beclin-1 expression. The inhibition of autophagy using 3-MA was found to be proapoptotic whereas the induction of autophagy by rapamycin was antiapoptotic, which was reflected by index such as flowcytometric apoptotic rates, expression of Bcl, Bax, and Cytochrome *c* ($p < 0.05$). TXL enhanced autophagy and decreased apoptosis in a dose-dependent manner. TXL reached its largest anti-apoptotic effect in CMECs at 800 μg/mL (Mean ÷ SEM, 9.92 ÷ 0.49% in the 800 μg/mL TXL group vs. 21.04 ÷ 1.11% in the H/R group, $p < 0.05$), and MDC-positive cell rate of the 800 μg/mL TXL group was significantly higher than that of the H/R group ($p < 0.05$). 3-MA attenuated the TXL-promoted autophagy and antiapoptotic effects ($p < 0.05$), whereas rapamycin failed to cause additional effects in comparison to TXL alone. TXL upregulated the phosphorylation of MEK and ERK, but ERK phosphorylation was abrogated by PD98059, which also decreased autophagy and increased apoptosis in comparison to TXL alone.
Conclusion: These results suggest that autophagy is a protective mechanism in CMECs subjected to ischemia/reperfusion injury and that TXL can promote autophagy via activation of the MEK/ERK pathway.

TCTAP A-148

Left Ventricular Remodeling with Preserved Function After Coronary Microembolization: The Effect of Methylprednisolone

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Background: The objective of this study was to evaluate changes in left ventricular ejection fraction (LVEF) and left ventricular remodeling after coronary microembolization (CME) and to investigate the effects of methylprednisolone (MTP) on these processes.
Methods: CME was induced by injection of microspheres (42 μm Dynospheres) into left anterior descending artery of mini swine. The animals were divided into two groups. Group 1 (n=9) received 120,000 microspheres and Group 2 (n=7) received 120,000 microspheres following intravenous administration of 30 mg/kg MTP.
Contrast enhanced MRI was performed at baseline, 6 h after intervention and 1 week later.
Results: In Group 1, LVEF was significantly decreased at 6 h but recovered 1 week This was accompanied by continuing left ventricular remodeling. In Group 2, LVEF remained unchanged at all assessment times. LVEF measured at 6th hour and at one week after CME in group 1 and group 2 was 0.39±0.06 and 0.44±0.04, and 0.44±0.04 and 0.48±0.03 respectively (Both P=NS). Hyperenchancement at the anterior wall of the left ventricle was shown by MRI at 6 h in Group 1 but not in Group 2. The hyper-enhanced area in Group 1 was 7.77±1.49% of left ventricular mass.
Conclusion: The consequence of CME is left ventricular dilation with preserved LVEF. Pretreatment with MTP appears to have a cardioprotective effect on left ventricular remodeling.

TCTAP A-149

Study of the Prognostic Importance of Albumin/Creatininee Ratio in Patients with Chronic Heart Failure

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Background: Increased excretion of albumin in urine is an established risk factor for mortality, cardiovascular events, and adverse renal outcomes in the general population. The mechanism underlying albuminuria in patients with HF without DM and HTN is not known. It may be due to renal congestion or hemodynamic disturbance.
The prevalence and prognostic importance of elevated urinary albumin creatinine ratio in heart failure are not known. We aimed to study the prognostic importance of albumin / creatininee ratio in non-diabeticpatients with systolic heart failure.
Methods: 50 patients diagnosed as systolic heart failure with evidence of LV systolic dysfunction (LV ejection fraction <40%) by echocardiography were recruited from Nasr City insurance hospital cardiology department from October 2011 till December 2011 and were followed up till june 2012. Patients were subjected to full history taking, detailed clinical examination, echocardiography, assessment of serum creatininee and Urinary albumin creatininee ratio. Patients with pure diastolic dysfunction, patients who suffered from another serious disease with a poor prognosis, abnormal kidney functions (creat> 1.5 mg/dl & e GFR < 60ml/min), diabetes were excluded. Patients were followed up after six months for echocardiography, Urinary albumin creatininee ratio, kidney functions and clinical follow up mortality and morbidity,readmission either due to heart failure or due to occurrence of complications such as ventricular tachycardia, AF, cerebrovascular strokes, anemias, development of renal or hepatic impairment, development of electrolyte imbalance and need for interventions, ICD or multiple pacing.
Results: Patients were divided into three groups according to their base line urinary albumin creatinine ratio, as group I normo-albuminuric where urinary albumin creatinine ratio <2.5 mg/mmol in men and <3.5 mg/mmol in women, group II micro-albuminuric where urinary albumin creatinine ratio 2.5 to 25.0 mg/mmol in men and 3.5 to 25.0 mg/mmol in women, group III urinary albumin creatinine ratio > 25.0 mg/mmol in men and women.
There were no significant changes among the three groups regarding heart rate, systolic blood pressure and diastolic blood pressure, medical history, and medications.
A significant positive relation was found between the 3 groups as regards the event rate of MACE as group III had the higher urinary albumin creatinine ratio.
NYHA class was significantly worse in group III which had more elevated urinary albumin creatinine ratio.
Follow up serum creatininee was significantly higher and ejection fraction was significantly lower in groups II, III than in group I. And all the three groups showed significant increase in urinary albumin creatinine ratio.
The cutoff value is > 2.26 the value above which we have to give more attention to patient's clinical status and pay more attention to treatment compliance and optimization of medical treatment, urinary albumin creatinine ratio above this value considered of more risk to develop cardiovascular and renal complications in non diabetic patients with sensitivity of 100% and specificity 92.6% where positive predictive value 92% and negative predictive value 100%.
Conclusion: Increasing urinary albumin creatinine ratio was associated with an increased hazard of adverse clinical outcomes, showing that urinary albumin excretion represents a continuous measure of risk.

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