75.7% patients had hypertension history and 33.0% patients had hypokalemia history. Forum: The patients with arterial hyperplasia had hypokalemia history than that without arterial hyperplasia (39.2% vs. 20.7%, P < 0.001). The frequency of CC genotype of rs3740835 in KCNJ5 gene was higher in control group rather than that in arterial hyperplasia group (67.5% vs. 58.2%, P = 0.047), though the significance only reached the borderline. (3) Logistic regression model was constructed to explore the potential factors that related to CT scan-based arterial hyperplasia in whole study population. The results showed that the rs3740835 in KCNJ5 gene (0.642 (0.424-0.971), P = 0.036) as well as plasma potassium [0.432 (0.261-0.715), P<0.01] played protective roles in arterial hyperplasia, adjusted for body mass index, gender, age. Conclusion: The rs3740835 variants in KCNJ5 gene as well as plasma potassium levels were contributing factors to arterial hyperplasia in patients with PA from Xinjiang.

GW25-e5201
The mechanism research of Compound Danshen dripping pills accruing myocardial infarction from the TLR4-NF-κB - PECAM-1 pathways
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Objectives: To observe the protection and its mechanism of Compound Danshen dripping pills on myocardial infarction.

Methods: (1) Eight Wistar rats were randomly divided into normal group, model control group, experimental model group, isosorbide dinitrate (ISD) group, Compound Danshen (FFDS) group, 10 rats in each group. The myocardial infarction models and the model of myocardial infarction with the Q and blood stasis were established respectively, and the models rats were treated with isosorbide dinitrate and Compound Danshen dripping pills intervention. Cardiac ejection fraction, myocardial infarction, GMP-140 of blood serum and TLR4, TRAF-6, IL-1 protein and NF-κB gene level of FFDS group was lower than that in model group (P<0.05). Immunohistochemical results shown TRL4, TRAF-6, IL-1 protein and NF-κB pathway.

Results: After NBT staining, myocardial tissue in the normal group were stained purple; There were large grey infarcted region in myocardial tissue of each model group. Immunohistochemical results shown TRL4, TRAF-6, IL-1 protein and NF-κB pathway. (2) GMP-140 of blood serum of FFDS group were lower than that of model group (P<0.01); isosorbide dinitrate and Compound Danshen dripping pills intervention, cardiac ejection fraction, myocardial infarction, GMP-140 of blood serum and TLR4, TRAF-6, IL-1 protein and NF-κB protein expression and mRNA, and inhibited the cholesterol efflux induced by AngII, by liquid scintillator.

Conclusion: The effect of Compound Danshen dripping pills on myocardial protection may be related to regulation of TLR4-NF-κB - PECAM-1 pathways.

GW25-e5348
Effects of Simvastatin combined with ezetimibe on atherosclerosis in ApoE-/- mice
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Objectives: To investigate effects of Simvastatin combined with ezetimibe on atherosclerosis in ApoE-/- mice with high-fat diet.

Methods: 36 male apoE-/- mice (age, 8weeks) on a C57BL/6J background were randomly divided into three groups with 12 animals in each group. Animals were treated with intragastric administration as follows: Model group received PBS buffer per day for 8 weeks; Simvastatin group received simvastatin (20mg/kg intragastric administration) every day for 8 weeks; Combination therapy group were treated with Simvastatin and ezetimibe (10mg/kg ezetimibe and 20 mg/kg simvastatin every day for 8 weeks. Then the whole aorta mice were collected for oil red O staining use. The mechanisms of homocysteine-mediated lipid disorder are poorly understood. Human monocytic U937 cells were infected with a shRNA lentiviral vector library targeting human RTKs upon LPS stimuli and PTX-3 expression was determined by ELISA analysis. The involvement of downstream signaling in the regulation of PTX-3 expression was analyzed by both Western blotting and ELISA assay.

Results: We found that knocking down of ErbB2/3, EphA7, and FGFR3 and RET impaired PTX-3 expression without effects on cell growth or viability. Moreover, inhibition of AKT, the downstream effector of ErbB2/3, also reduced PTX-3 expression. Furthermore, we showed that FGFR3 inhibition by anti-cancer drugs attenuated p38 activity, in turn induced a reduction of PTX-3 expression.

Conclusions: Overall, our study demonstrates the role of RTKs in the regulation of PTX-3 expression and uncovers a potential cardiotoxicity effect of RTK inhibitor infusions. To our knowledge, cancer patients who have symptoms of atherosclerosis are at the risk of atherosclerosis.

Effects of AngII and Ang-(1-7) on the Cholesterol Efflux in THP-1 Derived Foam Cells
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Objectives: To investigate the effects of angiotensinII (AngII) and angiotensin-(1-7) [Ang-(1-7)] on scavenger receptor class B type1 (SR-BI), ATP-binding cassette transporter Al (ABCAl) and cholesterol efflux in THP-1 derived foam cells. The Center of Cardiology, Navy General Hospital

Methods: Human monocytic U937 cells were infected with a shRNA lentiviral vector library targeting human RTKs upon LPS stimuli and PTX-3 expression was determined by ELISA analysis. The involvement of downstream signaling in the regulation of PTX-3 expression was analyzed by both Western blotting and ELISA assay.

Results: We found that knocking down of ErbB2/3, EphA7, and FGFR3 and RET impaired PTX-3 expression without effects on cell growth or viability. Moreover, inhibition of AKT, the downstream effector of ErbB2/3, also reduced PTX-3 expression. Furthermore, we showed that FGFR3 inhibition by anti-cancer drugs attenuated p38 activity, in turn induced a reduction of PTX-3 expression.

Conclusions: Overall, our study demonstrates the role of RTKs in the regulation of PTX-3 expression and uncovers a potential cardiotoxicity effect of RTK inhibitor infusions. To our knowledge, cancer patients who have symptoms of atherosclerosis are at the risk of atherosclerosis.

GW25-e5399
A RTK-based functional RNAi screen reveals determinants of PTX-3 expression
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Objectives: To observe the protection and its mechanism of Compound Danshen dripping pills on myocardial infarction.

Methods: (1) Eight Wistar rats were randomly divided into normal group, model control group, experimental model group, isosorbide dinitrate (ISD) group, Compound Danshen (FFDS) group, 10 rats in each group. The myocardial infarction models and the model of myocardial infarction with the Q and blood stasis were established respectively, and the models rats were treated with isosorbide dinitrate and Compound Danshen dripping pills intervention. Cardiac ejection fraction, myocardial infarction, GMP-140 of blood serum and TLR4, TRAF-6, IL-1 protein and NF-κB gene level of FFDS group was lower than that in model group (P<0.01). Immunohistochemical results shown TRL4, TRAF-6, IL-1 protein and NF-κB pathway.

Results: After NBT staining, myocardial tissue in the normal group were stained purple; There were large grey infarcted region in myocardial tissue of each model group. Immunohistochemical results shown TRL4, TRAF-6, IL-1 protein and NF-κB pathway.

Conclusion: The effect of Compound Danshen dripping pills on myocardial protection may be related to regulation of TLR4-NF-κB - PECAM-1 pathways.

GW25-e3426
Homocysteine impairs macrophage cholesterol efflux via LXR alpha hypermethylation
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Objectives: The mechanisms of homocysteine-mediated lipid disorder are poorly understood. Liver X receptors alpha, as a cholesterol-sensing nuclear receptors, are the key regulators of macrophage cholesterol efflux. This work aimed to explore the methylation modification mechanisms of LXR alpha in homocysteine impairing cholesterol efflux in THP-1 macrophage.
Conclusions:
MDA has significantly decreased (P<0.05) when HUVECs is injured with 0.6mmol/L Hcy for 24 hours (P<0.05), while pretreated with rosuvastatin was found to protect mtDNA against oxidative damage. Our recent study indicates lycopene significantly attenuated the I/R-injury in the heart. 

Methods: We established I/R-injury model with rat in vivo and we also established hypoxia/reoxygenation-injury model with H9c2 cells to simulate I/R-injury in vitro. Reactive oxygen species (ROS) and mitochondrial superoxide levels were determined. Mitochondrial 8-hydroxyguanine (8-OHdG), mtDNA content and mtDNA transcript levels were detected to find out if mtDNA were damaged; the protein expression of mitochondrial transcription factor A (Tfam) in mitochondrial, a key protein for mtDNA transcription, replication and component for nucleoid organization were also determined by western blot.

Results: I/R significantly increased reactive oxygen species (ROS) production and mitochondrial superoxide levels. In addition, I/R increased mitochondrial 8-hydroxyguanine (8-OHdG), mtDNA content and mtDNA transcript levels. Consistent with these findings, I/R was found to decrease the protein expression of Tfam in mitochondrial. Lycopene pretreatment efficiently attenuated the oxidative damage to mtDNA induced by I/R both in vivo and in vitro.

Conclusions: Our results suggest that mtDNA damage may account for I/R-injury. Lycopene has a great pharmacological potential in protecting mtDNA against the injury to the heart.

GW25-e0274
A polymorphism rs16164913 in pre-microRNAs may be associated with atrial fibrillation in Han Chinese population

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GW25-e0601
Up-regulation of neuronal nitric oxide synthase modulates myocardial Ca2+ sensitivity and controls left ventricular myocyte contractility in hypertensive rats

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Objectives: Hyperension is one of the major risk factors for developing cardiac hypertrophy and heart failure. Although cellular signaling pathways, intracellular Ca2+ handling and myofilament Ca2+ sensitivity are known to be altered in hypertensive myocardium, mechanisms mediating left ventricular (LV) contractile function remain to be defined. It is known that neuronal nitric oxide synthase (nNOS) is up-regulated in hypertensive myocardium, however, its role in myocyte Ca2+ handling and myofilament Ca2+ sensitivity and their interplays in regulating contractile function is not clear. Therefore, we aim to test the functional regulation by nNOS of LV myocyte contraction in angiotensin II (Ang II)-induced hypertensive rats.

Methods: Sprague-Dawley rats (8 weeks old, male) were subjected to Ang II infusion subcutaneously using osmotic mini-pump for 4 weeks (125ng/mkg). These animals were paired with sham-operated groups. LV myocytes were isolated using a standard enzymatic dispersion technique. Contraction and relaxation were measured in LV myocytes (field-stimulation at 2 Hz, 36±1°C) by using a video-sarcomere detection system (IonOptix Corp).

Results: Systemic blood pressure was elevated in Ang II-treated rats (osmotic mini-pump, 4 weeks) compared to that in sham. Functional analysis showed that LV myocyte sarcomere shortening was unchanged and relaxation was faster in Ang II-rats compared to those from shams. L-type Ca2+ channel activity (lCa) was not different between two groups, however, peak Ca2+ (lCa) was increased (Fura 2, F350/380) and Ca2+ L-channel current (ICa) was significantly increased (optical density of nNOS/GAPDH) in LV myocyte homogenates from Ang II-rats. Inhibition of nNOS with specific inhibitors, S-methyl-L-thiocitrulline (SMTCT, 100 nM) or N-(5-Imidazole-3-butenyl)-L-ornithine (L-VNIO, 100 uM) restored myocardial Ca2+ sensitivity and reduced [Ca2+]i in Ang II-rats. A specific inhibitor of protein kinase G (KT 8882, 1 uM) mimicked the effect of nNOS and increased myocardial Ca2+ sensitivity, [Ca2+]i and LV myocyte contraction.

Conclusions: These results collectively suggest that reduced myocardial Ca2+ sensitivity by nNOS plays predominant role in controlling intracellular Ca2+ handling and LV myocyte contractility in hypertension.