75.7% patients had hypertension history and 33.0% patients had hypokalemia history. 

Forty patients with adrenal hyperplasia had hypokalemia history than that without adrenal hyperplasia (39.2% vs. 20.7%, P <0.001). The frequency of CC geneotype of rs3740835 in KCNJ5 gene was higher in control group rather than that in adrenal 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 adrenal hyperplasia in whole study population. The results showed that the rs3740835 in KCNJ5 gene [O.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 adrenal hyperplasia, adjusted for body mass index, gender, age.

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: 100 male Wistar rats were randomly divided into four groups: Control group, experimental model group, isosorbide dinitrate (ISD) group, Compound Danshen (FFDS) group. The rats in each group were treated with ox-LDL to establish myocardial infarction model. Cardiac ejection fraction, myocardial infarction, GMP-140 of blood serum and TRL4, TRAF-6, IL-1β gene expression of myocardial tissue were observed.  

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. There were smaller gray infarction area than ISD group and FFDS group. The mRNA and protein expression of SR-BI, ABCA1 were determined. Compared with the control group, AngII decreased SR-BI and ABCA1 in both AngII group, Ang-(1-7) group, Ang-(1-7) +AngII group and AngII+Ang-(1-7) +A-779 group. The mRNA and protein expression of SR-BI, ABCA1 were determined by RT-PCR and Western blot, the intracellular cholesterol efflux rate was detected by liquid scintillator.  

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: Altogether, our study demonstrates the role of RTKs in the regulation of PTX-3 expression and uncovers a potential cardiototoxicity effect of RTK inhibitor in cancer patients who have symptoms of atherosclerosis or are at the risk of atherosclerosis.

GW25-e5399  

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 type 1 (SR-BI), ATP-binding cassette transporter A1 (ABC1A) and cholesterol efflux in THP-1 derived foam cells.  

Methods: Human monocytic cell line (THP-1) were induced into macrophages by 100nmol/L phorbol myristate acetate (PMA) for 48h, and treated with ox-LDL to construct the foam cells, then were randomly allocated into five groups: control group, AngII group, Ang-(1-7) group, Ang-(1-7) +AngII group and AngII+Ang-(1-7) +A-779 group. The mRNA and protein expression of SR-BI, ABCA1 were determined by RT-PCR and Western blot, the intracellular cholesterol efflux rate was detected by liquid scintillator.  

Results: Compared with the control group, AngII decreased SR-BI and ABCA1 in both protein and mRNA, and inhibited the cholesterol efflux (P <0.05). Those effects could be attenuated by cotreated with Ang-(1-7) (P <0.05). However when incubated with A-779, an inhibitor of Ang-(1-7), the effects of Ang-(1-7) on promoting the expression of SR-BI, ABCA1 and the cholesterol efflux were significantly abolished (P <0.05).  

Conclusions: In THP-1 derived foam cells, Ang-(1-7) via its specific receptor MAS attenuates the reduction of the expression of SR-BI and ABCA1 induced by AngII, and increases the cholesterol efflux.

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.