Objectives: The reduction of myocardial matrix metalloproteinase-2 (MMP-2) in early hypertension has been shown that may lead to collagen accumulation in extra celliar matrix (ECM) and myocardial fibrosis. CD147 is a natural inducer of MMP-2. We assume that the inhibition of CD147/MMP-2 pathway may contribute to this remodeling. So we intend to alter the level of CD147/MMP-2 in early hypertensive rats by injecting exogenous CD147 protein, and observe its effect on ventricular remodeling.

Methods: 30 healthy eight weeks male spontaneously hypertensive rats (SHR) were divided into 3 groups: SHR group (n=10): normal saline via tail vein injection iv. qd; CD147 group (n=10): CD147 600ng kg⁻¹ iv. qw; CD147+DOX group (n=10): CD147 600ng kg⁻¹ iv. qw and DOX (doxycycline, broad-spectrum inhibitor of MMPs) 30 mg kg⁻¹ iv qd via gavage. 10 healthy eight weeks male Wistar-Kyoto rats (WKY) were treated as the control group (WKY group, n=10). Left ventricular mass index (LVWI= LV mass (mg)/weight (g)) were measured and calculated; Myocardial sections were observed by HE coloration, VG coloration; Collagen volume fractions (CVF) were obtained by image analysis; The proteins of MMP-2, TIMP-1 and CD147 in myocardial tissue were assessed by western blot after the rats were sacrificed in Day 36th.

Results: The contents of CD147 protein in myocardium of SHR group were reduced significantly (P<0.05) and the contents of MMP-2 were also reduced slightly compare with those in WKY group (P>0.05); The abundance of CD147 and MMP-2 protein in CD147 group was increased obviously (P<0.05), however, that of MMP-2 protein was reduced in CD147+DOX group after DOX intervention (P<0.05); The levels of TIMP-1 in the myocardium of three SHR groups, particularly in the CD147 group, were significantly higher than WKY group (P<0.05); The ratios of myocardium MMP-2/TIMP-1 in SHR were less than WKY's obviously (P<0.05), but were increased significantly after CD147 intervention. LVWI's of SHR group were higher than WKY group (P<0.05), but they were downregulated obviously in CD147 group and CD147+DOX group (P<0.05). The myocardial biopsy showed that there were cardiac myocyte hypertrophy, some myocardial fibre rupture, myocyte dissolution and fuzzy myocardial fibre boundaries in SHR group . The cardiac structure was improved significantly after CD147 intervention, but it was surprised when DOX administrated simultaneously. VG coloration sections were seen more abundant collagen fibres and higher CVF in the myocardiums of SHR group than those in WKY group (P<0.05). The contents of collagen fibre and the CVFs were decreased significantly after CD147 intervention (P<0.05). While, they were increased again in CD147+DOX group (P<0.05).

Conclusions: The inhibition of CD147/MMP-2 pathway, the reduction of MMP-2 protein expression and the imbalance of MMP-2/TIMP-1 may contribute to early ventricular remodeling in SHR. Appropriate modulation of CD147 protein up-regulation may attenuate the process of early hypertensive ventricular remodeling.

GW25-e1393

Relationship between platelet count and the concentration of potassium

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Objectives: To study the relationship between the platelet counts (PLT) and the concentration of potassium in both serum and plasma.

Methods: 298 subjects with different PLT were enrolled in this study. Subjects with other possible causes of elevated serum potassium were excluded. PLT was counted by 3700XFX. The serum and plasma potassium were measured on Vitros 5.1FS automatic biochemical analyzer with a electrode method.

Results: Our study subjects includes 46 cases of patients with thrombocytopenia (PLT<300×10⁹/L), 90 cases of patients with normal platelet (PLT 100×10⁹/L–300×10⁹/L), and 82 cases of patients with thrombocytopenia (PLT<100×10⁹/L) respectively. The concentration of potassium in serum is higher than plasma in all three groups. Serum potassium in thrombocytopenia group is 4.48±0.91 mmol/L, which is significantly higher than thrombocytopenia group with a concentration of 4.02±0.84 mmol/L, while potassium plasma showed little difference. The difference of serum potassium and plasma potassium is positively correlated with PLT in patients with thrombocytopenia. The regression equation is Y (difference of serum potassium and plasma potassium) = 0.0015 (PLT×10⁹/L)−0.1449, which indicates that an increase of 1000×10⁹/L in the blood PLT would cause an increase of about 1.4 mmol/L in the serumpotassium concentration (P<0.01, r=0.945).

Conclusions: Serum potassium is positively correlated with PLT in the blood sample, which indicates that the clinician should be aware of pseudohyperkalemia due to the increased PLT. Potassium should be measured from plasma or a PLT based correction should be applied in such cases.

GW25-e3228

Beta-3-Adrenergceptor Stimulation Protects Against Atherosclerosis in Apopliprotein E-Deficient Mice

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Objectives: β3-Adrenergceptors (β3-AR) protects against the progression of atherosclerosis. However, the specific mechanism of this antiatherosclerotic effects is still not clear. Thus, the aim of this study was to determine the molecular basis of the anti-atherosclerotic effects.

Methods: Male homozygous apopliprotein E knockout (ApoE−/−) mice on a high-fat diet and wild-type (WT) C57BL/6J mice on a normal diet were used. Fifty ApoE−/− mice were randomized into five treatment groups: atherosclerotic model, atorvastatin, low-dose β3-AR agonist, high-dose β3-AR agonist and β3-AR antagonist groups. Serum lipids were measured when the treatments ended. mRNA expressions in liver were measured by quantitative real-time PCR. Protein expressions of ApoA-1 and SR-B1 were detected by quantitative real-time PCR. Protein expressions of ApoA-1 and SR-B1 in the livers were determined by western blot analysis.

Results: Compared with ApoE−/− control mice, chronic β3-AR agonist treatment significantly increased plasma high-density lipoprotein cholesterol levels. Compared with the age-matched WT mice, the ApoA-1 mRNA and protein expression level in ApoE−/− mice were significantly increased. Compared with ApoE−/− control mice, the ApoA-1 mRNA and protein expression level in liver were significantly increased in the atorvastatin and β3-AR agonist groups. The SR-B1 mRNA expression and protein level in liver of ApoE−/− mice was significantly decreased compared with wild type mice. The SR-B1 mRNA expression and protein in liver were significantly increased in the atorvastatin and β3-AR agonist groups, compared with the ApoE−/− atherosclerotic model mice.

Conclusions: The present study demonstrated that long-term β3-AR activation can regulate lipid metabolic disorders, and reduced progression of atherosclerosis. This effect may be related to ApoA-1 and SR-B1.