Methods: 150 cases of coronary heart disease (CHD) from department of cardio-logic medicine in our hospital were collected as CHD group, including 50 cases with stable angina pectoris, 50 cases with unstable angina pectoris, and 50 cases with acute myocardial infarction; and 80 healthy adults were selected as control group. The level of serum homocysteine in four groups were measured by chemiluminescent microparticle immunosassay (CMIA). The data were analyzed by SPSS 19.0.

Results: The levels of serum Hcy in patients with coronary heart disease [(23.61±8.53) μmol/L] were higher than those in the control group [ (19.62±2.16) μmol/L], the differences were statistically significant (P<0.05). In CHD group, the levels of serum Hcy in patients with acute myocardial infarction [(33.02±9.13) μmol/L] were higher than those in the unstable angina pectoris group [ (20.74±7.68) μmol/L]; In addition, the levels of serum Hcy in the patients with unstable pectoris angina were higher than those in the stable angina pectoris [(14.38±5.20) μmol/L], and the differences were statistically significant (P<0.05).

Conclusions: Hcy is an independent risk factor in cardiovascular diseases, and its expression level can be used as predictors to prompt the severity and type of coronary heart disease. Detection on serum level of Hcy has the important clinical value on diagnosis and treatment of cardiovascular diseases.

GW25-e5179

Effect of high salt on the proliferation of rat vascular smooth muscle cells and the intervention of capsaicin

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Objectives: High salt induces the proliferation of VSMCs, but the mechanism remains unclear. This study examined the influence of high salt on the proliferation of rat vascular smooth muscle cells (VSMCs) and the intervention of capsaicin in vitro.

Methods: VSMCs were obtained from the rat’s thoracic aorta by tissue adherent method and were cultured in a 95% O2 and 5% CO2 atmosphere at 37°C in DMEM supplemented with 10% FBS. VSMCs were identified by immunocytochemical staining with antibody against α-smooth muscle actin. After synchronization for 24h, VSMCs plated into 96-well plates at a density of 5×10^4 were randomly divided into control group (sodium 139mmol/L), high-salt group (sodium 159mmol/L) and capsaicin group (capsaicin 10μmol/L in sodium 159mmol/L), then cultured for 72h. The effect of high salt and capsaicin on the proliferation of VSMCs were evaluated by MTT assay. The cell cycle were detected by flow cytometry technique. VSMCs were stained with hematoxylin and eosin, then the area of each cell cytoplasm and cell nucleus were measured. The expressions of TGF-β1, P-Smad2/3, Smad7 were determined by immunocytochemical staining and the mRNA expressions of TGF-β1, Smad2, Smad3, Smad7 were detected by real time RT-PCR.

Results: Compared with control group, high salt significantly promoted VSMCs proliferation, the cell proliferation rate in the high salt groups were remarkably higher (P<0.05), the percentage of S phase of cell cycle was increased (P<0.05). On the contrary, the percentage of G0/G1 phase of cell cycle was decreased (P<0.05). HE dyeing shows that cell area, cytoplasm area and nuclear area of the VSMCs in the high salt group evidently increased, while the ratio of nuclear area to cytoplasm area [(4.38±5.20) μmol/L] were higher than those in the unstable angina pectoris group [(20.74±7.68) μmol/L]; In addition, the levels of serum Hcy in the patients with unstable pectoris angina were higher than those in the stable angina pectoris [(14.38±5.20) μmol/L], and the differences were statistically significant (P<0.05).

Conclusions: Hcy is an independent risk factor in cardiovascular diseases, and its expression level can be used as predictors to prompt the severity and type of coronary heart disease. Detection on serum level of Hcy has the important clinical value on diagnosis and treatment of cardiovascular diseases.

GW25-e5146

High-salt diet decreases ACE2 and activates TGF-β1/Smads signaling pathway in vascular remodeling

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Objectives: Angiotensin-converting enzyme 2 (ACE2) has been reported to be protective effect of vascular injury in hypertension, but the mechanism remains unclear. This study focused on the effects of ACE2 on vascular remodeling and determined whether or not these effects of ACE2 were dependent upon the TGF-β1/Smads signaling pathway.

Methods: Male Wistar rats (3-4 weeks of age) were given 4% high salt diet (HSD; n=34) for 24 weeks, whereas their age-matched controls (n=10) received normal salt diet (NS; 0.5% NaCl). In a subgroup of HSD rats (n=10), telmisartan was given at a dose of 1 mg/kg per day as a solution mixed with autologous artificial gastric juice. The medial thickness and collagen deposition of aorta were observed by HE and Masson staining, respectively. The protein expression of ACE, ACE2, TGF-β1, P-Smad2/3 and Smad7 in aorta were quantified by Western blotting.

Results: After 24 weeks, HSD caused hypertension. HE and Masson staining showed high-salt diet increased medial thickness and collagen deposition of aorta as compared with normal group. Compared with NS group, the protein levels of TGF-β1 and P-Smad2/3 were increased and Smad7 decreased in HSD group (P<0.05). Exposure to high salt intake increased ACE protein level (P<0.05), but decreased vascular ACE2 protein level (P<0.05). Compared with HSD group, telmisartan could improve vascular remodeling induced by high-salt diet, increase ACE2 and Smad7 protein expression and decrease ACE, TGF-β1 and P-Smad2/3 protein levels.

Conclusions: This study suggests that the adverse vascular effects of excessive salt intake may result from the activation of TGF-β1/Smads and the decrease of ACE2. The beneficial effect of telmisartan may be attributed, at least in part, to the increase of ACE2 expression and inactivation of vascular TGF-β1/Smads.