[gw22-e0957]

LEVELS OF APOA-I, APOA-IV AND SAA BOTH IN PLASMA AND HIGH-DENSITY LIPOPROTEIN IN CORONARY HEART DISEASE PATIENTS

Yeping-Zhang, Zhimin-Xu, Hong-Liu, Guoliang-Chen, Yiling-Huang, Bin-Duan, Yishi-Li *Key Laboratory of Clinical Trial Research in Cardiovascular Drugs, Ministry of Health, Cardiovascular Institute and Fuwai Hospital, Peking Union Medical College and Chinese Academy of Medical Sciences, Beijing, China*

10.1136/heartjnl-2011-300867.698

Background In recent years, studies have shown that elevated level of HDL may not have a cardiovascular protective effect. Our study is to focus on the protein components in HDL with CHD, and to provide a useful laboratory base for the pathogenesis of CHD and early diagnosis.

Objectives To investigate the differences of apolipoprotein A-I (apoA-I), apolipoprotein A-IV (apoA-IV) and serum amyloid A (SAA) found in Proteomics-2D-DIGE method earlier, both in HDL and plasma between the control group and CHD in a larger group.

Subjects and methods (1) 153 hospitalised patients will be selected into control group (n=74) and coronary heart disease (n=79), collected basic clinical data and biochemical indicators. (2) Collect fasting venous blood (EDTA anticoagulant), and ultracentrifugate HDL from plasma. (3) Obtain the levels of apoA-I, apoA-IV and SAA both in plasma and HDL, using a ELISA method. (4) Statistical methods: use a SPSS17.0 statistical package; normal data is expressed in x±SD, and independent sample t test, $p<0.05$ was considered statistically significant.

Results (1) The composition of HDL had changed in patients with CHD, the level of apoA-I were lower than the control group (p=0.000), the SAA is higher (p=0.000), while the apoA-IV did not change significantly (p=0.540). (2) The plasma level of SAA in CHD patients was higher than the control group (p=0.024), which was the same in HDL. In addition, the plasma level of SAA in younger CHD patients increased more significantly. (3) The plasma level of apoA-I in CHD did not change significantly (p=0.308), while the apoA-IV level was significantly higher (p=0.000), both of which were inconsistent with the changes in HDL.

Conclusion (1) Compared with the control group, the composition of HDL had changed in CHD patients. ApoA-I level decreased and SAA level increased significantly, while the apoA-IV level did not change significantly. These changes in HDL may have a close relationship with CHD. (2) The level of SAA with CHD was consistent in HDL and plasma, both were higher than the control group. The plasma SAA level in younger patients with CHD increased more significantly. SAA may be a risk factor in early-onset CHD. (3) The level of apoA-I in HDL with CHD was lower significantly, while the plasma level did not change significantly, indicating that the change of apoA-I in HDL may be more sensitive than in plasma. (4) The plasma level of apoA-IV was significantly higher than the control group, which is inconsistent with some previous studies. Further study is needed to explore new risk factor for CHD.