amplified when elevated VLDL cholesterol was combined with elevated LDL cholesterol and/or the presence of major CVD risk factors.

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e0305 THE EFFECT OF PROFILIN-1 ON VASCULAR INJURY CAUSED BY ADVANCED GLYCATION END PRODUCTS

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Changlu Wang, Tianlun Yang. *First Affiliated Hospital of Xiangya, Central South University in Hunan*

Objective The aims of this study were to explore the effect of Profilin-1 on vascular injury caused by advanced glycation end products, So as to provide a new therapeutic approach with diabetic vascular complications.

Methods Human umbilical vein endothelial cells were incubated with different concentrations of AGEs-BSA ($50 \text{ mg} \cdot \text{L}^{-1}$, $100 \text{ mg} \cdot \text{L}^{-1}$, $200 \text{ mg} \cdot \text{L}^{-1}$) for various periods of time (6–24 h). The levels of ADMA and NO in the conditioned medium, the protein expression of Profilin-1 for cells were determined.

Results AGEs-BSA increased the protein expression of Profilin-1 and ADMA in a concentration and time-dependent manner. Incubation with high concentration glucose (30 mmol/L) for 24 h elevated the levels of NO, and AGEs-BSA ($200 \text{ mg} \cdot \text{L}^{-1}$) decrease the levels of NO. AGEs-BSA ($200 \text{ mg} \cdot \text{L}^{-1}$) could decrease the levels of NO in the conditioned medium, the difference were significant after 24 h.

Conclusion Profilin-1 may be involved in “metabolic memory” induced by the advanced glycation end products.

e0306 SERUM CONCENTRATIONS OF RESISTIN AND ADIPONECTIN IN CASES WITH IMPAIRED GLUCOSE TOLERANCE

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Xiao-Ping Li, Guo-Hua Yuan, Xing-Yu Gao. *Department of Cardiology, The First Affiliated Hospital of Guangxi Medical University*

Objective Recent studies indicated that resistin and adiponectin take some roles in the glucose homeostasis and suggested there were associations among resistin, adiponectin and glucose tolerance. Therefore we investigate the serum levels of resistin and adiponectin in impaired glucose tolerance (IGT) cases in the present study.

Methods 82 subjects were included, divided into IGT group (27 cases) and normal glucose tolerance (NGT) group (55 subjects) based on the oral glucose tolerance test. Body mass index (BMI), waist circumference, blood pressure, fasting lipids, glucose, insulin and C-peptide were measured, and adipocytokines such as resistin, adiponectin, leptin, TNF- α , interleukin-6 (IL-6) and C-response protein (CRP) were also examined, insulin resistance was assayed by the homeostasis model assessment of insulin resistance (HOMA-IR) formula.

Results There were no differences in concentrations of resistin and adiponectin between IGT group and NGT group. Pearson relation analysis showed that serum resistin concentrations were positively correlated with age ($r=0.482$, $p<0.05$) and BMI ($r=0.389$, $p<0.05$), and serum adiponectin concentrations were positively correlated with HDL-c ($r=0.524$, $p<0.01$) and female ($r=0.437$, $p<0.05$), but negatively correlated with TNF- α ($r=-0.437$, $p<0.05$) in IGT cases. There were no correlations among resistin, adiponectin with fasting glucose, glucose tolerance and HOMA-IR. In IGT cases with overweight and obesity, serum resistin concentrations were higher than

those in IGT cases with normal weight ($16.3 \pm 7.5 \text{ ng/ml}$ vs $9.2 \pm 6.2 \text{ ng/ml}$, $p<0.01$), and serum resistin concentrations positively correlated with age ($r=0.482$, $p<0.05$) and BMI ($r=0.380$, $p<0.05$). In IGT cases, the concentrations of adiponectin were higher in females than males ($12.2 \pm 7.1 \text{ } \mu\text{g/ml}$ vs $6.1 \pm 5.5 \text{ } \mu\text{g/ml}$, $p<0.05$).

Conclusions In IGT cases, serum resistin and adiponectin concentrations are normal, however, serum resistin concentrations were higher in IGT cases with overweight and obesity while adiponectin were higher in female IGT case.

e0307 CLINICAL RESEARCH OF NONINVASIVE CARDIAC HAEMODYNAMICS IN MIDDLE-AGED AND OLD WITH TYPE 2 DIABETES MELLITUS

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Fei Yu, Bao-Ying Li, Shan-Shan Shi, Qian Wang, Zhi-Mian Zhang, Hai-Qing Gao. *Qilu Hospital of Shandong University, Jinan, China*

Objective To explore the characteristics of cardiac haemodynamics in middle-aged and old with type 2 diabetes mellitus, and to discussion the sensitive indicators to determine the early heart disease by Lifegard ICG Haemodynamic Monitor.

Methods 218 individuals (mean age 62.11 ± 10.71 years) were recruited in this study. Cardiac output (CO), cardiac index (CI), systemic vascular resistance (SVR), systemic vascular resistance index (SVRI), stroke volume (SV), stroke index (SI), thoracic fluid content (TFC), acceleration index (ACI), left cardiac works index (LCWI), pre-ejection period (PEP), left ventricular ejection time (LVET), velocity index (VI), contraction time ratio (STR), heart rate (HR) and mean arterial pressure (MAP) were measured using Lifegard ICG Haemodynamic Monitor.

Results (1) Compared with the healthy controls, there was significant difference in the waist-hip ratio (WHR), fasting blood glucose (FPG), total cholesterol (TC), systolic blood pressure (SBP), diastolic blood pressure (DBP), pulse pressure (PP), acceleration index (ACI), velocity index (VI), pre-ejection period (PEP), contraction time ratio (STR) of patients with type 2 diabetes mellitus ($p<0.05$ or $p<0.01$). (2) Compared with the healthy controls, there was significant difference in the fasting blood glucose (FPG), acceleration index (ACI), velocity index (VI) of patients with pre-diabetes ($p<0.05$ or $p<0.01$). (3) Correlation analysis: ACI related negatively with body mass index, waist-hip ratio, fasting blood glucose, systolic blood pressure, diastolic blood pressure, mean artery pressure and heart rate; PEP related positively with body mass index; VI related negatively with body mass index, waist-hip ratio, fasting blood glucose, systolic blood pressure, diastolic blood pressure, mean artery pressure and heart rate; STR related positively with body mass index and waist-hip ratio.

Conclusion Cardiac haemodynamics impaired to varying degrees in diabetes mellitus and pre-diabetes, the body mass index is the common risk factor on the reduction of these indicators. ACI and VI measurements are noninvasive and sensitive indicators of evaluating abnormalities of cardiac haemodynamics in diabetes mellitus.

e0308 PROTEOMIC FEATURES INDUCED BY INSULIN ON VASCULAR SMOOTH MUSCLE CELLS FROM SPONTANEOUS HYPERTENSIVE RATS IN VITRO

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Yan Wang, Bo Zhang, Yun Bai. *Third Military University*

Hyperinsulinemia is a risk factor in atherosclerosis formation that it stimulated VSMCs proliferation and migration. To understand the underlying molecular mechanism involved in the processes of