formation of collateral circulation of Coronary Heart Disease in Han people of Taiyuan area.

METHODS The polymerase chain reaction (PCR), gene sequencing and sequence flanking were used to detect and analyze the polymorphism of -3148C/G site of GDF15 gene for 92 ST-elevation myocardial infarction (STEMI) patients with 68 collateral circulation group, 24 non-collateral circulation group and 56 Patients with normal coronary angiography in a control group.

RESULTS The genotype frequencies of CC, GC were 80.43% and 19.57% in the AMI group, which were 60.71% and 39.29% in the control group respectively, P values of the two groups at -3148C/G CC, GC genotype frequencies distribution is <0.009. The risk genotype GC, OR = 2.660, 95% of confidence interval is 1.265 - 5.595. And the genotype frequencies of GC, GC were 85.29% and 14.71% in the AMI collateral circulation group and 66.67% and 33.33% in the AMI non-collateral circulation group individually, P values of two groups at -3148C/G CC, GC genotype frequencies distribution is <0.05; The risk genotype was GC, OR = 2.900, 95% of confidence interval is 0.983 - 8.526.

CONCLUSIONS There is a correlation the polymorphism of -3148C/G site in GDF15 gene and the Coronary Heart Disease patients with collateral circulation in Ham people of Taiyuan area.

GW26-e4774 β-adrenoceptor autoantibodies increase susceptibility to ventricular arrhythmias by shortening effective refractory period and prolonging MAPD90-30 in guinea pigs

Haixia Huang,1 Yuhui Zhao,1 Ping Liu,1 Suli Zhang,1 Yunhui Du,1 Huijun Zhang,1 Ye Wu,1 Peng Wang,1 Xiao Li,1 Tingting Lv,1 Li Wang,1 Huirong Liu1
1Capital Medical University; 2Shanxi Medical University; 3Beijing Key Laboratory of Metabolic Disturbance Related Cardiovascular Disease

OBJECTIVES Malignant ventricular tachyarrhythmias are life-threatening complications and are the main cause of sudden cardiac death (SCD). While the mechanisms of ventricular arrhythmias are complex and are not completely understood. High titers of autoantibodies against the second extracellular loop of β1-adrenergic receptors (β1-AAs) can be detected in the sera of patients with ventricular arrhythmias, but a causal relationship between β1-AAs and ventricular arrhythmias has not yet been finalized. This study is to investigate whether β1-AAs can induce ventricular arrhythmias directly and to reveal the underlying electrical mechanism.

METHODS Two peptides (HWVRAEDEARRCNYDCKCFDVTNR, CHWVRAEDEARRCNYDCKCFDVTNR) corresponding to the sequence of the second extracellular loop of the β1-adrenergic receptor respectively were used as immunogen to synthesize monoclonal β1-AAs (β1-AAs). While β1-AAs shortened QT interval (QTc) did not change 30 min after intravenous injection of β1-AAs. While β1-AAs shortened the QT interval of paced isolated guinea pig hearts from 360.0 ± 11.1 ms to 333.0 ± 14.0 ms (P < 0.05; n = 5). β1-AAs enhanced susceptibility to ventricular fibrillation evidenced by decreasing the ventricular fibrillation threshold from 11.0 ± 2.5 V to 8.8 ± 1.5 V (P < 0.05; n = 5) and prolonging the ventricular fibrillation duration from 833.0 ± 25 ms to 1608.0 ± 135.0 ms (P < 0.05; n = 5). β1-AAs shortened ERP from 100 ms to 84 ms (P < 0.05; n = 5). No changes in MAPD90, MAPD50, MAPD30, and MAPD90-30 were observed in the control group after a 10-min perfusion with normal Tyrode’s solutions (P > 0.05, n = 5). After perfusion with 0.1 μmol/L β1-AA, MAPD50, MAPD30, and MAPD90 were not affected, while MAPD90-30 was prolonged from 23.0 ± 4.2 ms to 40.0 ± 5.0 ms (P < 0.05; n = 5). ERP/MAPD90 was decreased by β1-AAs caused by β1-AAs may contribute to the repolarization abnormality which gave rise to ventricular arrhythmias.

GW26-e5336 Endoplasmic reticulum stress-mediated apoptosis contributing to high glucose induced vascular smooth muscle cell calcification

Runmin Guo, Keng Wu
Department of Cardiovasology, The Affiliated Hospital, Guangdong Medical College, Zhanjiang, China

OBJECTIVES To investigate whether high blood glucose-induced vascular calcification in diabetes mellitus is caused by the endoplasmic reticulum response and subsequent apoptosis.

METHODS We examined the effects of high glucose on the endoplasmic reticulum (ER) stress response of vascular smooth muscle cells (VSMCs). ALP activity was determined by using the ALP assay kit. Alizarin Red S staining were performed to detect calcium deposition. Runx2 expression in VSMCs was tested using Western blot analysis.

RESULTS High glucose treatment drastically induced the ER stress response in VSMCs. The high glucose-induced osteoblastic differentiation of VSMCs was significantly attenuated by pretreatment with 500 μM 4-PBA (a endoplasmic reticulum stress inhibitor) prior to