Objective of this work was to identify new biomarkers and establish serum protein fingerprints for early diagnosis and preoperative evaluation of luminal stenosis severity of CAD. Methods 48 eligible case subjects, having >50% luminal stenosis in at least one major coronary artery (defined as coronary artery disease (CAD) by angiography), 39 patients with luminal stenosis between 25% and 50% (defined as coronary atherosclerosis), and 52 eligible health individuals, were recruited randomly in this study. The above 139 samples were analysed by Surface-enhanced laser desorption/ionization time-of-flight mass spectrometry (SELDI-TOF-MS). Results The clinical and biochemical characteristics in three groups matched well. To detect CAD, thirty samples from patients with CAD (stenosis ≥50%) and thirty-two samples from the control group were analysed and designated as pattern 1. This pattern 1 model comprised ten potential biomarkers with m/z of 4276, 5326, 4481, 4520, 4205, 5814, 5551, 5689, 5344 and 6134 Da, respectively. The peaks at 5526, 5814, 5551, 5344, 6134 Da were expressed greatly in samples from patients with CAD, but weakly expressed in healthy individuals; and the other five peaks were expressed greatly in healthy individuals but weakly expressed in patients with CAD. Pattern 1 had a specificity of 78.1% and a sensitivity of 80.0%, as evaluated by leave-one-out crossvalidation. The remaining 38 serum samples, as a blind test set, were analysed on the second day to test pattern 1. The specificity and sensitivity of the blind test were 83.3% and 72.2%, respectively. To distinguish different severities of coronary artery stenosis, twenty-seven samples from patients with coronary atherosclerosis (between 25% and 50%) were analysed and compared with thirty CAD (stenosis ≥50%) patient samples. Pattern 2 was established similarly to distinguish coronary atherosclerosis (stenosis between 25% and 50%) from CAD. Pattern 2 comprised seven potential biomarkers with m/z of 3208, 5689, 5326, 4289, 6135, 3164 and 6439 Da, respectively. While the peaks with m/z 5326 and 6135 Da, were expressed more significantly in CAD samples than in coronary atherosclerosis (stenosis between 25% and 50%), the other five biomarkers appeared to be expressed in the opposite manner. This model had a specificity of 92.6% and a sensitivity of 93.3%, as evaluated by leave-one-out crossvalidation. Pattern 2 was tested blindly in another 30 serum samples on the second day. The specificity and sensitivity of the blind test were 75.0% and 77.8%, respectively. Conclusion Taken together, the SELDI-TOF-MS technique combined with bioinformatics approaches can not only facilitate the discovery of better biomarkers for CAD and its severity, but also provide a useful tool for molecular diagnosis.

The ROLE OF PRENATAL CHRONIC HYPOXIA ON MYOCARDIAL ISCHAEMIA-REPERFUSION INJURY IN ADULT RABBITS OFFSPRING

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Objective To evaluate the role of prenatal chronic hypoxia on myocardial ischaemia-reperfusion injury in adult rabbits’ offspring and explore the relevant mechanism. Methods The pregnant New Zealand rabbits were divided randomly into normoxic (n=8) and hypoxic (12% O2 from days 10 to 23 of gestation, n=8) groups. One male offspring of each maternal rabbit was randomly selected to study. The offspring rabbits were subjected to heat stress (42°C for 15 min) at 6 months of age. After 24 h, left anterior descending branches were excised and subjected to ischaemia for 30 min and reperfusion for 120 min. Cardiac histopathological observation was performed by light microscope and electron microscope. The expression of heat shock protein 70 (HSP70) in myocardium was detected by immunohistochemistry. Myocardial enzyme activity, apoptotic index and caspase-3 activity in myocardium were examined as well. Results Ischaemia-reperfusion after heat stress pretreatment increased myocardial enzyme activity, apoptotic index and caspase-3 activity in prenatal chronic hypoxia rabbits (4720.51 ± 744.39 IU/l, 1849.15 ± 416.58 IU/l, 40.43 ± 5.03%, 12.43 ± 1.77 unit, respectively) when compared with control (5388.95 ± 552.43 IU/l, 1435.15 ± 92.08 IU/l, 34.40 ± 4.66%, 10.58 ± 1.42 unit, respectively). Heat stress pretreatment induced HSP70 significant expression in left ventricular myocardium was not observed in prenatal chronic hypoxia rabbits but in normoxic control rabbits. Conclusions Prenatal chronic hypoxia inhibits HSP70 synthesis in the heart of adult offspring in response to body heat stress, which might insult cardioprotection against ischaemia-reperfusion injury.

EFFECT OF GRANULOCYTE-MACROPHAGE COLONY STIMULATING FACTOR ON COAGULATION AND FIBRINOLYSIS IN RABBIT MODEL WITH ILIAC DAMAGED BY BALLOON

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Objective Endothelial dysfunction after percutaneous coronary intervention (PCI) played a key role in perturbations of haemostatic equilibrium. Granulocyte-macrophage colony stimulating factor...