AC group (aspirin 0.3g plus clopidogrel 0.3g), ATG group (aspirin 0.3g plus ticagrelor 0.18g), and ACTE group (triple therapy with aspirin 0.3g, clopidogrel 0.3g and tirofiban 0.15ug/kg/min). Adenosine diphosphate(ADP)-induced platelet aggregation was measured using the multiplate analyzer at the time of initial diagnose of AMI, 2 hours after taking loading-dose of anti-platelet therapy, and the second morning.

RESULTS There were no significant differences in demographic characteristics among groups, including age, risk factors and sex. In the ADP-induced platelet aggregation ratio, there were not statistically significant among groups at the time of initial diagnose of AMI. However, there was a significant difference between before and after the administration of anti-platelet drugs (53.3%±11.2% vs 27.9%±8.5%, p<0.05) in all patients. The platelet aggregation ratio of triple therapy groups was significantly lower than dual anti-platelet therapy, both at the time of 2h after administration drugs and the second morning(4.3%±5.3% vs 23.5%±4.9%, p<0.05; 16.4%±8.5% vs 25.6%±9.4%). Compared with AC group, ATG group had a significantly lower platelet aggregation ratio at the time of 2 hours after administer drugs (26.4%±7.8% vs 20.6%±4.5%, p<0.05). There were no significant differences between AC group and ATG group at the second morning (22.5%±6.4% vs 34.5%±7.3%, p>0.05).

CONCLUSIONS Addition tirofiban to conventional dual anti-platelet therapy can reduce drastically platelet aggregation ratio for the patients with AMI. Compared to aspirin combination with clopidogrel, aspirin plus ticagrelor had better inhibiting effect in 2 hours after administration.

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Peripheral blood based discrimination of Coronary Heart Disease from healthy people by genome-wide gene expression profiling in Chinese Han people
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OBJECTIVES The polygenic inheritance and the dependence on environmental factors pose a challenge for the diagnosis and treatment of coronary heart disease (CHD). We hypothesized that a molecular diagnostic assay using easily accessible peripheral blood would greatly assist in the screening and diagnosis of CHD. In order to validate this a microarray analysis was carried out, the results indicated that there were some genes had differential expression. We discussed the function of these differential genes in the process of the occurrence and development of CHD and further evaluated their diagnostic and therapeutic potential in CHD.

METHODS A total of 26 subjects (n=26) were recruited in this study including 13 CHD and 13 healthy people. The total mRNA of the subjects were isolated from leucocyte in the peripheral blood within 4 hours after collection, and were reverse-transcribed to cDNAs as soon as possible. We constructed 2 pools of 3 subjects (1 CHD and 3 healthy people) for microarray screening with Affymetrix GeneChip® Scanner 3000 to discover new biomarkers and candidate genes. The results were analyzed with SAM, GO, KEGG. The other samples were used for RT-PCR to confirm the microarray on the next step. Randomly selected three candidate genes from the results of microarray for RT-PCR: CYP4F3 acted as up-regulated gene, IL13RA1 acted as normal expression group, MED6 acted as down-regulated group. The results of RT-PCR was analyzed by 2^ΔΔCt method.

RESULTS The results of microarray were analyzed by Affymetrix Expression Console Software. With the choice criterion of |logFC|>1, logFC<1, these 300 differential genes were selected, among them 30 genes had over expression and 270 genes had low expression. Analyzed by GO showed these 300 genes belonged to 105 cellular components, and involved in 295 biological processes and participated in the regulation of 212 molecular functions. Analyzed by KEGG showed that these genes took part in 75 gene pathways. The results of RT-PCR analyzed with 2^ΔΔCt method showed that compared to healthy people, the expression of CYP4F3 gene was 2.06 ± 0.57 (p=0.004), the expression of IL13RA1 gene was 0.96 ± 0.05 (p=0.870), and the expression of MED6 gene was 0.62 ± 1.10 (p=0.0002). All the results obtained from RT-PCR were in accordance with the results of microarray.

CONCLUSIONS The microarray can be used as the foundation of exploring the pathogenic gene for coronary heart disease, and the differential expression of CYP4F3 or MED6 may take effect on the process of the occurrence and development of coronary heart disease. It can be conjectured that these findings may serve to identify new...