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1. Tongxinluo Prevented Vasospasm Through Lowering Circulation Angiotensin II Level and Restraining Local Oxidative Stress

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[KEY WORDS] Adventitia; Vasospasm; 5 Hydroxytryptamine; Angiotensin II;
Angiotensin II Receptors; Oxidative Stress; Tongxinluo

[ABSTRACT]  Aim: The present study was undertaken to investigate the possible mechanism of Tongxinluo on preventing vasoconstriction and vascular hypersensitivity to 5 hydroxytryptamine induced by the injury of the adventitia.

Methods: Wistar Kyoto rats were assigned to 4 treatments (n=12 for each group): vehicle, low dose of Tongxinluo [200 mg/(kg ·d)], middle dose of Tongxinluo [400 mg/(kg ·d)] and high dose of Tongxinluo [800 mg/(kg ·d)]. After 1 week of treatment, adventitia injury was induced by positioning a silicone collar around the right carotid artery for one week. Blood flow and vascular reactivity to serotonin were determined 1 week after injury, the blood from the rat’s heart was taken to measure the concentration of angiotensin II (Ang II) in the serum with the ELISA, and both side of carotids were harvested for RT PCR analysis. Results: Collar induced adventitia injury decreased the carotid blood flow (P=0.0002), increased the vascular reactivity sensitivity to 5 hydroxytryptamine, and upregulated the expression of Ang II type 1 (AT 1) receptor (135% increase, P=0.0020), Ang II type 2 (AT 2) receptor (76% increase, P=0.0061), and p22phox (2.89 fold increase, P<0.0001) in collared arteries. Low dose of Tongxinluo did not affect vasoconstriction function, serum Ang II concentration, or the expressions of Ang II receptors and p22phox. Treatment with medium dosage and high dosage of Tongxinluo can effectively improve the carotid blood flow, normalize the hypersensitivity to 5 hydroxytryptamine (all with P<0.05), lower serum Ang II concentration (middle dosage group: 26.31±6.82 ng/L vs 45.21±4.52 ng/L, P=0.0480; high dosage group: 20.51±2.51 ng/L vs 45.21±4.52 ng/L, P=0.0183),
restrained the increase of p22phox expressions (79.1% decreasing in middle dosage group,  P=0.0002 ; 83.2% decreasing in high dosage group,  P=0.0001), did not affect the AT1 receptor expression, while high dose of Tongxinluo increased AT2 receptor expression. Conclusion: Tongxinluo can effectively prevent the vasoconstriction and the vascular hypersensitivity to 5HT induced by the adventitia injury in rat carotid through lowering serum Ang II level and restraining the significantly enhanced oxidative stress induced by the adventitia injury.

2. Effects of Rosiglitazone on Proliferation of Vascular Smooth Muscle Cells Induced by Angiotensin II

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[KEY WORDS] Rosiglitazone; Angiotensin II Type 2 Receptor; Vascular Smooth Muscle Cell

[ABSTRACT] Aim: To investigate the effects of rosiglitazone on the proliferation of vascular smooth muscle cells induced by angiotensin II (Ang II) and possible mechanism. Methods: Vascular smooth muscle cells (VSMC) were isolated from aortic media of four week old male Sprague Dawley rats by enzymatic digestion and cultured in monolayer. VSMC in passage 4 ~ 8 in log phase were used in following experiments. VSMC were treated by 1 μmol/L Ang II for 6 h and randomly divided into
the groups as follows: control group (10% FBS in DMEM), Ang II group (1 μmol/L Ang II), groups respectively treated with different concentration of rosiglitazone (20, 30, 40 and 50 μmol/L) for 12 h and groups respectively treated with 30 μmol/L rosiglitazone for 6, 12, 18 and 24 h. The VSMC growth, change of proliferation cycle and mRNA and protein expression of Ang II type 2 receptor (AT 2R) in all groups were detected by using MTT colorimetric assay, FCM, RT PCR and Western blotting. Results: The mean absorbance (A value) in the VSMC treated by Ang II was significantly higher as compared with that of the control group (P<0.01). The A values were markedly reduced in the VSMC treated by different concentration of rosiglitazone for 12 h or 30 μmol/L rosiglitazone for 6, 12, 18 and 24 h (P<0.05 or P<0.01). The proliferation index (PI) and S phase fraction (SPF) in the Ang II group were significantly higher than those of the control group (P<0.01). With the increase in rosiglitazone concentration and prolongation of treatment time, PI and SPF were greatly reduced (P<0.05 or P<0.01). Compared with the Ang II group, expression of AT 2R mRNA and protein in the VSMC with the treatment of 20, 30 and 50 μmol/L rosiglitazone for 12 h or of 30 μmol/L rosiglitazone for 6, 12 and 24 h were both markedly increased (P<0.05 or P<0.01), reaching a maximum in 50 μmol/L rosiglitazone for 12 h or 30 μmol/L rosiglitazone for 24 h. Conclusions: Rosiglitazone inhibited VSMC proliferation induced by Ang II at least partially through up regulating expression of AT 2R both at mRNA and protein levels in a concentration dependent and time dependent manner, in order to play vasculoprotective role.
3. The Model Establishment and Identification of RAW264.7 Macrophage Derived Foam Cell

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[KEY WORDS] RAW264.7 Cells; Foam Cell; Cholesterol Ester; Oxidized Low Density Lipoprotein

[ABSTRACT] Aim: To establish the model of murine macrophage RAW264.7 derived foam cell and identify them with simple and accurate method. Methods: The experiment was divided into normal control group and six experimental groups of different concentration of oxidized low density lipoprotein (ox-LDL) incubated with cell. On the incubation time for 24 hours that MTT method and flow cytometry (FCM) were used to determine the suitable range of ox-LDL concentration, then measure intra cellular cholesterol ester in different level of macrophage foam cell with total cholesterol and free cholesterol kit. Results: Cell viability had been significantly inhibited when ox-LDL concentration was at the range of 20~30 mg/L, when ox-LDL concentration was greater than 40 mg/L, apoptosis cells continuously necrosis. Ox-LDL at the concentration of 20 and 30 mg/L, incubated with cell for 24 hours, the proportion of intracellular cholesterol ester were 66.26% and 71.19%. Foam cell induced by 10 mg/L of ox-LDL was not typical, and by 40 mg/L of ox-LDL or more, severe degree of foam cell led to cells floating, most cells were rupture or apoptosis, lipid droplet dispersed in extra
cellular. Conclusion: Ox-LDL at the concentration range of 20~30 mg/L, incubated with RAW264.7 for 24 h, induced foam cell has good morphology, and model stability, meet the morphology feature of foam cell.

4. Effects of Insulin on Impaired Ischemia Induced Neovascularization in Diabetic Mice

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[KEY WORDS] Diabetes Mellitus; Neovascularization; Vascular Endothelial Growth Factor; Insulin

[ABSTRACT] Aim: To investigate whether ischemia induced neovascularization is impaired in diabetic mice and the effect of insulin administration on this dysfunction and the underling mechanisms. Methods: Unilateral hindlimb ischemia was performed in streptozotocin induced diabetic mice (C57BL/6) by left femoral artery ligation. The plasma vascular endothelial growth factor (VEGF) and stromal derived growth factor 1α (SDF 1α) levels were measured by ELISA before ligation and 1, 3, 7, 14 days after ischemia. Capillary density was determined in both ischemic and non ischemic gastrocnemius muscles by CD31 staining. The local expressions of VEGF, endothelial nitric oxide synthase (eNOS), phospho eNOS, protein kinase B (PKB, Akt) and phosphor Akt were quantified by Western blotting. Results: After ischemia, diabetic mice showed
abrogated capillary density increase in the ischemic tissue compared with non diabetic mice (day7: 7.65±1.74 vs 18.22±3.77, P<0.05), which were accompanied by impeded release of plasma VEGF and SDF 1α (P<0.01), and impaired upregulation of local VEGF protein expression as well as Akt and eNOS phosphorylation (P<0.05). Insulin administration significantly ameliorated ischemia induced angiogenesis in treated compared with non treated diabetic groups (15.36 ± 2.14 vs 7.65 ± 1.74, P<0.05), elevated plasma levels of VEGF and SDF 1α (P<0.01), as well as enhanced local VEGF expression and Akt/eNOS phosphorylation (P<0.05). Conclusion: The data suggested that insulin administration efficiently improved impaired ischemia induced neovascularization in diabetic mice which may be attributed to restoration of attenuated SDF 1α/VEGF/Akt/eNOS activation.

5. The Role of Intrauterine Chronic Hypoxia on Vascular Endothelial Function in Rats Offspring

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[KEYWORDS] Intrauterine Hypoxia; Vascular Endothelial Function; Fetal Programming; Hyperlipemia; Adult Hypoxia

[ABSTRACT] Aim: To investigate the effects of fetal intrauterine chronic hypoxia on the vascular endothelial function of adult offspring rats, and its relation to gender,
hyperlipemia, and adult hypoxia. Methods: Four factorial experiment was designed to explore the role of fetal intrauterine chronic hypoxia, gender, hyperlipemia, and adult hypoxia on endothelial dependent diastolic function. Four animal models of intrauterine chronic hypoxia, hyperlipemia and adult hypoxia were established in Sprague Dawley rats. Endothelial dependent diastolic function and histologic changes were determined in the rats offspring. Results: Except the factor of gender, the other three factors of intrauterine hypoxia, hyperlipemia, and adult hypoxia resulted in an impairment of endothelial dependent diastolic function with main effects of 14.1%, 14.2%, 12.9%, respectively (all P<0.01). There was a positive interaction between intrauterine hypoxia and hyperlipemia on endothelial function (F=4.889, P<0.05), but no other significant interactions among these four factors. Furthermore, marked histological changes, such as edema, necrosis, and desquamation of vascular epithelium, platelet aggregation and microthrombosis, subendothelial edema and infiltration of inflammatory cells, were observed in the fetal hypoxia offspring but not in the control group. Conclusion: Intrauterine chronic hypoxia can induce both functional and morphologic impairment in vascular endothelium from adult offspring rats. This effect on the impaired endothelial function was similar to hyperlipemia and adult hypoxia on that, and was enhanced with hyperlipemia.

6. Multiplication of Human Cytomegalovirus in Umbilical Arteries Vascular Smooth Muscle Cells
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[KEY WORDS] Human Cytomegalovirus;Atherosclerosis;Vascular Smooth Muscle Cells; Transplant Vasculopathy

[ABSTRACT] Aim: To explore the infectivity of human cytomegalovirus (HCMV) in vascular smooth muscle cells and provide a cellular model to the mechanism research of atherosclerosis and transplant vasculopathy induced by this virus. Methods: Vascular smooth muscle cells were isolated from umbilical arteries, and HCMV with 1 MOI was cocultured with these cell, the proliferation of virus in these cells was assured by cytopathic effect, the expression of HCMV IE gene, and virus particles observed by electric microscope. Results: Cytopathic effect was observed three days post infection, and HCMV immediate early (IE) gene was expressed in these cells post infection by RT PCR, the correctness was assured by DNA sequencing. With electronic microscope technique, the virus particles were observed in these cells. Conclusion: HCMV can infect vascular smooth muscle cells and establish proliferating infection. The work provide a model for the research of mechanisms of atherosclerosis and transplant vasculopathy induced by human cytomegalovirus.

7. Expression of CD34 in Human Coronary Atherosclerotic Lesion

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**KEY WORDS** Coronary Atherosclerosis; CD34; Angiogenesis; Immunohistology

**ABSTRACT** Aim: To study the expression of CD34 in human coronary atherosclerotic lesions, and the correlation between the expression of CD34 and the lesion types of coronary atherosclerosis, the degree of luminal stenosis and its significance. Methods: 312 of coronary artery tissue samples were selected in 53 cases of autopsy. Coronary atherosclerotic lesion and its types were diagnosed by light microscope, and CD34 positive microvessels and CD68 positive macrophages in coronary atherosclerotic lesion were counted by immunohistochemistry. Results: CD34 positive microvessels in intima of coronary atherosclerotic lesions were increased with progress of coronary atherosclerotic lesions and aggravation of luminal stenosis with positive correlation (r=0.344, r=0.284, P<0.01), respectively, and the number of CD34 positive microvessels had significant differences between early lesions (type I III) and advanced lesions (IV VI, P<0.05); The number of CD34 positive microvessels had significant difference between normal cholesterol and hypercholesterolemia (P<0.05); CD68 positive macrophages in intima of coronary atherosclerotic lesions mainly distributed around the CD34 positive blood microvessels, and had positive correlation with CD34 positive microvessels (r=0.303, P<0.01). Conclusion: CD34 positive neo microvessels in intima of human coronary atherosclerosis are increased along with the progress of coronary atherosclerotic lesions and the aggravation of luminal stenosis and the increased infiltration of CD68 positive macrophages and hypercholesterolemia facilitate
angiogenesis in atheromatous plaque of coronary atherosclerosis.

8. Expression of Angiotensin II Type 2 Receptor and the Effects of Rosuvastatin on it After Vascular Balloon Injury in Rats

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**[KEY WORDS]** Rosuvastatin; Balloon Injury; Angiotension II Type 2 Receptor

**[ABSTRACT]** Aim: To investigate the expression of angiotension II type 2 receptor (AT 2) and the effect of rosuvastatin on it after vascular balloon injury in rats. Methods: Rat models of aortic endothelial denudation were established by 2F balloon catheters. Rats were randomly allocated into three groups: control group, vascular balloon injury group and rosuvastatin treatment group. The expression of AT2 mRNA and protein were investigated at day 14 and 28 after injury by reverse transcription polymerase chain reaction (RT PCR) technique and immunohistochemistry method, respectively. Results: Significant intimal thickening was observed at 14 days and 28 days after injury. Rosuvastatin significantly prevented intimal thickening at 14 days and 28 days after balloon injury. The expression of AT 2 mRNA and protein increased significantly at 14 days and 28 days after injury (P<0.05), and they increased obviously after rosuvastatin treatment (P<0.05). Conclusion: The expression of AT 2 mRNA and protein increased significantly after endothelial injury, and rosuvastatin upregulated
the expression of AT 2.

9. Effects of Streptococcus Mutans on the Expression of Toll Like Receptor 2 and 4, Interleukin 6 and 8 in EAhy926 Cells

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(Department of Microbiology, Capital Medical University, Beijing 100069, China) [KEY WORDS] Streptococcus Mutans ; EAhy926 Cell ; Toll Like Receptor 2 ; Toll Like Receptor 4 ; Interleukin 6 ; Interleukin 8

[ABSTRACT] Aim: To investigate the effects of streptococcus mutans on the expression of toll like receptor 2 (TLR2), TLR4, interleukin 6 (IL-6) and IL-8 in EAhy926 cells (the human endothelial hybridoma of human umbilical vein endothelial cells (HUVEC) and the human epithelial cell line A549, characterized by endothelial phenotype and biology). Methods: EAhy926 cells were treated with Streptococcus mutans. The mRNA expression of TLR2, TLR4, IL-6 and IL-8 in EAhy926 cells was detected by reverse transcription polymerase chain reaction (RT-PCR). TLR2 and TLR4 were analyzed by flow cytometry. The production of IL-6 and IL-8 in the cultured supernatants was measured by biochemical method and ELISA respectively. TLR2 and TLR4 blocking assay was used to investigate the relationship between IL-6, IL-8 and TLR2, TLR4 mRNA expression.

Results: The expression of mRNA and protein for TLR2 and TLR4 in EAhy926 cells increased after stimulated by Streptococcus mutans, peaked the maximal level at 6 h (P<0.05), and then decreased. The expression of IL 6 and IL 8 mRNA was significantly induced when exposed to Streptococcus mutans, reaching the maximal level at 12 h,
respectively (P<0.05). Meanwhile, Streptococcus mutans induced the production of IL-6 and IL-8 with peaking at 12 h (P<0.01). The mRNA expression of IL-6 and IL-8 in EAhY926 cells was significantly blocked by anti human TLR2 and anti TLR4. Conclusion: Streptococcus mutans upregulated the expression of TLR2 and TLR4 and induced the production of inflammatory cytokines IL-6 and IL-8. The expression of TLR2 and TLR4 of EAhY926 cells may elicit a TLR2 and TLR4 mediated innate immune response and contribute to production of inflammatory cytokines IL-6 and IL-8.

10. Association of TNFRSF1B Exon 6 rs1061624 Polymorphism and Coronary Heart Disease

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[KEY WORDS] TNFRSF1B Gene; Single Nucleotide Polymorphism; Tumor Necrosis Factor; Coronary Heart Disease

[ABSTRACT] Aim: To investigate the relationship between the single nucleotide polymorphism (SNP) of TNFRSF1B gene and coronary heart disease (CHD) in Chinese population. Methods: The polymerase chain reaction ligase detection reaction (PCR LDR) was used to detect TNFRSF1B gene rs1061624 in the 104 controls and 208 coronary heart disease patients. Results: The frequency of this polymorphism was consistent with the law of Hardy Weinberg. The frequency of rs1061624 genotype did not differ
between the patients and the controls (13.7% vs 18.2%, P>0.05). These results were independent on age, gender, hypertension, diabetes and hyperlipidemia. Conclusion: The results support that there is no significant correlation between the polymorphism of TNFRSF1B rs1061624 and coronary heart disease.

11. The Change of Cerebrovascular Hemodynamics of Cerebrovascular Atherogenesis

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[KEY WORDS] Hemodynamics; Cerebrovascular Atherogenesis; Cerebroinfarction

[ABSTRACT] Aim and Methods: The change of cerebrovascular hemodynamic index (CVDI) and Doppler expression of 26 patients with cerebrovascular atherogenesis using the surveying instrument of cerebrovascular hemodynamics were studied. Results: ① In most of the patients two sides of total cerebral blood flow and the velocity didn’t decrease, the cerebrovascular resistance, elasticity and critical pressure increased. The function of cerebrovascular regulation deteriorated. ② The Trans Link Doppler found: the blood flow and the velocity of anterior cerebrovascular artery, middle cerebrovascular artery were in normal range. Cerebral vascular atherogenesis index especially the (Pulstility P1) increased. ③ The CVDI had remarkable changes in the lesion side of brain hemisphere, especially the total cerebral blood flow and the velocity decreased. Conclusion: The cerebrovascular resistance and the function of
cerebrovascular regulation are more serious than that of cerebrovascular atherogenesis.

12. Different Inflammatory Markers Differ in Predicting the Severity and Instability of Coronary Artery Disease

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[ KEY WORDS ] Soluble CD40L; Monocyte Chemotactic Protein 1; Interleukin 8; Interleukin 6; Gensini Score

[ ABSTRACT ] Aim: To investigate the predicting value of plasma levels of soluble CD40L (sCD40L), monocyte chemotactic protein 1 (MCP-1), interleukin 8 (IL-8), and interleukin 6 (IL-6) for the severity and instability of coronary artery disease. Methods: sCD40L, MCP1, IL-8 and IL-6 were measured in 129 cases of coronary artery disease patients, including 39 stable angina pectoris, 43 unstable angina pectoris and 47 acute myocardial infarction, using flow cytometric method. Gensini scores were evaluated for each patient. Correlation of the above inflammatory factors with Gensini scores was analyzed. The predicting value of the above biomarkers for acute coronary syndrome was investigated. Results: Concentrations of the tested inflammatory factors were higher in all the three
coronary artery disease groups than those in contrast (all P<0.01). Concentrations of sCD40L, MCP 1 and IL 6 were increased in acute myocardial infarction patients than stable angina pectoris patients (P=0.001, P=0.009, P=0.011, respectively). Plasma concentrations of both MCP-1 and IL-6 manifested positive correlation with Gensini scores (r=0.322, P<0.00001; r=0.203, P=0.026, respectively). Multiple Logistic regression analysis showed that IL 6 could predict acute coronary syndrome (OR=1.275, P=0.037). Conclusion: The plasma concentrations of sCD40L, MCP 1, IL-8 and IL-6 can predict the existence of atheroma; MCP-1 and IL-6 levels correlate to the severity of atheroma in coronary artery disease patients; IL 6 plays a role in predicting acute coronary syndrome.

13. Changes in the Number and Function of Endothelial Progenitor Cells from Peripheral Blood in the Patients with Low Extremity Arteriosclerosis Disease

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[KEY WORDS] Low Extremity Arteriosclerosis Disease; Endothelial Progenitor Cells

[ABSTRACT] Aim: To investigate the changes in the number and function of endothelial progenitor cells (EPC) from peripheral blood in patients with low extremity arteriosclerosis disease (LEAD). Methods: Sixty cases were divided into LEAD group (n=30) and control group (n=30). Mononuclear cells (MNC) were isolated from peripheral
blood by density gradient centrifugation. After 7 days induced differentiation, the adherent cells were identified as EPC by fluorescein staining and flow cytometry. EPCs’ s number, proliferation, migration and adhesion were assayed by Giemsa’ s staining, MTT chromatometry, modified Boyden chamber assay and adhesion activity assay. Results: The number of EPC was significantly reduced in patients with LEAD compared with control group (27.2 ± 3.6 : 52.6 ± 5.9, Cells/× 200 fields, P<0.05) and the number of cell clusters was also reduced in patients with LEAD compared with control group (16.6 ± 4.8 : 22.3 ± 4.9, CFU/×40 fields, P<0.05). In addition, the proliferation (0.193 ± 0.064 : 0.243 ± 0.078, P<0.05), migration (12.1 ± 2.7 : 17.8 ± 4.2, Cells/× 200 fields, P<0.05) and adhesion (47.3 ± 4.3 : 51.9 ± 3.7, Cells/× 200 fields, P<0.05) in patients with low extremity arteriosclerosis disease were impaired respectively. Conclusion: EPC number and function are abnormally decreased in patients with low extremity arteriosclerosis disease.

14. The Homocysteine Levels and Methylene tetrahydrofolate Reductase Gene Polymorphisms in Atherosclerotic Cerebral Infarction

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[ KEY WORDS ] Homocysteine; Methylenetetrahydrofolate Reductase; Gene Polymorphism; Cerebral Infarction; Atherosclerosis
[ABSTRACT] Aim: To investigate the relationship between plasma homocysteine (Hcy), polymorphisms of the methylenetetrahydrofolate reductase (MTHFR) gene and atherosclerotic cerebral infarction. Methods: Plasma Hcy, polymorphisms of the MTHFR gene in 68 patients with atherosclerotic cerebral infarction (CI) and 50 controls were measured by fluorescence polarization immunoassay (FPIA) and polymerase chain reaction and restriction fragment length polymorphism (PCR RFLP) respectively. Results: The frequencies of TT genotype and T allele were significantly higher in CI group than in controls (P < 0.05). Plasma Hcy levels was significantly higher in CI group than in controls (P < 0.05). The homocysteine concentration was significantly higher in TT genotype than CT and CC genotypes both in CI group and in control group (P < 0.05). Conclusion: Elevated Hcy levels is a risk factor for atherosclerotic cerebral infarction. MTHFR C677T polymorphism was significantly related to plasma Hcy levels and atherosclerotic cerebral infarction.

15. Disposition of Plasma NT proBNP Level in the Community Cohort

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[ABSTRACT] Aim: To explore the disposition of plasma NT proBNP level in the community cohort. Methods: 734 subjects with high risks of cardiovascular diseases in Beijing Shougang community were investigated. The plasma NT proBNP level were measured with the automated electrochemiluminescence immunoassay. Their history of myocardial infarction, angina pectoris, hypertension, atrial fibrillation, and diabetes mellitus were collected, their cardiac function were also evaluated with New York Heart Association functional classification. The left ventricular ejection fraction (LVEF) were measured by Echocardiography. To the normal LVEF subjects, they were put in different diastolic heart function groups (the normal one, the mild damaged and the moderate/severe damaged ones) according to mitral inflow, annulus of mitral valve tissues and pulmonary venous flow of echocardiography. Then the disposition of plasma NT proBNP level in the community cohort were analyzed. Results: The percentage of NYHA functional class I and class II were 86.5% and 12.9%, and LVEF less than 50% was 3.5%. The median of plasma NT proBNP level was 69.75 ng/L. The plasma NT proBNP level in the group with coronary artery disease was higher than that in the group without coronary artery disease (108.60 ng/L vs 65.68 ng/L, P<0.05), the plasma NT proBNP level in the group with hypertension was higher than that in the group without hypertension (72.71 ng/L vs 61.51 ng/L, P<0.05), the plasma NT proBNP level in the group with atrial fibrillation was higher than that in the group without atrial fibrillation (93.31 ng/L vs 67.61 ng/L, P<0.05), and the plasma NT proBNP level in the group with diabetes mellitus was higher than that in the group without diabetes mellitus (80.05 ng/L vs 66.04 ng/L, P<0.05). It was found that the plasma NT proBNP level...
in the group of NYHA functional class II was higher than that in the group of class I (115.5 ng/L vs 65.01 ng/L, P<0.05), and the plasma NT proBNP level in the lower LVEF group was higher than that in the normal LVEF group (293.8 ng/L vs 67.85 ng/L, P<0.05). In the group with normal LVEF, the plasma NT proBNP level in the group with different diastolic heart function were 53.73 ng/L, 75.07 ng/L, 101.85 ng/L and 269.75 ng/L. Conclusion: Although the plasma NT proBNP level in the community cohort is low, but it may be a biochemical marker to monitor the change of their heart function which results in various kinds of cardiovascular diseases.

16. Changes of Plasma Interferon γ , Interleukin 10 and C Reactive Protein in Patients with Acute Coronary Syndrome

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[KEY WORDS] Acute Coronary Syndrome; Interferon γ ; Interleukin 10; C Reactive Protein

[ABSTRACT] Aim: To study the clinical value of interferon γ, interleukin 10 and CRP in acute coronary syndrome. Methods: Using a high sensitivity multiplex assay, previously untested in the context of atherosclerotic disease, we determined plasma concentrations of interferon γ, interleukin 10, CRP, interleukin 2, interleukin 6, interleukin 8, TNF α, interleukin1 β, interleukin 12p70 and GM CSF in 68 acute
myocardial infarction (AMI) patients, 28 unstable angina (UA) patients and 22 healthy controls. Results: Interferon γ and CRP levels were significantly higher in AMI compared to UA group (P<0.05) and control group (P<0.05). Interleukin 10 also showed higher expression levels in AMI group compared to UA group and control group (P<0.05). Conclusion: This up-regulation may reflect the extent of plaque instability and/or rupture in MI patients. Our observations provide evidence that interferon γ, interleukin 10 and CRP merit further investigation in atherosclerotic disease states as potential markers of disease and therapeutic targets.

17. Clinical Significance of Platelet CD62P and CD63 Expression in Patients with Acute Cerebral Infarction

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[KEY WORDS] Acute Cerebral Infarction; Activated Platelets; Flow Cytometry

[ABSTRACT] AimTo investigate the relationship between platelet CD62P and CD63 expression and acute cerebral infarction (ACI). Methods: The expression of CD62P, CD63 was detected by flow cytometry in 61 patients with acute cerebral infarction (ACI), and 59 healthy control subjects. Results: CD62P, CD63 in acute cerebral infarction were 6.26%±1.68% vs 2.32%±1.14%, 6.14%±1.89% vs 2.88%±1.15% respectively, and the result has statistical significance compared with those of the control group (P<0.01). Conclusion: The formation of thrombus in acute cerebral infarction patients is highly
related to the platelets activation, which can be a diagnostic indicator of cerebral infarction disease.