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Comparison Study of gene expressions of integrin β2 between acute myocardial infarction and stable angina pectoris patients

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OBJECTIVES It has been shown that atherogenesis is a chronic infectious process involving various kinds of immune cells caused by endothelial cell injuries. The adhesion process which promotes inflammatory reactions is mediated by adhesion molecules among injured intima and activated leukocytes, platelets and endothelial cells. Integrin β2 is specifically expressed on surface of leukocytes, including αDβ2, αLβ2, αMβ2 and αXβ2. Abnormalities of human integrin β2 gene (ITGB2) expressions could cause changes in adhesion depended processes, such as chemotaxis, phagocytosis and aggregation. This study aimed to detect and compare the expression intensity of integrin β2 and its related genes among acute myocardial infarction (AMI) patients, stable angina pectoris (SA) patients as well as healthy people, and analyze the gene expression characteristics of integrin β2 on the surface of leukocytes in different phases.

METHODS 20 patients with AMI were enrolled into AMI group. Another 20 SA patients (18 males and 2 females, average age: 64±10 yrs) were recruited into SA group. There were no significant differences of age, gender, smoke, body mass index, systolic blood pressure, diastolic blood pressure, low density lipoprotein, high density lipoprotein and triglyceride among three groups ($P>0.05$).

RESULTS A total of 76 genes associated with integrin β2 were detected. The mRNA expressions in three groups are shown as follows. Compared with controls, mRNA expressions of α/β subunit (ITGAD, ITGAL, ITGAM, ITGAX and ITGB2), ligands of integrin β2 (RAGE, JAM-1, Fibrinogen, ICAM-1, ICAM-3, ICAM-5 and uPAR), chemokine (fMLP and RAF), inside-out signaling pathway (RIAM, ADAP, SLP-76, PLCγ, RAPL, SPA1, α-actinin, calpain, Dok1, radixin, talin and 14-3-3ζ) andoutside-in signaling pathway (Fgr, Hck, Lyn, PKC, c-ab1, Syk, Vav1/3, cdc42, mDia, Rac, RhoA and ROCK1/2) in AMI patients were significantly upregulated. However there was no significant difference of gen expressions of integrin β2 and its signaling pathway in SA patients compared to control group.

CONCLUSIONS Taken together, the inside-out signaling pathway of integrin β2 is inhibited while the outside-in signaling pathway activated in AMI patients, suggesting that activation of integrin β2 in AMI patients might be caused by extracellular factors.

GW27-e1102

Serum Pentraxin 3 and NT-proBNP Levels in pulmonary arterial hypertension

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OBJECTIVES Pulmonary arterial hypertension leads to increased pulmonary vascular resistance, vascular remodelling and plexiform lesions. Some recent studies have shown that inflammation has an important role in the pathophysiology of pulmonary arterial hypertension. The aim of this study is to evaluate Serum Pentraxin 3, NT-proBNPtandem and several other potential PAH biomarkers levels in pulmonary arterial hypertension (PAH)., and evaluate the role of inflammation in pulmonary hypertension.

METHODS After ethics committee approval and receiving consent from parents, a total of 31 patients, 5 with idiopathic PAH. (IPAH) and 26 with PAH associated with connective tissue disease (CTD), and 8 age-matched, non-relative controls were studied. After recording data about all the patients including age, gender, weight, haemodynamic studies and vasodilator testing, a physical examination was done for all subjects. Blood was taken from patients. Serum Pentraxin 3, N-terminal pro-Brain Natriuretic Peptide (NT-ProBNP) and hs-CRP levels were measured. Serum Pentraxin-3 levels were measured by enzyme linked immunosorbent assay (ELISA) and expressed as ng/mL. The serum concentration of NT-proBNP was determined by a chemiluminescent immunumetric assay and expressed as pg/mL.

RESULTS Serum Pentraxin- 3 levels were determined to be 1.38±2.16 ng/mL in the PAH group and 0.42±0.65 ng/mL in control group. There was a statistically significant difference between the two groups ($p<0.01$). Serum NT-proBNP levels were measured as 2800.27±700.82 pg/mL in PAH group and 355.36±217.34 pg/mL control group.

CONCLUSIONS Our study showed that Pentraxin 3 and NT-proBNP levels were increased significantly in the PAH group. We consider that inflammation plays an important role in severe pulmonary hypertension.

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New Non-invasive Test to Screen Patient for Pulmonary Hypertension

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OBJECTIVES Current diagnosis of pulmonary HTN is by measuring the pulmonary pressure non-invasively with echo Doppler or invasively by direct measurement of the pulmonary artery pressure. However, with Doppler echocardiography, the positive predictive value is not very high because of many false positives. We suggest a new test which can give better predictive value.

METHODS Patients with suspected of pulmonary HTN by echocardiography and Doppler studies were enrolled. First, the patients underwent the new Expansibility of the Femoral Vein test (EFV). Then the patient underwent the right heart catheterization (RHC) which confirmed the results of the EFV. During the RHC, the baseline femoral vein pressure and during cough were measured. The study group underwent the new Expansibility of the Femoral Vein (EFV) and had treatment based on its results. The EFV is the ultrasound study of the femoral vein examining its size and expansibility during strong cough. In general, the location of the femoral artery and vein to be checked is the sagittal plane immediately proximal to the bifurcation of the superficial and deep femoral artery. The size of the femoral vein is a little larger than the size of the femoral artery. If the size of the femoral vein during cough is 3 times larger than the one at baseline, the test is considered normal. If the size of the femoral vein is >3 times larger than then baseline, it is considered abnormal suggesting excessive venous pooling. If the femoral vein expands only <2 times of the baseline during cough, it is considered abnormal suggesting present or future pulmonary hypertension. If this test was done in conjunction with a right heart catheterization, then the femoral vein pressure at baseline and during cough is recorded.

RESULTS 25 patients were enrolled from January 2015 to April 2016. All came with history of high suspicion of pulmonary HTN by echo Doppler. They were the patients with connective tissue disease (WHO Classification group 1), dilated cardiomyopathy (group 2), severe chronic obstructive pulmonary disease (COPD) (group 3), or old pulmonary embolism (group 4), chronic kidney disease (group 5). The results showed that 20/25 patients had abnormal EFV test confirmed by RHC (positive predictive value of 80%). All five patients with normal EFV test showed normal pulmonary artery pressure (no pulmonary HTN). So the negative predictive value of the EFV test was 100%. The data of femoral vein pressure at baseline and during cough will be presented.

CONCLUSIONS The patients with high risk of pulmonary HTN should have the EFV early and if the results are positive, the patient should undergo the RHC to confirm its presence. A negative EFV test suggests no pulmonary HTN. Larger scale of clinical trial or registries of this new technique are needed.

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Proposal of a New Mechanism Causing Pulmonary Hypertension

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OBJECTIVES Current diagnosis of pulmonary arterial hypertension (PAH) is by measuring the pulmonary pressure non-invasively with echo Doppler and confirming it with invasive measurement of the pulmonary artery pressure. However, once the PAH is confirmed, the prognosis is poor. In the study of PAH, the pathogenesis of PAH is still being debated. In this study, we suggest a new mechanism explaining the cause of the PAH.