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Galectin-3 in patients with coronary heart disease and atrial fibrillation

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ABSTRACT

Objective: To observe the change of the inflammatory factor Galectin-3 in patients with coronary heart disease, and the correlation between Galectin-3 and the severity of the disease. To observe changes of Galectin-3 in patients with atrial fibrillation (AF) before and after radiofrequency ablation, and the changes of Galectin-3 before and after an interim treatment with a high dose of atorvastatin on patients with acute myocardial infarction(AMI).

Methods: Patients with coronary heart disease and atrial fibrillation having normal heart function were selected, among them, the patients with AMI were given a short term treatment of 80 mg atorvastatin before PCI, and patients with atrial fibrillation underwent radiofrequency catheter ablation. ELISA technique was equipped to observe the Galectin-3 changes in patients with coronary heart disease and that of patients with AF before and after radiofrequency ablation.

Results: Galectin-3 level of the AMI group was higher than that of the unstable angina pectoris (UAP) group, and its levels were higher than that of the stable angina pectoris (SAP) group, the differences were statistically significant among both groups ($P < 0.05$); Galectin-3 level of multivessel coronary disease group was higher than that of single vessel group, in which a statistically significant difference was noted ($P < 0.05$); There was no statistically significant difference associated in the drop of Galectin-3 levels in patients with AMI after PCI ($P > 0.05$); Galectin-3 of patients with AF decreased after RFCA, but no statistical significance noted ($P > 0.05$); Galectin-3 was negatively correlated with the LVEF value($r = -0.405$, $P < 0.05$).

Conclusion: Galectin-3 belongs to a class of inflammatory mediators that is associated with the degree of myocardial inflammation and fibrosis. It is related to the severity of myocardial ischemia and is negatively correlated with the cardiac ejection fraction.

1. Introduction

Coronary thrombosis is a major cause of mortality in the western countries and developing countries. The fundamental pathological process is due to the formation of atherosclerotic plaque which leads to artery wall thickening, along with the evolution of tube wall inflammation and fibrosis, ultimately resulting in acute coronary syndrome (ACS). ACS constitutes unstable coronary artery disease, from UAP to AMI. The characteristics of AMI include myocardial necrosis, myocardial tissue loss, infracted myocardial cell inflammation and fibrosis. The above can cause changes in the cardiac structure and biomechanics, which consists of ventricular collagen deposition, scar formation and myocardial hypertrophy…etc. Electrical remodeling, structural remodeling and contractile function remodeling are three essential pathological features of AF, and fibrosis is the symbol of proarrhythmia structure reconstruction [1]. Atrial fibrosis is more severe in patients with persistent AF than that in patients with paroxysmal AF [2].

Galectins exist in the cytoplasm, nucleus, on the cell surface and in the extracellular matrix of vertebrates. Amidst all galectins, Galectin-3 has a unique and a similar chimera structure; the single polypeptide chain forms two unique domains: the atypical N-terminal domain which is a tandem repeat composed of short amino acid fragments and the C-terminal carbohydrate-recognition domain (CRD).

Galectin-3 links with a series of extracellular matrix proteins, carbohydrates, nonglycosylated molecules and extracellular receptors (collagen IV) through CRD, and is involved in many pathophysiological processes, such as inflammation and fibrosis.

As a novel biological factor which can reflect inflammation and fibrosis, Galectin-3 may be involved in the evolution and the advancement of coronary heart disease and AF progression.

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2. Method

From June 2016 to December 2016, 70 consecutive patients were hospitalized in the cardiology department of Tianjin Medical University General Hospital, including 15 patients with AMI who underwent emergency PCI surgery, 20 patients with UAP, 20 patients with SAP, and 15 patients with AF who received radiofrequency ablation successfully after admission. This study was approved by the hospital Ethics Committee. All patients participated in the study after signing a written informed consent.

2.1. AMI patient identification

AMI patient identification includes: (1) typical chest pain duration for > 30 min; (2) the ST segment elevation/depression to a value > 0.1 mV and changed dynamically; (3) CK-MB elevated twice the normal value; (4) admitted within 12 h of disease onset without surgical contraindications; (5) all patients underwent emergency PCI treatment; (6) no statins taken out of hospital.

2.2. UAP identification

UAP identification: (1) typical chest pain lasting for < 30 min; (2) horizontal or slanted ST segments of adjacent leads, lowered down to > 0.1 mV; (3) negative troponin; (4) no statins taken out of hospital.

2.3. SAP identification

SAP identification includes: (1) the frequency of chest pain attacks and duration of chest pain remained unchanged in a period of three months; (2) the presence of ST segment changes in ECG during the chest pain attacks; (3) no statin consumption out of hospital.

2.4. Normal group

No clinical symptoms and no risk factors for coronary heart disease.

2.5. Exclusion criteria

Age > 80 years or < 18 years; statins taken out of hospital; ejection fraction of < 50%; combined with dilated cardiomyopathy, hypertrophic cardiomyopathy, valvular heart disease, rheumatic heart disease or hyperthyroid heart disease and other cardiovascular diseases; abnormal liver and renal function (serum creatinine > 2 mg/dl); combined with infection, tumor or immune system diseases; cerebral or peripheral vascular disease; myopathy or rhabdomyolysis and other systemic diseases.

Immediately after the hospitalization of AMI patients, 2 ml venous blood was drawn followed by the administration of atorvastatin 80 mg, Plaix 300 mg and aspirin 300 mg before PCI. On the second day after PCI, another 2 ml venous blood was drawn again and centrifuged. One milliliter blood serum was obtained from the centrifuged venous blood and stored at − 80 °C. From the second day onwards, atorvastatin dose was altered to 20 mg/d and other drugs were added according to the disease condition. Patients with UAP or SAP were given atorvastatin 20 mg/d after admission, and patients with AF were not given statins.

Similarly, patients with UAP or SAP or AF had 2 ml venous blood drawn on admission; another 2 ml venous blood was drawn from patients with AF after successful radiofrequency ablation. The venous blood drawn was treated according to the protocol mentioned above. Galectin-3 double antibody sandwich ELISA kit of CLOUD-CLONE CORP·WUHAN was used to detect serum samples, with a detection range of 0.156 ng/ml–10 ng/ml.

2.6. Biochemical examination

ALT, AST, CK, CK-MB, cTnT, renal function, NT-proBNP, blood lipids, high sensitive C reactive protein (Hs-CRP) and coagulation function were assessed in all the patients on the second day after admission to the hospital. After admission, the Left ventricular function of all patients was assessed by echocardiography. The general data and surgical data of the two groups were compared.

2.7. Coronary angiography

In addition to the patients with AF, the rest of the patients were selected for coronary angiography, and according to the results of coronary angiography patients were divided into single lesion group and multiple lesion groups.

2.8. Statistical methods

SPSS17.0 was used to analyze and process the data. The mean and standard deviation ($x \pm SD$) of all the measured data were calculated, and the two groups were compared using LSD test and variance analysis. Linear regression Pearson analysis was utilized to perform the correlation analysis, values with $P < 0.05$ infer statistical significance, and GraphPad. Prism was used to process drawings.

3. Result

General clinical data comparison between patients with coronary heart disease (CHD) and the control group, demonstrated no statistical significance between groups in terms of age, total cholesterol, triglycerides, high-density lipoprotein cholesterol, low density lipoprotein cholesterol, CTNT, RA, RV, and creatinine (see Tables 1–5; Figs. 1–5);

4. Discussion

Galectin-3 involves in the cardiac fibrosis, inflammation and reconstruction processes, it is a novel biomarker in heart disease [3]. In normal kidney, heart, brain, pancreas, liver and other organ tissues, Galectin–3 appears in minute levels [4], however, in pathological states, the expression of Galectin-3 is significantly increased.

In our experiment, for patients with coronary heart disease with normal cardiac function, Galectin-3 levels in plasma increased significantly with the gradual worsening of the myocardial ischemia. Plasma Galectin-3 levels of AMI patients were higher than that of UAP patients (0.96 ± 0.09 pg/ml vs. 0.77 ± 0.02 pg/ml), $P < 0.05$ vs. UAP group; Galectin 3 levels of AMI and UAP patients were higher than that of SAP patients (0.61 ± 0.01 pg/ml), $P < 0.05$ vs. SAP; But there was no significant difference between the SAP group and normal group. Role of Galectin-3 as a biomarker in the risk stratification of heart failure has been corroborated [5]. In our experiment, patients were selected with normal cardiac function in order to eliminate the effect of cardiac insufficiency on Galectin-3.We found that the plasma concentration of Galectin-3 was significantly related to the degree of myocardial ischemia.

Galectin-3 is involved in the pathogenesis and pathological process of atherosclerosis, such as inflammation, hyperplasia and macrophage