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Original article

Evaluation of galectin-3 levels in acute coronary syndrome

Évaluation des niveaux de la galectine-3 dans le syndrome coronarien aigu

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Abstract

Galectin-3 is a new biomarker that is assumed to reflect fibrogenesis and inflammation. In this study, we aimed to evaluate the levels of galectin-3 in patients with acute coronary syndrome (ACS) and the relation of galectin-3 to the burden of atherosclerosis. Nineteen patients with ACS who underwent coronary angiography and 17 age-matched healthy controls were enrolled. The burden of atherosclerosis was assessed with Gensini score and with the number of involved vessels. Galectin-3 levels were measured on admission by using ELISA. The mean age of the cohort was 62.8 ± 10.6 and 56% of the patients were male. Compared to control group, median galectin-3 levels were significantly higher in ACS patients (0.77 ng/mL [0.50–1.19] vs. 0.51 ng/mL [0.41–0.78], $P = 0.01$). Patients were classified into three groups according to the number of involved vessels. Median galectin-3 levels did not differ significantly among groups (one vessel: 0.68 ng/mL [0.55–0.74], two vessels: 0.67 ng/mL [0.46–1.84], three vessels 0.90 ng/mL [0.53–1.38], $P = 0.62$). There was a strong correlation between galectin-3 levels and Gensini score ($r = 0.625$, $P = 0.004$). In conclusion, galectin-3 levels were elevated in patients with ACS and there was a strong correlation between galectin-3 levels and Gensini score.

Keywords: Acute coronary syndrome; Atherosclerosis; Biomarker; Galectin-3; Gensini

Résumé

La galectine-3 est un nouveau biomarqueur qui est supposé refléter la fibrogenèse et l'inflammation. Dans cette étude, nous avons l'intention d'évaluer les niveaux de la galectine-3 pour les patients avec un syndrome coronarien aigu (SCA) et la relation de la galectine-3 à la charge de l'athérosclérose. Dix-neuf patients avec un SCA qui ont subi une angiographie coronarienne et 17 témoins sains appariés selon l'âge étaient inscrits. La charge de l'athérosclérose a été évaluée avec le score Gensini et le nombre de navires concernés. Les niveaux de la galectine-3 à l'admission ont été mesurés en utilisant le test ELISA. L'âge moyen de la cohorte était de $62,8 \pm 10,6$ et 56 % des patients étaient masculins. Par rapport au groupe témoin, les médianes de la galectine-3 étaient significativement plus élevées pour les patients avec SCA (0,77 ng/mL [0,50–1,19] vs 0,51 ng/mL [0,41–0,78], $p = 0,01$). Les patients ont été classés en trois groupes selon le nombre de navires concernés. Les médianes des niveaux de la galectine-3 n'ont pas différé significativement entre les groupes (un navire : 0,68 ng/mL [0,55–0,74], deux navires : 0,67 ng/mL [0,46–1,84], trois navires : 0,90 ng/mL [0,53–1,38], $p = 0,62$). Il y avait une forte corrélation entre les niveaux de la galectine-3 et le score Gensini ($r = 0,625$, $p = 0,004$). En conclusion, les niveaux de la galectine-3 ont été élevés pour les patients avec un SCA et il y avait une forte corrélation entre les niveaux de la galectine-3 et le score Gensini.

Mots clés : Athérosclérose ; Biomarqueur ; Galectine-3 ; Gensini ; Syndrome coronarien aigu

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1. Introduction

Despite significant improvements in diagnosis and treatment in the recent decades, cardiovascular diseases still remain as the leading cause of mortality and morbidity, with coronary artery disease being the major cause [1]. Acute coronary syndrome (ACS) represents the main concern to the investigators within the spectrum of coronary artery disease. Early diagnosis and risk stratification can alter the course of the disease and improve the survival. There have been a number of studies worldwide to find a useful biomarker in order to make an early and accurate diagnosis, predict prognosis and identify high-risk patients [2].

Galectin-3 is a soluble B-galactoside-binding lectin that mediates different pathways of inflammation and fibrosis [3]. It is expressed by activated macrophages and regulates several inflammatory cells including lymphocytes, neutrophils, monocytes and mast cells [4,5]. Studies have shown that galectin-3 induces the migration of monocytes and macrophages, triggers antioxidant secretion from active phagocytic cells, promotes fibroblasts proliferation and increases collagen synthesis [6–8]. A genetic mutation in galectin-3 has been shown to impede these pathways causing inadequate phagocytosis and impaired immune response [9]. Those properties emphasize the pivotal role of galectin-3 in inflammation and fibrosis, motivating numerous studies evaluating the function of galectin-3 in cardiovascular diseases [10–12]. Although several studies have been conducted to assess the association of galectin-3 in heart failure and remodeling, less is known about the role of galectin-3 in coronary artery disease and in ACS [11,12]. In this study, we aimed to evaluate the galectin-3 levels in patients with ACS and the relation of galectin-3 with the severity of the coronary artery disease.

2. Methods

2.1. Study patients

Nineteen patients with ACS who underwent coronary angiography and 17 age-matched controls without a history of coronary artery disease or heart failure were prospectively enrolled in our study. Patients diagnosed with ST and non-ST elevated myocardial infarction were included as ACS. Non-ST elevated myocardial infarction diagnosis was based on elevated cardiac enzymes with typical chest pain and/or electrocardiographic changes suggestive of myocardial ischemia. ST elevated myocardial infarction diagnosis was based on presence of prolonged chest pain and ST-segment elevation (>1 mm in two or more standard leads or ≥2 mm in two or more contiguous precordial leads), or the presence of new left bundle branch block. Routine treatment was initiated according to current ACS guidelines.

Information regarding risk factors including age, gender, diabetes mellitus, hypertension, hyperlipidemia, and smoking status was obtained. Hypertension was defined as blood pressure >140/90 mmHg on >2 occasions during office measurements or use of antihypertensive treatment. Diabetes mellitus was defined as fasting blood glucose >126 mg/dL or use of antidiabetic treatment. Hyperlipidemia was considered to be present in patients with fasting total cholesterol ≥200 mg/dL or triglyceride ≥150 mg/dL. Body mass index was calculated by dividing weight in kilograms by height in squared meters. Glomerular filtration rates were estimated with the Modification of Diet in Renal Disease equation [13]. Coronary artery disease was defined as documented coronary stenosis of >50%. The severity of the coronary artery disease was assessed by Gensini score and with the number of involved vessels [14]. Transthoracic echocardiography was performed for each patient immediately after hospitalization using a commercially available machine (Vivid 3®, GE Vingmed Ultrasound) with a 3.5-MHz transducer. Simpson's method was used to assess the left ventricular ejection fraction. The Institutional Research Ethics Committee approved the study and informed consent was obtained from each patient.

2.2. Laboratory

Venous blood samples were obtained immediately after index admission. Plasma glucose, aspartate aminotransferase, alanine aminotransferase, creatinine, troponin I, triglyceride, total cholesterol, and high-density lipoprotein cholesterol levels were measured by using standard methods. Low-density lipoprotein cholesterol levels were calculated according to the Friedewald formula [15]. Hemoglobin and white blood cell count values were measured using an automated hematology analyzer. Blood samples were centrifuged within 60 minutes of sampling, serum was isolated and stored at −20 °C for galectin-3 measurement. After collecting all serum samples, galectin-3 was measured by solid phase enzyme linked immunosorbent assay (ELISA) with a commercially available kit (eBioscience, San Diego, USA) at Hacettepe University, School of Sports Sciences Laboratory.

2.3. Statistics

Continuous variables were expressed as mean ± standard deviation or as median with interquartile range; and categorical variables were expressed as number and percentages. A χ^2 test or Fisher's exact test was performed to compare the categorical variables. Student's *t*-test was used for normally distributed continuous variables, and Mann-Whitney U test or Kruskal-Wallis test were used when the distribution was skewed. The optimal cut-off level of galectin-3 to detect ACS was evaluated by using the area under the receiver operating characteristic (ROC) curve. Spearman correlation analysis was performed to evaluate the relationship of galectin-3 with clinical and biochemical parameters. All statistical analyses were performed with SPSS software version 17.0 (SPSS Inc., Chicago, IL). A *P*-value of 0.05 was considered statistically significant.

3. Results

The mean age of the study participants was 62.8 ± 10.6, with men comprising 56% of the cohort. The baseline demographics, risk factors and laboratory findings of the enrolled subjects