Clinical Utility of Resistin as a New Marker of Acute Coronary Syndrome

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By

Mohamad Mahmoud Ahmad Awad

M.B., B.Ch
Ain Shams University

Supervised by

Professor /Sawsan Said Hafez

Professor of Clinical and Chemical Pathology
Faculty of Medicine –Ain Shams Univeristy

Professor / Mona Mostafa Osman

Professor of Clinical and Chemical Pathology
Faculty of Medicine –Ain Shams Univeristy

Assistant Professor / Nermine Helmy Mahmoud

Assistant Professor of Clinical and Chemical Pathology
Faculty of Medicine –Ain Shams Univeristy

Faculty of Medicine
Ain Shams Univeristy
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مقدمة من

الطبيب / محمد محمود أحمد عوض

بكالوريوس الطب والجراحة العامة
كلية الطب – جامعة عين شمس

تحت إشراف

الأستاذ الدكتور / سوسن سعيد حافظ

أستاذ الباثولوجيا الإكلينيكية والكيميائية

كلية الطب – جامعة عين شمس

الأستاذ الدكتور / منى مصطفى عثمان

أستاذ الباثولوجيا الإكلينيكية والكيميائية

كلية الطب – جامعة عين شمس

الأستاذ الدكتور / نيرمين حلمي محمود

أستاذ مساعد الباثولوجيا الإكلينيكية والكيميائية

كلية الطب – جامعة عين شمس

كلية الطب
جامعة عين شمس
٢٠١١

SUMMARY AND CONCLUSION

The present study included 80 subjects; 60 patients with ACS and 20 controls. Patients were classified into GIa (stable angina SA, n=20),GIb (unstable angina UA, n=20) and GIc (myocardial infarction MI, n=20) according to clinical picture, ECG and serum cardiac enzymes.

The aim of this study was to assess the clinical utility of resistin as a marker of vascular inflammation in patients with acute coronary syndrome and to assess the correlation of circulating resistin levels with other metabolic parameters and biomarkers of cardiac necrosis as troponin I, total CK, and CK-MB.This necessitated the study of serum levels of resistin in conjugation with the routinely measured cardiac markers including total CK, CK-MB, and troponin I in MI, UA and SA patients as well as the control group. The adopted assay methods were ELISA in case of resistin and spectrophotometrically in case of total CK and CK-MB. Meanwhile, troponin I was measured by chemiluminescent technique.

On assessment of the different studied parameters, it was found that serum resistin levels were significantly higher in patients with MI, UA and SA when compared to the control group. The serum

resistin levels were highly significantly increased in UA group compared to SA group, also the serum resistin levels were highly significantly increased in AMI patients compared to UA patients. Thus our study concluded that serum resistin levels increased with the pathogenic progress of CAD. A remarkable finding was that there was a significant positive correlation between serum resistin and total Ck and serum CK-MB fraction in MI patients, but there was no significant correlation between serum resistin and troponin I recorded in MI patients. Meanwhile in case of UA patients, there was a significant positive correlation between serum resistin and total CK, but there was no significant correlation between serum resistin, CK-MB and troponin I. Finally, there was no significant correlation recorded between resistin and total CK, CK-MB and troponin I in SA group.

Receiver operating characteristic curve (ROC) analysis was applied to assess the diagnostic utility of resistin in myocardial infarction patients versus healthy controls. The best diagnostic cutoff for resistin was 4.1 ng/mL. This had a diagnostic sensitivity of 95.2%, specificity 100%, negative predictive value 100%, positive predictive value 100% and efficiency 100%. The area under curve (AUC) was 0.9.

Also receiver operating characteristic curve (ROC) analysis was applied to assess the diagnostic performance of resistin for

discriminating myocardial infarction patients from angina patients. The best diagnostic cutoff for resistin was 5.8 ng/mL. This had a diagnostic sensitivity of 85.7%, specificity 82.5%, negative predictive value 94.3%, positive predictive value 72% and efficiency 85%. The area under curve (AUC) was 0.8.No other authors discuss the diagnostic performance of resistin in ACS.

In conclusion, serum resistin is proposed to be a valuable diagnostic marker for ACS as its level increase significantly with the pathogenic progress of CAD as it can differentiate with high ability between MI, UA and SA. Moreover, serum resistin must be introduced into the laboratory with the conventional markers of cardiac necrosis to help proper diagnosis and treatment of ACS patients.

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Aim of the work

The aim of the present study is to assess the clinical utility of resistin as a marker of vascular inflammation in patients with acute coronary syndrome and to assess the correlation of circulating resistin levels with other metabolic parameters and biomarkers of cardiac necrosis as troponin I, total CK, and CK-MB.

Introduction

Coronary artery disease (CAD) is increasing in prevalence and predicted to become the dominant cause of mortality worldwide by 2020 **(Buffon et al., 2002).**

Acute coronary syndromes (ACS) result from acute obstruction of a coronary artery. Consequences depend on the degree and location of obstruction **(Corti et al., 2003).** The subtypes of acute coronary syndrome include unstable angina (UA) not associated with heart muscle damage and two forms of myocardial infarction (MI), in which heart muscle is damaged. These types are named according to the appearance of the electrocardiogram (ECG) as non-ST segment elevation myocardial infarction (NSTEMI) and ST segment elevation myocardial infarction (STEMI). ACS should be distinguished from stable angina, which develops during exertion and resolves at rest. In contrast to stable angina, unstable angina occurs suddenly, often at rest or with minimal exertion **(Grech and Ramsdale, 2003).**

Differentiating acute coronary syndrome from non cardiac chest pain is the primary diagnostic challenge. The main diagnostic categories of acute coronary syndrome, unstable angina and myocardial infarction are defined by the serum concentration of cardiac enzymes and markers **(Fox et al 2004).** Although new biomarkers as cardiac troponins have increased the abilities to detect and or exclude cardiac injury a normal troponin measurement is not

synonymous with a lack of risk. Accordingly, there are still unnecessary admissions to expensive coronary care units, step-down units and non-intensive care beds when discharge might be equally appropriate because the presentation of an ACS is often atypical and because a missed diagnosis of ACS carries a considerable risk to patients **(Jaffe and Katus, 2004).**

Presence of new markers that would further define risk would not only reduce the number of patients admitted to the hospital, but would also allow for prevention of substantial numbers of new events **(Bertrand et al., 2002)**

Resistin is a hormone secreted by adipose tissue. It is also known as "serine/cysteine-rich adipocyte-Specific Secretory Factor" (ADSF or FIZZ3). The length of the resistin pre-peptide in human is 108 aminoacids; the molecular weight is ~12.5 kDa **(Degawa et al., 2003).**

Resistin may contribute to the atherosclerotic process by activation of endothelial cells leading to endothelial dysfunction and thereby stimulating multiple pro-atherosclerotic pathways **(Fan et al., 2007).**

SUBJECTS AND METHODS

I .SUBJECTS:

This study was conducted at Ain Shams University Hospital on 60 patients with acute coronary syndrome and 20 apparently healthy subjects serving as control group.

A. Patients Group (Group I, n = 60):

This group included 38 males and 22 females. Their ages ranged between 37 and 83 years with a mean age of 62.6 ± 10.7 years. They were selected from the Cardiology Unit at Ain Shams University Hospital. They were subdivided into groups according to their diagnosis, which was based on clinical picture, ECG findings and serum cardiac biomarkers values as follows:

1. Subgroup Ia (stable angina SA, n=20):

This group included patients with stable angina (episodic pain lasting 5-15 minutes that is precipitated by exertion and relieved by rest or nitroglycerine - no ECG abnormalities - normal cardiac biomarkers). They were 13 males and 7 females. Their ages ranged between 47 and 80 years with a mean age of 64 ± 9.4 years.