The Egyptian Journal of Medical Human Genetics (2014) 15, 241–247

Ain Shams University

The Egyptian Journal of Medical Human Genetics

www.ejmhg.eg.net
www.sciencedirect.com

ORIGINAL ARTICLE

Impact of copeptin on diagnosis of acute coronary syndrome

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Received 14 January 2014; accepted 9 February 2014
Available online 17 March 2014

KEYWORDS

Copeptin;
Acute coronary syndrome;
Acute myocardial infarction;
Unstable angina pectoris

Abstract *Background:* Acute coronary syndrome remains the principal cause of death, so the early diagnosis is of great importance. Cardiac troponin is the preferred biomarker for acute myocardial infarction. Cardiac chest pain immediately increased copeptin secretion. The combination of copeptin and cardiac troponin I is being suggested for early diagnosis of acute coronary syndrome.

Subject: It was done to emphasize the importance of association of copeptin, cardiac troponin I and high sensitive C reactive protein to confirm the diagnosis of acute myocardial infarction or unstable angina pectoris in patients with a cardiac chest pain.

Method: The current study enrolled 22 patients with acute myocardial infarction as group i and 33 patients with unstable angina pectoris as group ii. The third group consisted of 23 apparently healthy persons. Patients and controls were subjecting to laboratory investigations, which include the levels of copeptin, high-sensitivity cardiac troponin high sensitive C reactive protein creatine kinase MB fraction, lipid and I profile.

Results: We found a significant increase of copeptin in group i when compared to group iii (30.01 ± 12.92) (9.54 ± 3.55), respectively, p value = 0.000 and group ii (30.01 ± 12.92) (11.16 ± 4.58) respectively, p value 0.000, but a non-significant difference in group ii when compared to group iii (11.16 ± 4.58) (9.54 ± 3.55) respectively, p value = 0.160. Also cardiac troponin

Abbreviations: ACS, acute coronary syndrome; cTn, cardiac troponins; AMI, acute myocardial infarction; UAP, unstable angina pectoris; AVP, arginine vasopressin; hs-cT I, high-sensitivity cardiac troponin I ; hs-CRP, high sensitive C reactive protein; CK-MB, creatine kinase MB fraction.

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Peer review under responsibility of Ain Shams University.

Production and hosting by Elsevier

http://dx.doi.org/10.1016/j.ejmhg.2014.02.005

I showed a significant increase in group i when compared to group ii (136.73 ± 26.07) (11.18 ± 3.79), p value = 0.000, and group iii (136.73 ± 26.07) (9.61 ± 3.70) respectively, p value = 0.000, but a non-significant difference between group ii (11.18 ± 3.79), and group iii (9.61 ± 3.70), p value = 0.129. There was a positive correlation between copeptin and cardiac troponin I within group i, r = 0.718, p value = 0.000.

Conclusion: In suspected acute coronary syndrome, determination of copeptin and cardiac troponin I provides a remarkable negative predictive value, which aids in early and safe ruling out of myocardial infarction.

1. Introduction

Acute coronary syndrome (ACS) remains the principal cause of death around the world in 2011 [1]. Diagnosis and accurate exclusion are of great importance in the emergency department to ensure early effective treatment. Cardiac troponins (cTn) are the preferred biomarkers for detection of myocardial cell necrosis and they are essential for the diagnosis of acute myocardial infarction (AMI) [2]. However, there remains a troponin-blind interval after onset of chest pain due to the delayed release of cTn following a cardiac injury [3], and requires repeat measurement of cTn to discriminate between AMI and unstable angina pectoris (UAP) [4]. Therefore, there is a need for a biomarker that is released immediately in the event of AMI [3]. The hope is that this new biomarker will enable early decision making in clinical practice. AMI activates the main hypothalamic stress hormone, arginine vasopressin (AVP) [2]. Hence, AVP becomes an important marker; however, due to its unstable nature and rapid clearance from plasma, measurements of AVP are rarely reproducible [5]. Copeptin is stored in the neurohypophyseal vesicles together with AVP until they are secreted [2]. Copeptin is stable and easier to measure. Therefore, its level represents the production of AVP. Thus, copeptin is a mirror of AVP concentration with high prognostic accuracy [6]. Therefore, in patients with ACS, the new biomarker copeptin as a marker of acute endogenous stress [7] is expected to be elevated very early after AMI [8]. Therefore, the combination of a marker of endogenous stress and a marker of cell necrosis has been suggested to improve the diagnostic performance in chest pain patients at presentation in the emergency department [9].

1.1. Aim of work

This study was done to emphasize the importance of association of copeptin, cardiac troponin i, high sensitive C reactive protein and creatine kinase MB fraction, to confirm the diagnosis of acute myocardial infarction or unstable angina pectoris in patients with a cardiac chest pain.

2. Patients and methods

2.1. Patients and controls

AMI was the final diagnosis in 22 patients (14 males and 8 females), their ages ranged between 46 and 73 years, and 33 patients with UAP (24 males and 9 females), and their ages ranged between 47 and 71 years. They presented to the emergency department of the National Heart Institute, Imbibe, Giza, Egypt, complaining of typical cardiac chest pain between January 2013 and June 2013. Patients with hepatic or renal disease were excluded. Other 23 apparently healthy persons of matching age served as the healthy control group of this study for baseline normal value assessment. All investigations and diagnosis of AMI and UAP were performed in accordance with a standardized protocol in the National Heart Institute, Health and Human Ethics Clearance Committee guidelines for Clinical Researches. Local ethics committee approved the study protocol and informed consents were obtained from all subjects.

Patients and controls were divided into the following groups (According to the current guidelines) [10]:

1- AMI patients as group i: consisted of 22 patients with a mean age/years and SD of 59.59 ± 6.75.
2- USP as group ii: consisted of 33 patients with a mean of age/years and SD of 58.82 ± 6.20.
3- Healthy control as group iii: consisted of 23 age matched, with mean age/years and SD of 56.04 ± 6.27.

Comprehensive adult health history was taken and comprehensive physical examination was done for all studied participants.

All studied participants were subjected to the following:

1- Standard 12-lead electrocardiography (ECG).
2- Trans-thoracic echocardiography.
3- Chest radiography (postero-anterior and lateral).
4- Laboratory investigations:-
   a) Serum random blood glucose level.
   b) Serum lipid profile (total cholesterol, serum low-density lipoprotein – cholesterol (LDL-c) and triglycerides).
   c) Liver function tests (total serum bilirubin, total serum protein, prothrombin time, and international normalized ratio).
   d) Renal function tests (serum creatinine, serum blood urea).
   e) Total serum creatine kinase (CK).
   f) Serum creatine kinase-MB fraction (CK-MB) .
   g) Serum highly sensitive cardiac troponin i (hs-cTn I).
   h) Serum copeptin.
   i) Serum highly sensitive C reactive protein (hs-CRP).

2.2. Sampling collection

- Five ml of venous blood was withdrawn, under aseptic condition, from each patient and control. Then it was divided as follows: