Case Report / *Olgu Sunumu*

DOI: 10.4274/Haseki.850

Unexplained Elevated Troponin

Açıklanamayan Troponin Yüksekliği

Okay Abacı, Veysel Oktay, Cüneyt Kocaş, Şükrü Aslan, Zerrin Yiğit

Istanbul University Institute of Cardiology, Department of Cardiology, Istanbul, Turkey

Abstract

Although cardiac troponins play a central role in the diagnosis and risk stratification of patients with acute coronary syndrome and are frequently used to determine whether a patient with chest pain has had cardiac damage, a false positive value can result in unnecessary hospitalization and enrollment of potentially invasive tests. Clinicians should be aware of the possibility of false positive troponin levels, especially when the results are not consistent with clinical presentation of the patient. *(The Medical Bulletin of Haseki 2012; 50: 153-5)*

Key Words: Troponin, acute coronary syndrome

Özet

Kardiyak troponinler akut koroner sendromların tanı ve risk sınıflamasında merkezi bir rol oynayıp göğüs ağrısının nedeninin kardiyak hasara bağlı olup olmadığının belirlenmesinde kullanılsa da yanlış pozitif sonuçlar hastaların gereksiz hospitalize edilmesine ve invazif işlemlerin uygulanmasına yol açabilir. Klinisyenler hastaların klinik özellikleri troponin seviyeleri ile korelasyon göstermediği durumlarda, troponin yüksekliğinin yanlış pozitif olabileceği ihtimalini göz önünde bulundurmalıdır. *(Haseki Tıp Bülteni 2012; 50: 153-5)*

Anahtar Kelimeler: Troponin, akut koroner sendrom

Introduction

In the setting of acute coronary syndromes, troponin levels are indicator for the therapeutic decision-making. In this paper, we aimed to draw attention to false positive troponin which can confound the interpretation of the clinical status of patients and cause application of unnecessary interventions.

Case

A 46-year-old woman was admitted to our emergency service with a complaint of chest pain. The pain that started eight hours ago was sharp, worsened by breathing and in supine position indicating a non ischemic origin. She had no cardiac risk factors and was not taking any medication, but her medical history was surprising with hospitalization four times because of elevated troponin results in different medical departments within the last two years. In these hospitalizations, two angiographic studies were performed and it was observed that she had entirely normal coronary anatomy. The patient's physical examination was normal and electrocardiogram showed sinus rhythm without ischemic changes (Figure 1). Chest X-ray examination demonstrated normal findings without cardiac enlargement and pulmonary consolidation. Biochemical test revealed that total CPK and CK-MB activity were within normal range except for a high level troponin of 2 ng/ml (normal value <0.014 ng/ml) which remained elevated constantly during her hospital stay. Bedside echocardiography showed preserved left ventricular systolic and diastolic functions with normal wall thickness and motions. In order to exclude a possible pulmonary embolism or aortic dissection, thorax computed tomography (CT) angiography was performed and no pathology was found. We also performed coronary angiography for definitive diagnosis of troponin elevation and the result was normal as the previous examinations (Figure 2). Provocation test for determining vasospastic angina was negative. Other clinical laboratory tests that can imitate positive troponin levels such as thyroid function tests, rheumatologic panels (anti nuclear antibody,anti ds DNA, C-ANCA, P-ANCA, Lupus anticoagulant, Anti Sm, Anti RNP, Anti SS A, Anti SS B, Anti Jo 1, Anti Scl 70), coagulation tests (Protein C, Protein S, Factor 5 Leiden mutation test) had no diagnostic ability to

Address for Correspondence/Yazışma Adresi: Okay Abacı
Istanbul University Institute of Cardiology, Department of Cardiology, Istanbul, Turkey
E-mail: drokayabaci@hotmail.com

Received/Geliş Tarihi: 26 Eylül 2012 **Accepted/Kabul Tarihi:** 26 Eylül 2012

explain elevated troponin values. Reanalyses of serum samples for excluding any assay interferences by different laboratories revealed the results 3 consistent with our laboratory results. The patient denied any exposure to mice, rodents or other animals. At discharge, troponin levels were still elevated with normal CPK and CK-MB levels. Finally, we reached the conclusion that it was an extraordinary case by having idiopathic elevated troponin levels.

Discussion

There are three troponin proteins which might be detected both in cardiac and skeletal muscles. Cardiac troponin C (cTnC) is identical to the troponin C expressed in skeletal muscle. However, both cardiac troponin T (cTnT) and troponin I (cTnI) derive from genes specifically expressed within myocardial tissue that called cardiac-specific troponins (1). Cardiac-specific troponins (I or T) play an important role in the diagnosis, therapeutic decision-making and early risk stratification in patients with suspected acute coronary syndromes (2). In healthy individuals, cardiac troponins do not exist in plasma but do pass into the bloodstream with myocardial cell injury caused by different conditions (3). These biomarkers reflect myocardial damage but do not demonstrate its causes. Although cardiac-specific troponins are the preferred biomarkers because of high sensitivity and specificity, a false-positive result may be observed in some cases. The most common cause of an increased troponin level is atherosclerotic heart disease. However, there can be many causes of troponin elevation such as cardiac and non-cardiac conditions e.g. tachycardia, acute heart failure, myopericarditis, pulmonary embolism, sepsis, stroke, heavy exercise, hypothyroidism, gastrointestinal bleeding, rheumatologic diseases, cardiac contusion, severe renal dysfunction, cardiotoxic drugs, heterophilic antibodies (4,5,6). In our case, these possible diagnoses were tried to be excluded by clinical and laboratory tests such as ECG, echocardiography, thorax CT, coronary angiography, biochemical parameters and complete blood count. Re-analysis of serum samples excluding for heterophilic antibody-dependent analytic errors by different laboratories revealed the results consistent with our laboratory results. Rheumatic parameters were investigated in order to eliminate the presence of rheumatoid factor interference with cardiac troponin assay. The patient denied any drug use, thus, Kounis syndrome (7) was excluded.

Although cardiac troponins play a central role in the diagnosis and risk stratification of patients with acute coronary syndromes and frequently used to determine whether a patient with chest pain has had cardiac damage, sometimes increased troponin levels cannot be explained despite detailed clinical examination as in our case. If the test results are not consistent with patient's clinical presentation, a false-positive result must be considered and should not be forgotten that the patient may be have chronically elevated troponin.

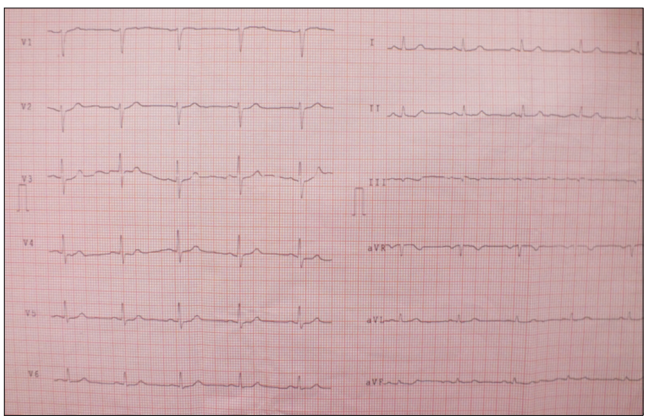

Figure 1. Electrocardiogram showed sinus rhythm without ischemic changes

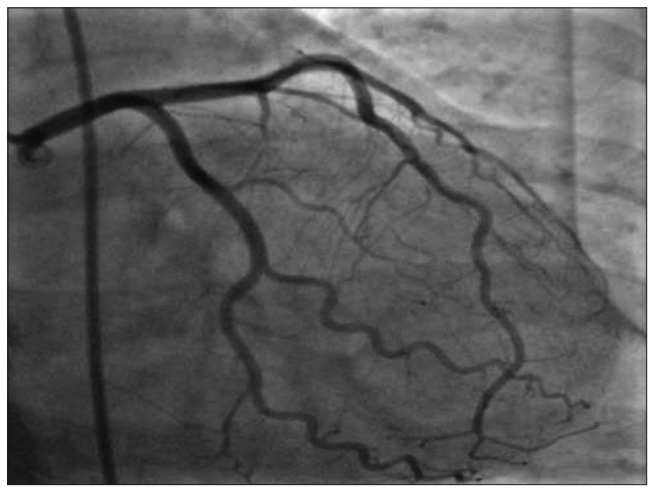

Figure 2a. Angiography of leftcoronary arteries

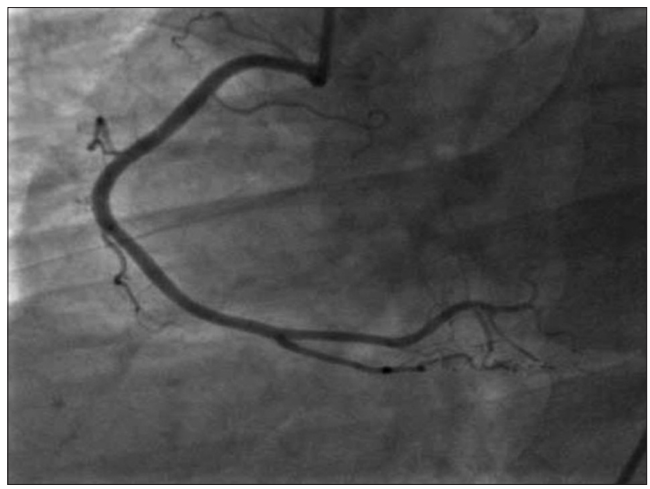

Figure 2b. Angiograğhy of rightcoronary artery

References

1. Coudrey L. The troponin. Arch Intern Med 1998;158:1173-80.
2. Hamm CW, Bassand JP, Agewall S, et al. ESC Guidelines for the management of acute coronary syndromes in patientspresenting without persistent ST-segment elevation: The Task Force for the management of acute coronary syndromes (ACS) in patients presenting without persistent ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J 2011;32:2999-3054. doi: 10.1093/eurheartj/ehr236. Epub 2011 Aug 26.
3. Antman EM. Decision making with cardiac troponin tests (editorial). N Engl J Med 2002;346:2079-82.
4. Jaffe AS, Babuin L, Apple FS. Biomarkers in acute cardiac disease:the present and the future. J Am Coll Cardiol 2006;48:1-11. Epub 2006 Jun 12.
5. De Gennaro L, Brunetti ND, Cuculo A, et al. Increased troponin levels in non-ischemic cardiac conditions and non-cardiac diseases. J Interv Cardiol 2008;21:129-39. Epub 2008 Jan 28.
6. Kenny PR, Finger DR. Falsely elevated cardiac troponin-I in patients with seropositive rheumatoid arthritis. J Rheumatol 2005;32:1258-61.
7. Calogiuri GF, Nettis E, Di Leo E, Vacca A, Ferrannini A, Kounis NG. Kounis Syndrome induced by intravenous administration of piperacillin/tazobactam: a case report. Int J Cardiol 2012;155:e42-4. Epub 2011 Aug 11.