OLGU SUNUMU • CASE REPORT

Kosuyolu Kalp Derg 2013;16(1):65-68 • doi: 10.5578/kkd.3625

A Case Report of Unexplained Isolated Elevation of Troponin

Açıklanamayan Yalın Troponin Yüksekliği Olgusu

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ABSTRACT

Cardiac troponins have high clinical sensitivity and specificity for the detection of myocardial injury. Elevated cardiac troponin levels may also occur in non cardiovascular conditions, leading to a misdiagnosis of acute coronary syndrome. In this paper, we report a case of unexplained isolated elevation of troponin.

Key Words: *Troponin, acute coronary syndrome.*

Received: *17.04.2012* • ***Accepted:*** *03.05.2012*

ÖZET

Kardiyak troponinler miyokart enfarktüsü tanısında yüksek duyarlık ve özgüllüğe sahiptir. Yüksek troponin seviyeleri bazen kalp dışı nedenlerle ortaya çıkar ve yanlışlıkla akut koroner sendrom tanısı konulabilir. Bu çalışmada izole troponin yüksekliği saptanmış bir olguyu sunuyoruz.

Anahtar Kelimeler: *Troponin, akut koroner sendrom.*

Geliş Tarihi: *17.04.2012* • ***Kabul Tarihi:*** *03.05.2012*

INTRODUCTION

Cardiac troponins remain the gold standard markers for use in patients with acute coronary syndromes. The success of troponin is due to its high clinical sensitivity and specificity in the diagnosis of myocardial injury[(1)]. However, these results may also be seen in non cardiac conditions, leading a misdiagnosis of acute coronary syndrome. In this paper, we report an unusual case of unexplained isolated elevation of troponin with no documented cause.

CASE REPORT

A 38-year old woman was referred to an emergency department with the suspicion of acute coronary syndrome. Her chest pain that started one day ago was atypical, sharp and worsened by breathing. She was transferred to our cardiology department

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for further investigation due to elevated troponin levels. She had no previous diseases or cardiac risk factors except a family history of premature coronary artery disease. She was not taking any medicine regularly, but had had a tooth extracted one week ago, and had received ciprofloxacin and dexketoprofen trometamol therapy for one day. Her physical examination was entirely normal. Electrocardiogram (ECG) and chest X-ray were normal (Figure 1).

The results of laboratory tests, including blood count and biochemical analysis, were normal except the elevation of cardiac troponin-I: 96 ng/mL (normal laboratory level < 0.04 ng/mL) and eosinophilia: 6.2% (0.9-2.9%). Creatine kinase-MB level was also in the normal ranges: 0.9 ng/mL (normal laboratory level 0.6 6.39 ng/mL). For differential diagnosis, echocardiography was performed first. Echocardiography revealed normal cardiac functions, and an ejection fraction of %67. Interventricular septum and posterior wall thickness were normal. There was also no signs of myocarditis or pericarditis. In order to verify the diagnosis of a possible pulmonary embolism and aortic dissection, computed tomography angiography was performed and revealed normal lungs and aorta. Coronary angiography and ventriculography performed for a definitive diagnosis of coronary artery disease were all normal (Figure 2). Additionally, a provocation test for vasospastic angina was negative.

Given the normal coronary arteries and ventricular functions of patient in the setting of elevated troponin levels, detailed biochemical and immunological tests were performed. Thyroid function tests, coagulation tests, factor V Leiden mutation analysis, protein S, protein C, anti-ds-dna, anti-nuclear antibody, C-ANCA, P-ANCA, Ena-jo1, Ena-scl 70, Ena-sm, Ena-ssa, Ena-ssb, lupus anticoagulant, Pro-bnp, sedimantation rate and rheumatoid factor levels were within normal ranges. Only C-reaktive protein (CRP) levels were found to be high: 24.40 (< 5 mg/L) and there was also a slight increase in homocysteine levels: 15.9 IU/mL (< 15 IU/mL). A microscopic examination of urine samples revealed 18 to 20 leukocytes and bacteria but there were no bacteria in urine culture.

The chest pain improved after the patient was admitted to the coronary care unit. No further pain or electrocardiographic changes were noted. Vital signs were stable and troponin levels remained elevated for the following four days. A reanalysis of the samples by our laboratory revealed the same results. No other direct causes of interference were found by the laboratory. The final troponin level was 96 ng/mL before the patient's discharge, and CK-MB and CK levels were not elevated. Our diagnosis was unexplained isolated elevation of troponin with no documented cause. The patient was administered pantoprazole, naproxen sodium and cefuroxime axetil for urinary disease, and outpatient clinic controls were recommended but she has not attended so far.

DISCUSSION

Although troponin is considered a specific marker for the diagnosis of acute coronary syndrome, recent studies have shown elevated troponin in a variety of non-ischemic conditions[2]. Other cardiovascular diseases where elevated troponin levels may be encountered are; acute pulmonary embolism, aortic dissection, left ventricular hypertrophy,

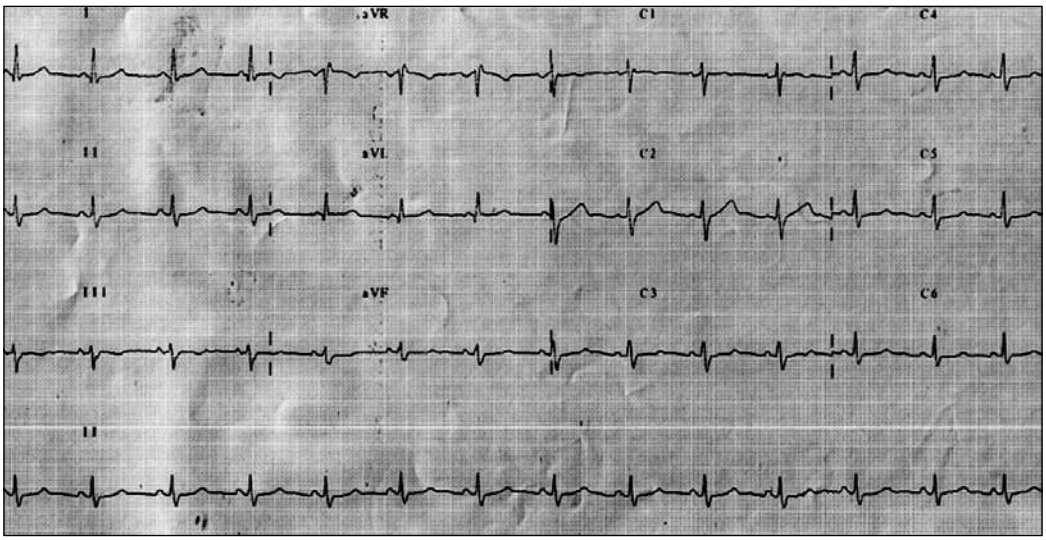

Figure 1. Electrocardiogram.

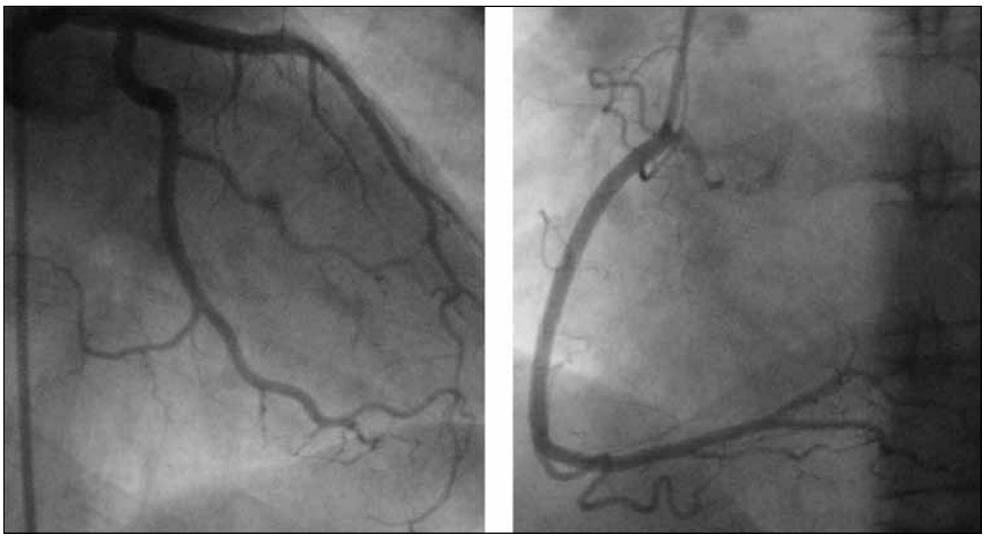

Figure 2. Right anterior oblique caudal view of the left angiogram and right anterior oblique view of the right angiogram.

tachycardia, myocardial bridging, aortic valve disease, chronic heart failure, non-ischemic cardiomyopathy, chemotherapy induced cardiomyopathy, cardioversion-cardioverter discharges, ablation, cardiac surgery, cardiac contusion, cardiotropic viral infection, endocarditis, myocarditis, pericarditis and human heart transplantation. Troponin levels may also be elevated in non-cardiovascular conditions like renal insufficiency, septic shock, gastrointestinal bleeding, cirrhosis, diabetic ketoacidosis, hypothyroidism, chronic obstructive pulmonary disease, aneurysmal subarachnoid hemorrhage, ischemic and hemorrhagic stroke, seizure, strenuous endurance exercise, rhabdomyolysis, pregnancy, rheumatologic disease, and also, not uncommonly, in traumatic noncardiac surgery[3].

In this case, we excluded most of the causes of troponin elevation by coronary angiography, echocardiography, thoracic CT angiography and laboratory tests. Thoracic CT angiography was helpful in clarifying that the patient's diagnosis was not aortic dissection or acute pulmonary embolism which may elevate troponin with an atypical chest pain. Laboratory findings were not helpful in establishing the diagnosis. Some virology tests such as CMV, EBV, coxsackie virus antibodies were not investigated due to laboratory limitations. Myopericarditis might have developed due to the viral agents, however, given normal CK-MB levels and normal ECG and echocardiography, myopericarditis was excluded as a diagnosis. Acute coronary syndrome and vasospastic angina were also excluded because of normal coronary angiography, atypical presentation, and a negative provocation test. As the patient presented late and CK-MB levels were normal we are not sure about spontaneous fibrinolysis of coronary artery thrombosis. We also suspected a rare syndrome, the Kounis syndrome, because of eosinophilia, high CRP levels, isolated troponin elevation, previous drug history and normal coronary arteries at coronary angiography. Kounis syndrome, also known as allergic angina syndrome, was described in 1991 by Kounis and Zafras as "the coincidental occurrence of chest pain and allergic reactions accompanied by clinical and laboratory findings of classic angina pectoris caused by inflammatory mediators released during the allergic insult"[4]. Additionally, some drugs are thought to be responsible for the Kounis syndrome. The Kounis syndrome presents with ST segment elevation as well as elevated CK-MB and troponin levels. Only one case of Kounis syndrome has been reported with no ECG changes, as in our case[5]. There is no report of ciprofloxacin associated Kounis syndrome in the literature. The patient presented in this case report had normal CK-MB levels and ECG readings in addition to atypical chest pain, which made us exclude the possibility of Kounis syndrome.

Certain effects on immunoassays of heterophilic antibodies could also be the cause of elevated troponin level but the troponin values reported by the epicenter and our laboratory were almost the same, which made us consider that the results obtained are reliable. The main limitation of this report was inadequate follow up of the patient.

Determination of troponin levels is essential for the rapid diagnosis of acute coronary syndromes. The rising and/or falling pattern of troponin levels has a crucial va-

lue in distinguishing chronic, nonischemic causes from acute ischemic injuries. Hence, unexplainable elevations of troponin may be seen in some patients, as in our case, and more specific and sensitive cardiac biomarkers may help overcome the diagnostic difficulties in the near future.

CONFLICT of INTEREST

None declared.

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