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A Rare Cause of Non-Cardiac Troponin Elevation: Acute Cholecystitis; Case Report

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ABSTRACT:

A rare cause of non-cardiac troponin elevation: acute cholecystitis; case report

Objective: Cardiac troponins are accepted as the standard markers for diagnosis and risk management of patients with acute coronary syndromes. However, cardiac troponins are elevated in some patients presenting with non-cardiac situations. In this paper, a rare case of non-cardiac troponin elevation with acute cholecystitis is presented.

Case: A 56 year old female patient admitted to the emergency room with pain in the epigastric region. The abdominal ultrasound revealed acute cholecystitis. She had some coronary risk factors and electrocardiogram showed no segment deviation but troponin was positive. Acute coronary syndrome couldn't be ruled out and coronary angiography revealed normal coronary arteries. Therefore, elevated troponin was linked to acute cholecystitis.

Conclusion: Although troponin elevation is a cornerstone marker for diagnosis the patients with acute coronary syndromes, there are various conditions with non-coronary-related troponin elevations. This can lead to misdiagnosis and unnecessary interventions. As a result, in patients with similar sypmtoms (pain in the epigastric region), electrocardiogram wave changes and elevated troponin levels, acute cholesystitis diagnosis should be considered.

Keywords: Acute cholecystitis, acute coronary syndrome, troponin

ÖZET:

Nadir görülen non-kardiyak troponin yüksekliği nedeni: Akut kolesistit; olgu sunumu

Amaç: Kardiyak troponinler miyokard infarktüsünün tanı ve risk değerlendirilmesinde standart belirteçler olarak kabul edilmiştir. Ancak kardiyak troponinler akut koroner sendrom dışı nedenle başvuran bazı hastalarda da yüksek bulunmaktadır. Burada nadir görülen bir kardiyak dışı troponin yüksekliği akut kolesistit vakası sunulmuştur.

Olgu: Acil servise epigastrik bölgede ağrı ile başvuran 56 yaşında bayan hastanın yapılan batın ultrasonografide akut kolesistit lehine bulgular izlendi. Koroner arter hastalığı açısından risk faktörleri olan hastanın elektrokardiyografi bulguları normal olmakla birlikte bakılan troponin pozitif saptandı. Akut koroner sendrom ekarte edilemeyen hastanın yapılan koroner anjiyografisinde koroner arterleri normal saptandı ve hastadaki troponin yüksekliği akut kolesistite bağlandı.

Sonuç: Troponin yüksekliği akut koroner sendromlarda bir belirteç olarak kullanılsa da, akut koroner sendrom dışı nedenle başvuran bazı hastalarda da yüksek bulunmaktadır. Bu durum yanlış tanılara ve gereksiz girişimlere yol açabilmektedir. Sonuç olarak benzer semptomları olan (epigastrik bölgede ağrı), elektrokardiyografide değişiklikleri olan ve troponin yüksekliği olan hastalarda akut kolesistit tanısı akılda tutulmalıdır.

Anahtar kelimeler: Akut kolesistit, akut koroner sendrom, troponin

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INTRODUCTION

Cardiac troponins (cTnT and cTnI) are accepted as the gold standard for the diagnosis of acute myocardial infarction (AMI), which are the most specific and sensitive laboratory markers indicating myocardial cell damage (1,2). In clinical practice, cardiac troponin elevations are common and can occur as a result of many acute coronary syndromes, as well as other clinical conditions, which can lead to inaccuracies and even unnecessary invasive or non-invasive therapeutic interventions. Here we present a rare case of high non-cardiac troponin elevation.

CASE REPORT

A 56-year-old female patient with known hypertension was admitted with abdominal pain to emergency room. She did not describe chest pain and shortness of breath. Vital findings were as follows; blood pressure: 132/78 mmHg, heart rate: 84/min, respiratory rate: 12, oxygen saturation: 98%, body temperature: 36.8°C. In the abdominal examination, there were sensitivitiy at the right upper quadrant and the epigastrium, and the Murphy's sign was negative. Pulmonary and cardiovascular examination were normal. The patient's ECG (electrocardiography) was at sinus rhythm and was evaluated as normal. Abdominal ultrasonography showed that the bile duct was mildly distended, its walls were slightly thickened and no intraluminal pathology was detected. No pericholecystic fluid was found, choledoch had normal width. The biochemistry of the patient was as follows: leucocyte: 11800/µL (4500-10500/µL), creatinine: 0.8 mg/dL (0-1.17 mg/dL), sodium: 142 mmol/L (135-148 mmol/L), potassium: 4.3 mmol/L (3.5-5.5 mmol/L), aspartate aminotransferase: 70 U/L (0-40 U/L), alanine aminotransferase: 86 U/L (0-41 U/L), alkaline phosphatase: 103 U/L (30-120 U/L), total bilirubin: 0.87 mg/dL (0.3-1.2 mg/dL), hsCRP: 17.8 mg/L (0-5 mg/L), D-Dimer: 170 ng/L (0-500 ng/L), troponin I: 3.25 ng/ml (0-0.06 ng/ml). The chest X-ray of the patient was normal. The patient whose abdominal pain decreased in follow up, was evaluated as acute cholecystitis as a result of general surgery consultation, and emergency surgery was not considered. Acute coronary syndrome was suspected, and antiischemic treatment (heparin, clopidogrel, acetylsalicylic acid, ramipril, nitroglycerin, metoprolol, atorvastatin) was initiated. On day 4 of the follow-up of the patient, biochemical values returned to normal, while troponin I was found to be 2.81 ng/ml. Left ventricular systolic functions were normal in the echocardiography, there was no left ventricular wall motion abnormality and evidence of myocarditis, pericarditis were not found in the patient. However, coronary angiography was performed to exclude acute coronary syndrome in the patient whose troponin value was still high. Elevated troponin levels in the patient with normal coronary arteries at coronary angiography was associated with acute cholecystitis.

CONCLUSION

Although elevation of troponin is used as a marker in acute coronary syndromes, it is also found as high in some patients admitted with a cause other than acute coronary syndrome (1,2). This can lead to misdiagnosis and unnecessary interventions. Myocardial troponin release suggests transient or permanent myocardial damage. This damage may be due to a number of causes such as ischemia, inflammation, infection, toxins, increased ventricular wall tension. Elevated troponin may be due to non-cardiac causes such as pulmonary embolism, central nervous system diseases (intracranial hemorrhage, intracranial pressure increase, ischemic stroke), aortic dissection, pneumothorax, acute cholecystitis, pancreatitis, etc., and ST-T wave change in ECG may be observed in these cases (3-6). There are no clear data in literature regarding troponin elevation and ST-T wave changes in ECG during acute cholecystitis. Coronary arteries were found normal in coronary angiography performed in 3 cases with ST segment elevation in ECG. ECG changes returned to normal after cholecystectomy and antibiotherapy in acute cholecystitis, and these changes were observed to be the result of coronary vasospasm (cardiobiliary reflex) (7). In the animal studies, it was

determined that the flow in the coronary arteries decreased after the biliary conduits were tied in dogs and pigs (8,9). In conclusion, acute cholecystitis, which can cause symptoms similar to acute coronary syndrome (pain in the epigastric region), ST-T wave changes in ECG, and troponin elevation, should be kept in mind in the differential diagnosis of acute coronary syndrome.

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