The Dilemma of Two Diagnoses for One Patient

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Abstract: Myocarditis is a myocardium disease where myocytes are damaged as a result of inflammatory infiltration. It was found out that chest pain accompanied by fear of death and feeling of sickness of a 49-year-old female patient applying at the emergency room with chest pain complaint was continuing uninterruptedly for the last 30 minutes in a stenocardiac manner. Acute myocarditis should always be considered for the purpose of acute coronary syndrome differentiating diagnosis for patients with chest pain complaints and high levels of cardiac enzymes in particular.
Keywords: Myocarditis, emergency, troponin

INTRODUCTION

Myocarditis is a myocardium disease where myocytes are damaged as a result of inflammatory infiltration [1]. In general, it progresses asymptomatically; therefore it is hard to determine its incidence. Postmortem studies are types of study used the most for such purpose [2]. During such studies, it has been found out that myocarditis is one of prominent reasons (20%) for sudden unexpected deaths in adults younger than 40 years of age. Clinical presentation of the disease ranges from non-specific symptoms (fever, myalgy, palpitation or exercise dyspnea) to severe hemodynamic collapse and sudden death. Its clinical manifestations include asymptomatic ECG anomalies to cardiogenic shock [3].

It has been reported that myocarditis mimics acute coronary syndrome. If young patients with acute coronary syndrome symptoms and findings lack coronary risk factors, and ECG anomalies spread beyond the single coronary artery zone or echocardiography indicates global dysfunction findings rather than segmenter, acute myocarditis should be considered. Echocardiographic findings could be variant and relatively non-specific. Serial studies have shown that echocardiography is beneficial in evaluation of treatment response in various forms of myocarditis [4].

Frequent evidence includes seeing segmenter or global wall movement disorder on echocardiography of those patients with coronary arteries that are angiographically normal. No standard guide has been created for diagnosis and treatment of myocarditis to date although it is known to have high levels of morbidity and mortality. The major reason of this is the fact that etiologies giving rise to myocarditis is variant while clinical presentations are heterogeneous [4].

CASE PRESENTATION

It was found out that chest pain accompanied by fear of death and feeling of sickness of a 49-year-old female patient applying at the emergency room with chest pain complaint was continuing uninterruptedly for the last 30 minutes in a stenocardiac manner. Having learnt from the patient's history that she was previously healthy, it was found out that the patient was a non-smoker and was not on medication. The family history showed that her father had ischemic cardiac disease. The patient was conscious, oriented and cooperative. It was determined that TA was 140/70 mmHg, pulse 90/min, fever 37 ^{0}C, and saturation 95%. Her systemic physical examination was found normal. During cardiac examination, cardiac murmur or rubbing was not heard. For chest pain differential diagnosis, the patient had hemogram, biochemistry, blood gas tests done and underwent an ECG (Fig-1).

Fig-1: ECG

Her test results were as follows: WBC: 6.86 K / uL (4-11 K / uL), Hemoglobin: 12.5 g / dL (13-17.5 g / dL), Platelets: 208 K / uL (150 – 400 K / uL), Aspartate transaminase (AST): 29 U/L (15 – 32 U/L), Alanine Transaminase (ALT): 29 U/L (10 – 33 U/L), LDH: 201 U/L (135-214 U/L), Troponin: 3,87 ng/mL.(<0.12 ng/mL) CKMB:1.19 ng/mL (0-4.88 ng/mL), PH:7.44, PCO2:36.2, PO2:88.5, SO2:97.1, CRP: 20.97 mg/L, and ECG conformed to normal sinus rhythm. At DII, DIII and aVF derivations, ST rise above 1 mm was detected and T negativity was determined at V1-3, she had no reciprocal (Figure 1). PA lung radiography was considered normal. At her echocardiography, there was no regional wall movement disorder, and no pericardial effusion was detected. Upon observing EF60%, light dilation on right heart and PAB: 30-35 mmHg, the patient underwent thorax tomography with contrast for pulmonary embolism exclusion, and radiology provided a comment showing no pulmonary embolism in the thorax tomography. Cardiac enzymes studied six hours later were troponin: 3.35 ng/mL (<0.12 ng/mL), CKMB: 0.96 ng/mL (0-4.88 ng/mL). Due to lack of reciprocal on the patient's ECG, no wall movement defect being detected on ECHO, no progress on cardiac panel, cardiology was consulted with preliminary diagnoses such as MI-myocarditis and the patient was admitted to coronary intense care unit. Since they were not able to exclude MI, treatment with Clopidogrel 300mg, Enoxaparin sodium 0.6 SC, Metoprolol 50 mg, Acetylsalyclic acid 300 mg, ibuprofen 400 mg was initiated and a coronary angiography was planned. Coronary arteries were considered normal on angiography. Myocarditis treatment was continued since no coronary artery anomaly and coronary slow flow were detected. The patient was discharged with full recovery after cardiac enzymes became negative.

DISCUSSION

For patients applying with chest pain, a diagnosis can be made with physical examination, ECG, cardiac enzymes and radiological examinations. As is the case with this patient, we may have a hard time in making a diagnosis since acute myocarditis, MI and prizmetal angina have similar clinical, ECG findings and laboratory findings [5, 6].

Myocardial infarction is characterized by a constant pain felt as a pressure on the chest which can spread to the chin and the arm. If the sore vein is re-canalized, such pain goes away. The pain with prizmetal angina is similar to MI pain, yet is intermittent and lasts for a shorter period. ECG is normal when there is no pain. In presence of acute myocarditis, chest pain is severe and pleuritic, and linked with movement. Patients may not always apply at a hospital with such typical symptoms [6, 7, 8, 9].

The chest paint of our patient was a type of pain in the manner of pressure, accompanied by fear of death, feeling of sickness and cold sweat. The patient's pain did not spread. The pain had regressed during follow-ups on the patient. The family history showed that her father had ischemic cardiac disease. Presence of a family history, the pain being in the pressure form, fear of death and cold sweat led us to consider MI.

At chest derivations showing relative vein with acute MI, ≥1mm convex ST elevation and reciprocal ST depression ECG finding can be observed. In case of prizmetal angina, ECG findings are similar to MI ECG findings. When it comes to pericarditis ECG finding, no ST depression is observed apart from widespread concave ST elevation and V1-aVR. ECG findings at myocarditis are more localized in comparison to pericarditis and there is more convex ST elevation, and ST depression is rare. At 12 lead ECG

the patient had done, ST rise above 1 mm was detected at DII, DIII and aVF derivations, and T negativity was determined at V1-3.

Increased level of troponin is a more reliable marker of myocardial damage than kreatine kinase [10]. Troponin may be measured 4 to 6 hours after onset of the chest pain. It is not beneficial for patients applying during the early stage. Troponin was positive with our patient, yet CK-MB was not significantly high. Coronary arteries were considered normal on angiography. No coronary artery anomaly and coronary slow flow were detected. Upon such detection, myocarditis diagnosis was made.

In case of myocarditis, cardiac symptoms develop in connection with systolic or diastolic dysfunction, brady or tacyarrhytmia. Patients' histories may include a history of pyretic disease such as cold they suffered from days or weeks ago. 60% of patients have a history of previous viral infection. The history may include fever, malaise and fatigue, muscle and joint pain, and skin rash. Feeling of pain on the chest is existent with 35% of patients [11].

Persons with obvious cardiac insufficiency may demonstrate dyspnea, tiredness, malaise, palpitation and exercise intolerance. Patients may come with sudden unexpected deaths, arrythmia or thromboembolic cases [12]. The patient did not have a history of previous infection, yet she had a chest pain and fear of death.

Those patients who are doubted to have a myocarditis should be hospitalized and they should be monitored in terms of cardiac insufficiency symptoms, arrythmia, conduction defects and embolism. The patient should be given bed rest until recovery. Antipyretic drugs except for NSAID agents may be administered to the patient. Analgezic may be added to the treatment for pleuretic or pericardial pain. Oxygen should be given to patients since hypoxia cardiac reduces the flow rate [13].

While thrombolytic and anticoagulant treatment is administered for acute coronary syndrome, application of thrombolytic treatment to cases with myocarditis may give rise to cardiovascular complications. Therefore, it is important to differentiate these with regards to treatment and follow-up plan. In case of AMI doubt, universal reason for full recovery of cardiovascular conditions in previously healthy adults is myocarditis [4].

To conclude, acute myocarditis should always be considered for the purpose of acute coronary syndrome differentiating diagnosis for patients with chest pain complaints and high levels of cardiac enzymes in particular.

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