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The Dilemma of acute myocarditis and acute myocardial infarction: A case report

Akut miyokardit ve akut miyokart infarktüsü ikilemi: Vaka sunumu

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Özet

Göğüs ağrısı ve St segment elevasyonu ile gelen hastalarda düşünülecek ilk tanı akut miyokart infarktüsüdür. Ancak akut miyokart infarktüsü bazen miyokardit gibi bazı hastalıklarla karışabilir. Akut miyokart infarktüsü ve miyokardit ayrımı hikaye, fizik muayene, elektrokardiyografi, ekokardiyografi ve kardiyak-spesifik enzimlerle yapılır.Bu iki hastalığın tedavi ve prognozu birbirinden tamamen farklıdır. Ayırıcı tanıya varmak ve koroner anjiografi kararı verebilmek hayat kurtarıcıdır. Bu iki hastalığın ayırımında elektrokardiyografi çok önemlidir. Elektrokardiyografi bulguları farklı olmasına rağmen, bazen akut miyokart infarktüsü ve akut miyokardit birbirini taklit edebilir. Fokal ST elevasyonu ile birlikte resiprokal ST depresyonu bulunan 22 yaşında miyokardit tanısı konulan bir hastayı sunduk.

Anahtar Kelimeler: Miyokardit, miyokart infarktüsü, prinzmental Angina, koroner anjiografi.

Abstract

The first diagnosis in patients with chest pain and ST segment elevation is acute myocardial infarction. But acute myocardial infarction sometimes can be confused with other diseases such as myocarditis. Discrimination of acute myocardial infarction and acute myocarditis can be made by history, physical examination, electrocardiography, echocardiography and cardiac-specific enzymes. The treatment and prognosis of those two diseases are completely different. The differential diagnosis and decision of coronary angiography is lifesaving. Electrocardiography is very important in the differentiation of these two diseases. Although ECG findings are different, acute myocarditis and acute myocardial infarction sometimes imitate each other. 22 years old male patient diagnosed as myocarditis and focal ST elevation with reciprocal ST depression presented in this case.

Keywords: Myocarditis, myocardial infarction, prinzmental angina, coronary angiography.

Introduction

Chest pain is one of the most common causes admitting to the emergency department (1). Although there are not any life-threatening conditions in these patients, the physicians must distinguish the patients with acute coronary syndromes which requiring emergency intervention from non-severe patients. First evaluation of patients with chest pain is identifying the chest pain characteristics with electrocardiographic (ECG) findings and cardiac-specific enzymes and then the patient usually can be diagnosed. But in some cases it is very difficult (2). Although the first diagnosis that comes to mind in a patient with chest pain and ST segment elevation is myocardial infarction, also other diagnoses such as pericarditis, myocarditis, prinzmental angina should be considered. ECG is important especially in giving a decision of emergency coronary angiography (3). We present a 22-year-old patient who admitted with chest pain and diagnosed with myocarditis by coronary angiography.

Case Presentation

A 22-year-old male patient was admitted to the emergency department with sudden chest pain onset 2 hours ago. He was a nonsmoker and had no hypertension, diabetes, dyslipidemia and cocaine usage in his history. The patient had been severe influenza infections two weeks ago, he had used paracetamol 500mgx2 and chlorpheniramine 4mg x2 treatment for 1 week and the complaints has declined. On physical examination, arterial blood pressure was 110/70 mmHg, pulse 100/min, fever 37.4 ° C, respectively. On cardiac auscultation S1 and S2 were normal, additional sound, pathologic murmur and pericardial friction sound were not observed. Minimal PR depression and concave ST elevation of 1 mm in first derivation, also 1 mm concave ST elevation in lead aVL was observed in 12-lead electrocardiography. In third derivation and in aVF 1 mm evident ST segment depression was observed (Figure 1). On admission troponin and d- dimer values were negative. The other laboratory findings were normal except the

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leukocyte 10600 x10 ^ 3/uL and c reactive protein 2.3mg/dL. Regional wall motion abnormalities was not observed on transthoracic echocardiography.

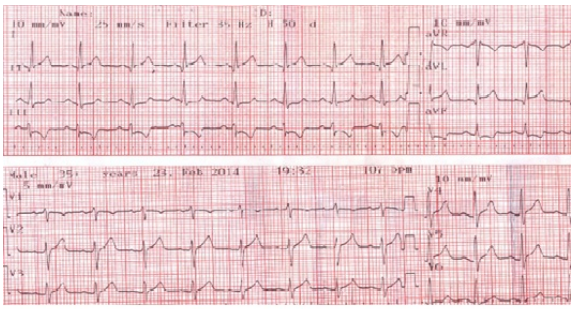

Figure 1- Initial 12 lead ECG. ST elevation ve PR depresion in lead I and aVL, ST depresion in leads III and aVF.

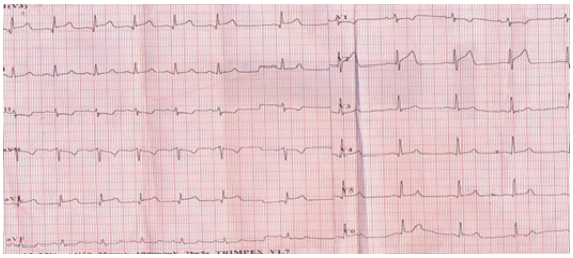

Figure 2- ECG at 15 hour. ST elevation in leads I and aVL. ST depresions in leads III ve aVF, ST elevation just started in leads V5-V6.

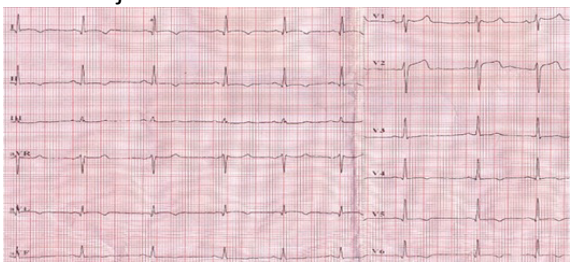

Figure 3- ECG during discharge (5th day).

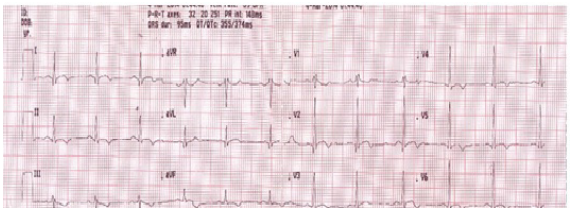

Figure 4- ECG after 1 week from discharge.

The patient was hospitalized in the coronary care unit because of severe chest pain and ST segment changes in the electrocardiography. Chest pain on precordium was pressured and spread to the patient's lower jaw, severity of pain was decreasing by leaning forward or changing the position. First of all intramuscular diclofenac(İM) sodium 75 mg / im was injected and acetylsalicylic acid 300 mg, metorolol intravenously(İV) 25 mg, nitroglycerin 5 micrograms / min was added as a precaution for the possible acute lateral myocardial infarction or vasospastic angina, the pain was relieved, 4 hours later ECG and troponin tests were repeated. Concave ST elevations and reciprocal depression has continued and value of troponin was detected 3.2 ng / ml. ECG was repeated at frequent intervals, but the dynamic changes in ECG were not observed. After 10 hours of his admission, chest pain of the patient has intensified again, as a precaution to possible myocarditis or myocardial infarction diclofenac sodium 75mg/IM applied again and nitroglycerin dose increased 10 micrograms / min, then 20 micrograms/ min. Chest pain did not change significantly. After 12 hours of admission, troponin was detected 10.6 ng / ml. 800 mg of oral ibuprofen and prednisolon 40 mg / IV was applied. There was no response to nitrate, analgesic and anti-inflammatory agents. With tramadol 50 mg / IM chest pain decreased. After 15 hours of admission, in ECG, ST elevation in leads of V5-V6 viewed which had not been in the first ECG (Figure 2). For this reason coronary angiography was performed on the patient. Congenital anomalies, thrombosis or vasospasm was not observed in the coronary arteries. It was evaluated as normal. Treatment of Ibuprofen 800 mg x 3 and during the times when the pain was severe, tramadol 50 mg / IM was continued, at 2nd, 3rd, 4th days chest pain has relieved day by day, at 5th day the pain greatly reduced, ST depression in 3 and aVF in ECG and ST elevations in 1 and aVL came isoelectric line (Figure 3). The patient was discharged with the treatment of oral ibuprofen 800mgx3 and after one week, in the ECG (Figure 4) it was observed that ST elevation in I and aVL derivations, also ST depression in II and aVF derivations have decreased, common t negativity viewed.

Discussion

Initial assessment of patients admitted with chest pain made with physical examination, electrocardiography, cardiac specific enzymes and often can be diagnosed. But in somecases it is difficult to diagnose (2,3).

In acute myocardial infarction, on precordium, a suppressing, overwhelming and tightening pain occurs that can spread to the jaw and left arm and that pain is more severe and continuous. Pain disappears with revascularization of related vessel or necrositation of myocardial area. In prinzmental (vasospastic) angina, the character of the pain is similar to myocardial infarction but the pain is intermittent and usually takes less time. ECG is in normal appearance when there was not pain. In acute myocarditis, chest pain is sharp and pleuritic, it is related with the movement. However, patients are not always applying to the hospital with typical symptoms such that (3,4,5,6). In our patient chest pain had started suddenly. It was such a suppressing pain and spreading to his lower jaw (these symptoms were suggestive of an ischemic pain) and decreasing by leaning forward (that is in favor of myocarditis). Spreading and the character of pain suggested acute myocardial infarction in the first, but the patient's age, history and risk factors were incompatible with this diagnosis. Periodically increases of the patient's pain was suggestive vasopastic angina. Because of chest pain being subjective and variable and determination of cardiac enzymes in the blood takes time, 12-lead electrocardiogram during the application is very important (2,3). In acute myocardial infarction, ST segment elevation in successive leads belongs to related vessel is observed by ≥ 1 mm convex ST elevation, there is a reciprocal ST depression. In vasospastic angina it is encountered with the same findings as myocardial infarction (1-6). According to our classic information it is usually seen prevalent concave ST segment elevation in acute pericarditis. ST depression is not observed except V1 and aVR, in acute myocarditis ST elevation is seen more convex and more localized than pericarditis because of myocardial involvement. In acute pericarditis ST depression is never observed except V1 and aVR, in acute myopericarditis ST depression is rarely observed (6). In our patient, ST elevation is in I, aVL V5, V6 leads (lateral derivations) and ST depression (reciprocal change) is in III and aVF, so this was in favor of acute myocardial infarction. However, these ST elevations were concave and there was a PR depression in the first derivation that was in favor of acute myopericarditis. In the literature it has been reported that in very rare cases ST elevation and reciprocal ST depression is observed together in myopericarditis.

Troponin is the most specific and most commonly used marker that indicating myocardial damage. It is measurable in the blood approximately 4-6 hours later from inception of chest pain. For this reason, it is not benefit in patients who admitted in the early period (8). Chest pain of our patient had started two hours ago and troponin values were determined negative during the application and then. at 4th and 12nd hours measured again and sequentially 2.3 and 10.6 ng / dl were determined. We suggested that a serious damage at the myocardium because of these high values and gradually increasing pain, but it did not help to distinguish these two diseases.

Acute myopericarditis, acute MI and prinzmental angina imitate to each other because of similar clinical, electrocardiographic and laboratory findings and it is difficult to diagnose. In most of these group of patients there are not life-threatening situations, physicians should separate those patients with acute coronary syndrome that is requiring emergency intervention from non-severe patients. In the cases of difficult discrimination, coronary angiography can be performed to exclude of acute coronary syndrome (2). It could not be diagnosed to our patient differential diagnosis of acute MI, acute myopericarditis and prinzmental angina with clinical and non-invasive methods, but after coronary angiography myopericarditis were diagnosed as a result of the coronary arteries are normal. In the period till diagnosis what kind of treatment protocol will be applied is open to debate.

When myocardial infarction diagnosed it should be given acetylsalicylic acid by 300 mg. at the earliest time up to revascularization, if there is not a contraindication it should be continued with beta-blockers with repeated doses first IV then oral. In order to reduce the severity of chest pain and coronary vasodilatation, nitroglycerin should be applied.

If chest pain continues sedatives can be applied. In vasospastic angina, calcium channel blockers and perlinganit should be given. In acute myopericarditis, nonsteriod anti-inflammatory drugs use as initial therapy, especially ibuprofen is preferred because of the wide range of doses and safety (10). Because of corticosteroids can cause to recurrent pericarditis attacks; it is recommended to use in a controlled manner, it is added to treatment if there is no response to NSAIDs and colchicine (11). Because of his age, history, risk factors, concave ST elevation and PR depression NSAII was performed to our patient principally by intramuscularly. 300 mg acetyl salicylic acid, IV metoprolol and nitroglycerin were also given to the patient but we couldn't get a possitive response. Because of recurrence and increase of chest pain tramadol by 50 mg IM applied and the pain is passed. After effect of tramadol the patient's chest pain started again, where upon, with the suspicion of acute myocardial infarction coronary angiography has been applied. Because of determining normal coronary arteries on coronary angiography, NSAII therapy was continued and the patient's pain gradually decreased.

Considering his age, risk factors and history, the first diagnosis that came to mind was myopericarditis but absence of reciprocal ST-segment depression in ECG and being a compressive pain in central part of the chest and not responding to non-steroidal anti-inflammatory drugs; acute coronary syndrome was suggested. The most important feature of our case was observation of such a severe pain and also absence of unexpected reciprocal ST depression in inferior (III and AVF) leads which seen only in few cases in the literature. This can due to widespread and severe inflammation in the lateral wall of the left ventricle compared to other regions. So electrocardiography act as an acute lateral myocardial infarction and reciprocal ST depression was seen.

Patients with chest pain are not always admitted with typical complaints. Patients with myopericarditis with atypical presentation can be confused with myocardial infarction. Unlike our traditional knowledge, reciprocal ST depression also may be observed in myopericarditis, physicians should have knowledge on this subject. There is not enough information in the literature on this subject. It should be made a wider research for the separation of these two entity which treatment and prognosis completely different to each other

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