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Case report

Acute coronary syndrome or acute aortic syndrome - correct diagnosis prevents fatal complication

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Abstract A 78-year-old man was admitted to local hospital due to chest pain. Electrocardiogram was performed three times at intervals of one hour, in which the progression of ST elevation on the anterior wall was clearly visible. However, aortic wall dissection was suspected on echocardiography, and CT without contrast also described aortic dissection. Patient was referred to cardiac surgery, but dissection was not confirmed intraoperatively. After two days, the patient developed severe chest pain with severe heart failure and cardiogenic shock. Cardiac echocardiography was performed, and it showed severe ischemic cardiomyopathy with reduced left ventricle ejection fraction (EF 19%). Coronary angiography was performed and it verified three-vessel coronary artery disease but interventional cardiologists estimated that the risk of percutaneous coronary intervention was too high at that time. All recommended therapy was applied and the patient's condition was improved, the symptoms and signs of heart failure were disappeared. The patient was discharged after 10 days in stable condition, compensated with the following therapy: warfarin, clopidogrel 75 mg, bisoprolol 2.5 mg, valsartan/sacubitril 26 mg / 24 mg two times per a day, amiodarone 200 mg, furosemide 2x40 mg, eplerenone 25 mg, empagliflozin 10 mg, rosuvastatin 20 mg, pantoprazole 40 mg. Cardiologists and cardiac surgeons decided to try pecutaneous intervention on two arteries Cx and RCA in the next step.

Key words acute coronary syndrome, aortic dissection

Introduction

Aortic dissection is one of the most urgent and dramatic conditions in clinical medicine with a very high early mortality rate of 1-2% per hour in untreated patients in the first 24 to 48 hours. The most common symptom of aortic dissection, that occurs in over 96% of cases, is chest pain. The chest pain in aortic dissection begins suddenly and the intensity is strongest at the beginning ("decresendo" type of pain), which distinguishes it from pain in acute coronary syndrome, which is usually of a "crescendo" character. About two thirds of patients with ascending aortic dissection have aortic insufficiency, so early diastolic murmur can be heard auscultatory, and also in the physical finding there is a difference in pressure between on the left and right arm, and pulse deficit, ie. weakened or absent arterial pulses and very often neurological manifestations[1,2].

Acute coronary syndrome (ACS) is significantly more common than aortic dissection. It is the most severe form of ischemic heart disease and it is one of the most common causes of urgent hospital admission and sudden cardiac death. In addition to the characteristic chest pain, there are also ischemic changes in the electrocardiography (ECG) and an increase level of cardiac enzymes. Aortic dissection and acute coronary syndrome may have a similar clinical presentation, and careful history, electrocardiogram, and cardiac enzymes are necessary to distinguish these two very serious conditions.

Here we will present the case of a patient in whom the correct diagnosis of acute myocardial infarction was not made, which led to a wrong referral to cardiac surgery and development of severe ischemic cardiomyopathy with severe heart failure and cardiogenic shock.

Case presentation

A 78-year-old man was admitted to local hospital due to chest pain. Electrocardiogram was performed three times at intervals of one hour, in which the progression of ST elevation on the anterior wall was clearly visible. Troponin I (Tn I) was 25.1 pg / mL, CK MB 2.20 ng / mL. Echocardiographic examination showed that the ejection fraction of the left ventricle (EF) was reduced by 44%, the distal two thirds of left ventricle were hypokinetic, as well as the apex of the heart (Figure 1). Also, aortic wall dissection was suspected, and the patient was referred for a CT scan of the chest in a local hospital. CT was performed, but without contrast, and it described that the aortic root was dilated to 48 mm with signs of dissection on a short segment above the aortic valve. Due to this finding, the patient was urgently referred to the Univer-

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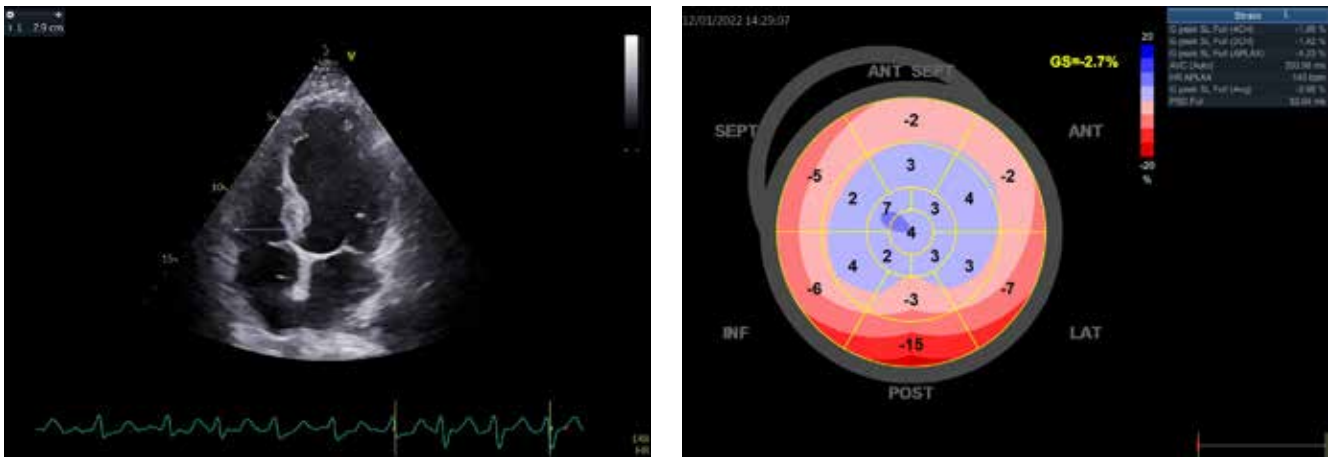

Figure 1a and b. Echocardiography findings with wall motion abnormalities of the anterior, antero-septal wall and the apex

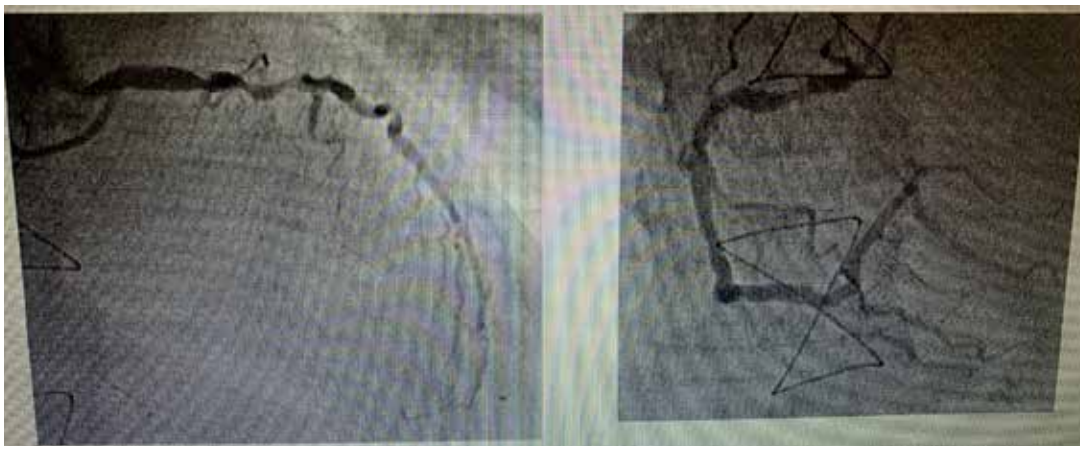

Figure 2. Selective coronarography presenting occluded ostial left main, with significant stenosis in LM, Cx and RCA

sity Clinical Center of Serbia, to the Cardiac Surgery Clinic for urgent surgical treatment. On admission to our clinic, the patient was stabile, with chest pain, blood pressure 135/85 mmHg on both arms, heart rate was 90/min, auscultatory without heart murmurs, all peripheral pulses were palpable. The patient was introduced to the operating room and under general anesthesia, a medial sternotomy was performed. An arterial cannulation of the ascending aorta was performed, cannulation of the right atrium too, and extracorporeal blood flow was started (ECC). The exploration did not show a rupture of the aortal intima, nor a rupture of the aorta. Laboratory analyzes that was taken immediately upon admission to the Clinic of Cardiac Surgery, which arrived subsequently, showed elevated markers of myocardial necrosis (troponin T 3267 ng/L, creatine kinase (CK) 1775 U/L). Intraoperatively, surgeons saw that the coronary arteries were extremely calcified and not suitable for grafting. Next day, selective coronary angiography was performed and it verified three-vessel coronary artery disease (Figure 2) with stenosis in the distal part of the left main stenosis, ostial left anterior descending (LAD) occlusion, 90% proximal circumflex artery (Cx) stenosis, and 80% stenosis in the right coronary artery (RCA). In the first postoperative day, there was a further increase in troponin T (Tn T 6959 ng/L), and the next two days there was a gradual decrease (3595 ng/L, then 2391 ng/L). After two days, the patient developed severe chest pain, ECG showed atrial fibrillation, heart rate 150/min, ST elevation in V1-V3 leads, Q wave in V2-V3, ST depression in D2, D3, aVf leads (Figure 3). Clinically, severe heart failure and cardiac shock was developed. Intravenous antiarrhythmic therapy (amiodarone) was prescribed, and electroconversion was attempted but without success. Inotropic support was prescribed. Due to the cessation of diuresis, continuous intravenous diuretic therapy was prescribed, too. Laboratory analyzes showed a new elevation in markers of myocardial necrosis (troponin T 4094 ng/L), an elevation in transaminases (AST 190 U/L, ALT 134 U/L). Cardiac echocardiography was performed, and it showed severe ischemic cardiomyopathy with a large part of akinesia that involved the entire septum, medio-apical segment of the anterior, posterior and lateral wall, better contractility was only in basal segments of the postero-lateral wall (Figure 4). Ejection fraction was severe reduced (EF 19%). Longitudinal LV function was also severe decreased (AFI: EDV 106 ml, ESV 85 ml, EF 19%, GLS -2.7%, PSD -52), signs of diastolic dysfunction was presented, too and also significant increased in filling pressure of LV (DCT -92, E'lat- 7.0, E's-5.0, E/e'avg-18.0), moderate mitral regurgitation, right ventricle was with normal size, decreased systolic function (TAPSE 1.1 cm), severe tricuspid regurgitation 2-3 +, pressure in the right heart was 50 mmHg. In the further

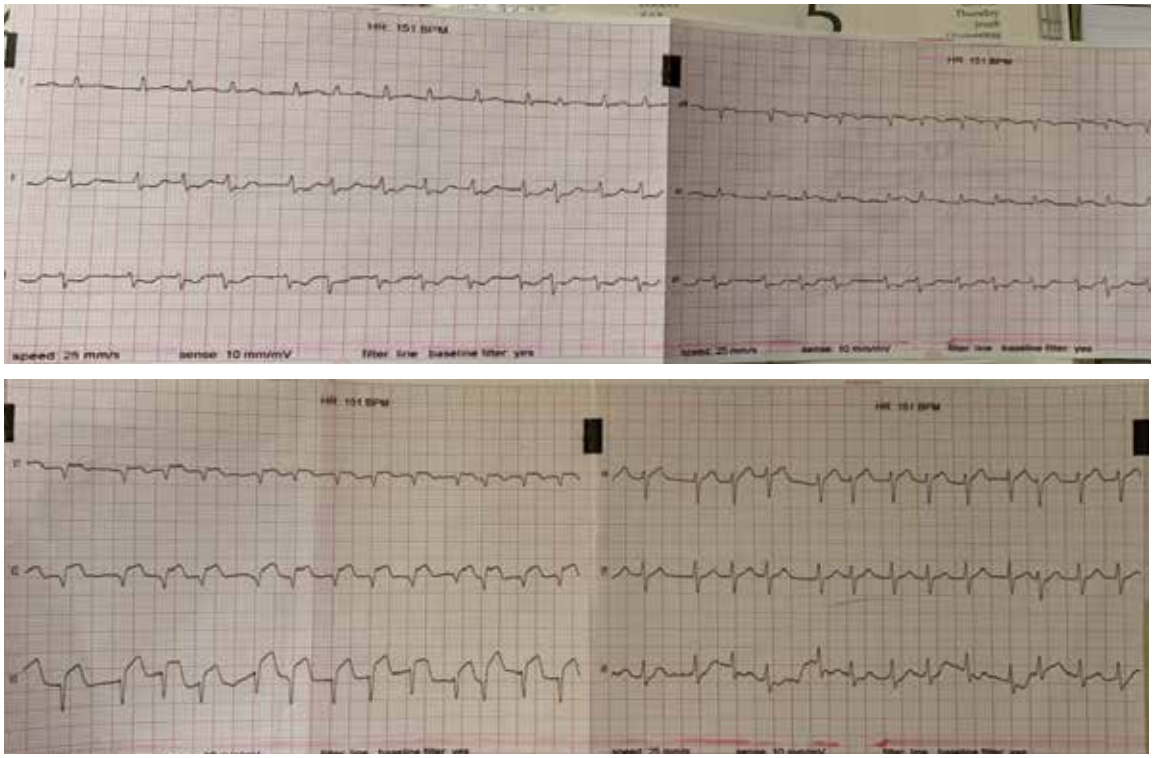

Figure 3. Electrocardiogram-atrial fibrillation, Q wave in V2-V3 leads with ST elevation, ST depression in D2, D3, aVf leads

course, the patient's condition was improved, the symptoms and signs of heart failure were disappeared. The sinus rhythm was established. ECG showed a scar in V2-V4 leads with persistent ST elevation. The patient was discharged after 10 days in stable condition, compensated with the following therapy: warfarin, clopidogrel 75 mg, bisoprolol 2.5 mg, valsartan/ sacubitril 26 mg / 24 mg two times per a day, amiodarone 200 mg, furosemide 2x40 mg, eplerenone 25 mg, empagliflozin 10 mg, rosuvastatin 20 mg, pantoprazole 40 mg. Cardiologists and cardiac surgeons decided to try pecutaneous intervention on two arteries Cx and RCA in the next step.

Discussion

Here we present a typical patient that is seeing every day in cardiac ambulances. The patient came with chest pain, first ECG showed unconvincing elevation of the ST segment, troponin was taken but troponin I, which was not elevated, then an echocardiography of the heart where performed ant it showed disorder in segmental kinetics but aortic wall stratification was suspected. However, the next two ECGs, showed a clear progression of elevation, but due to the suspicion of dissection, a chest CT scan was performed and it showed misdiagnosed of ascending aortic dissection and the patient was referred to wrong direction.

In all patients with chest pain electrocardiogram (ECG) should be performed first. If acute coronary syndrome is ruled out, aortic dissection should be considered. In the IRAD study, ECG was normal in 31% of patients, nonspecific ST and T changes were found in 42% of patients, ischemic changes in 15% of patients, and signs of acute myocardial infarction in 5% of patients with ascending

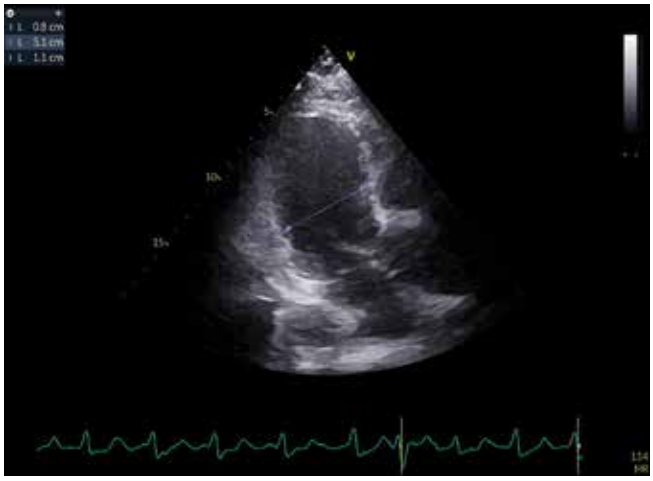

Figure 4. Echocardiography findings showing deterioration of LV function and ejection fraction

aortic dissection[3]. Aortic dissection can be extended to the branches of the aorta, as well as to the coronary arteries, mainly the right coronary artery, so it can give a picture of inferior myocardial infarction with ST-segment elevation in the inferior leads in the ECG.

Aortic dissection can mimic many diseases, so early recognition of this entity is crucial in order to reduce early mortality. Analyzing 26 clinical variables at initial presentation of aortic dissection, Von Kodolish singled out the following variables as independent predictors for the diagnosis of acute aortic dissection: (1) pain (sudden onset, tearing, tearing), (2) dilated aorta or mediastinum at radiography (3) difference in pulse or arterial pressure. If all three predictors are present, the probability of aortic dissection is 96%. The probability is lower if there are one or two predictors and is 35%, and only 7% if none of these predictors exist[4].

The choice of diagnostic methods depend on the patient's condition, the availability of diagnostic techniques and experience in interpreting the results. Diagnostic techniques should primarily confirm the existence of aortic dissection and the type of dissection. Aortography has long been the gold standard for the diagnosis of dissection. Its sensitivity is 86-88% and specificity 75-94%. However, with the development of non-invasive methods, primarily contrast computed tomography, aortography as an invasive method has been suppressed[5]. Transthoracic echocardiographic examination is very useful, with sensitivity for proximal aortic dissection of 78-100%, but for distal is lower 31-55%[6,7,8]). The most significant sign is intimate flap (floating intimate membrane) that separates the true from the false lumen. Using the color Doppler technique, the flow through the false lumen can be seen. However, today, computed tomography (CT) with the use of intravenous contrast is used for the diagnosis and confirmation of dissection, which is a very sensitive and specific method for the diagnosis of aortic dissection. Multidetector CT (MDCT) with the possibility of three-dimensional image reconstruction further improves the diagnostic accuracy of this method. The sensitivity and specificity of CT for the diagnosis of aortic dissection is 96-100%[9,10]. What is important to point out is that CT must be done with contrast in order to avoid situations like the one that happened to our patient.

If an acute myocardial infarction is not diagnosed on time a number of complications resulting from myocardial necrosis can be developed. The most common complication of infarction, especially anterior wall infarction, is heart failure. Our patient developed severe ischemic cardiomyopathy with a significantly reduced left ventricular ejection fraction, which was resulted in the development of acute heart failure. Cardiac shock is one of the manifestations of acute heart failure[11]. Treatment of cardiac shock should be started as early as possible. Early identification and treatment of the underlying causes, along with hemodynamic stabilization and treatment of organ dysfunction, are essential components of treatment[11]. In the case of our patient, hemodynamic stabilization was performed, therapy was prescribed according to recommendations, but treatment of the underlying cause, ie. treatment of coronary heart disease was not possible, because interventional cardiologists estimated that the risk of percutaneous coronary intervention was too high at that time. Therefore, it was decided to stabilize the patient with medication. However, severe ischemic cardiomyopathy and the potential new decompensation and the development of malignant heart rhythm disorders, remain a problem. Therefore, in accordance with the latest recommendations, the patient received therapy (valsartan/sacubitril, empagliflozin, eplerenone and bisoprolol), which has been shown to reduce mortality and prolong survival in heart failure. Of course, loop diuretics, which prevent congestion, are important parts of therapy. It remains to be seen how the situation with our patient will continue.

Conclusion

Acute coronary syndrome is much more common than aortic dissection, so in patients with chest pain, ACS should be confirmed or ruled out first. Although CT is a more sophisticated method than electrocardiograms, if it is not done adequately, or like in this case without contrast, or if it is read by someone with insufficient experience in recognizing of aortic dissection, misdiagnosis and misdirection and treatment of patients can occur. A new approach to the treatment of patients with heart failure, provides us new opportunities in the treatment of these patients, with prolonging their lives, reducing mortality and improving quality of life.

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Sažetak

Akutni koronarni sindrom ili akutni aortni sindrom – pravilna dijagnoza sprečava fatalne komplikacije

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Muškarac star 78 godina primljen je u regionalnu bolnicu zbog bolova u grudima. Urađeni su elektrokardiogrami u tri navrata u razmaku od po sat vremena, u kojima se jasno vidi progresija ST elevacije na prednjem zidu, ali je ehokardiografski (EHO) postavljena sumnja na raslojavanje zida aorte, te je urađena CT grudnog koša, ali bez kontrasta, gde je opisana disekcija ushodne aorte. Pacijent je htno upućen na kardiohirurgiju, ali disekcija nije potvrđena intraoperativno. Posle dva dana, pacijent je dobio jak bol u grudima uz razvoj teške srčane insuficijencije i kardiogenog šoka. Urađen je EHO srca koji je pokazao tešku ishemijsku kardiomiopatiju sa smanjenom ejekcionom frakcijom leve komore (EF 19%). Urađena je koronarna angiografija i verifikovana trosudovna koronarna bolest, ali su interventni kardiolozi procenili da je rizik od perkutane koronarne intervencije tada bio previsok. Primenjena je sva terapija prema preporukama i stanje pacijenta se poboljšano, simptomi i znaci srčane insuficijencije su nestali. Pacijent je otpušten posle 10 dana u stabilnom stanju, sa sledećom terapijom: varfarin, klopidogrel 75 mg, bisoprolol 2,5 mg, valsartan/akubitril 26 mg/24 mg dva puta dnevno, amiodaron 200 mg, furosemid 2k40 mg, 25 mg, empagliflozin 10 mg, rosuvastatin 20 mg, pantoprazol 40 mg. Kardiohirurški konzilijum je odlučio da se naknadno pokuša PCI preostale dve arterije C xi RCA.

Ključne reči: akutni koronarni sindrom, disekcija aorte