



Double heart rupture after acute myocardial infarction: A case report

Dupla ruptura srca nakon akutnog infarkta miokarda

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Abstract

Introduction. Double heart rupture is a rare complication of acute myocardial infarction with high mortality. **Case report.** We presented a 67-year-old female patient with symptoms and signs of myocardial infarction, diagnosed with echocardiography, rupture of the septum, the presence of a thrombus and a small pericardial effusion. Soon after admission the patient died. Autopsy revealed tamponade and double myocardial rupture, free wall rupture and ventricular septal rupture, as a cause of death. **Conclusion.** This case highlights the need to evaluate patients with myocardial infarction, recurrent chest pain, echocardiographic signs of effusion and the presence of thrombus in the pericardium in terms of double rupture of the heart.

Key words:

myocardial infarction; acute disease; heart rupture, post-infarction; echocardiography.

Apstrakt

Uvod. Dupla ruptura srca retka je komplikacija akutnog infarkta miokarda sa visokom stopom smrtnosti. **Prikaz bolesnika.** U radu je prikazana 67-godišnja bolesnica sa simptomima i znacima akutnog infarkta miokarda, kojoj je ehokardografski postavljena dijagnoza rupture septuma i prisustvo tromba i manjeg perikardnog izliva. Ubrzo po prijemu bolesnica je umrla. Obdukcijom je ustanovljena tamponada i dupla ruptura srca, ruptura slobodnog zida leve komore i ventrikularnog septuma, što je bilo uzrok smrti. **Zaključak.** Prikaz ove bolesnice ukazuje na potrebu procene bolesnika sa infarktomiokarda, ponavljanim bolovima u zoni grudi, ehokardografskim znakovima izliva i prisustva tromba u perikardu u smislu dvostruke rupture srca.

Ključne reči:

infarkt miokarda; akutna bolest; srce, postinfarktna ruptura; ehokardiografija.

Introduction

There are three manifestations of heart rupture which include the left ventricle (LV): free wall rupture (FWR), ventricular septal rupture (VSR) and papillary muscle rupture (PMR). Double heart rupture (DHR) implies the combined presence of the two of the above forms. DHR is a very rare complication, present in about 0.3% of acute myocardial infarction, with the most common combination of FWR and VSR¹. In a relatively small study, it was revealed that FWR in half of the cases occurred after VSR¹. It was shown that primary percutaneous coronary interventions (pPCI) are protective factor for FWR compared to thrombolysis². Older age, female gender, and a prolonged time from the onset of symptoms to treatment are independent risk factors for rupture³.

Echocardiography is a reliable method for the diagnosis of mechanical complications in acute myocardial infarction

in terms of localization and size, but it is also crucial in the decision about treatment and postoperative follow-up⁴.

We presented a patient with late presentation of inferior myocardial infarction involving the right ventricle, complicated with a combination of FWR and VSR. Caution is obligatory in such patients because of their high mortality, and also the necessity of prompt diagnosis and treatment.

Case report

A 67-year-old female was admitted in the intensive care unit because of prolonged chest pain, shortness of breath, and electrocardiographic signs of acute myocardial infarction with ST-segment elevation in the inferior and right ventricle leads. Symptoms occurred three days before admission, with epigastric pain, nausea and vomit, which were initially diagnosed as gastritis. The next day the pain spread to the chest, and increased in intensity two hours before admission. The

patient was previously treated for angina pectoris, with risk factors for coronary disease of a positive family history and high blood pressure.

Objective examination on admission revealed somnolent state of the patient, diaphoretic skin, dyspnea, tachycardia, low blood pressure, bilateral crackles and strong precordial holosystolic audible murmur. The laboratory findings reported elevated levels of cardiac enzymes.

The 12-lead electrocardiogram (ECG) recorded sinus tachycardia with signs of myocardial infarction of inferior localization (Figure 1a). In the right precordial leads, ST segment elevation was seen in VR4 lead as a sign of right ventricle myocardial infarction (Figure 1b).

discontinuity in the ventricular septum was recorded, measuring up to 1.6 cm, with the occurrence of left-right shunt, which was verified by two-dimensional echocardiography and with color and continuous Doppler (Figure 2a). Due to the large dimensions of rupture, the gradient at the shunt was not high. The other walls of the left ventricle were moved hyperkinetically. No significant valvular heart disease was found.

Around the heart a large thrombus was observed, that has partially compromised the free wall of the right ventricle (Figure 2b). There was no sign of the free wall rupture in the lower part of the left ventricle.

The cardiothoracic surgeon was immediately consulted due to life treating condition, in the first place state of shock.

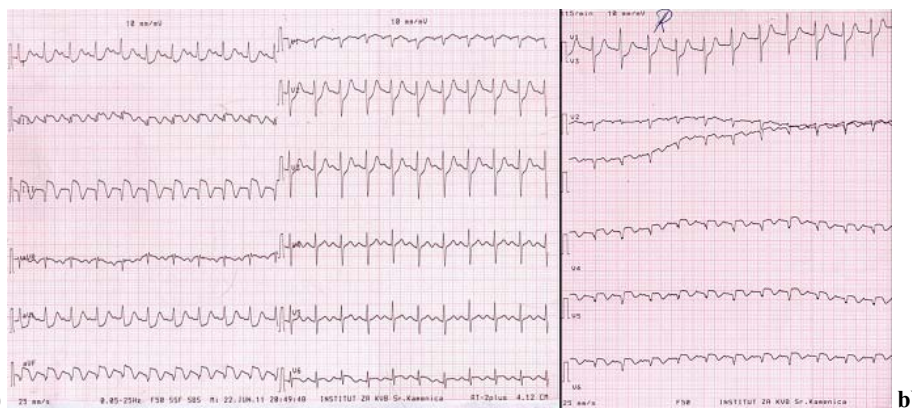


Fig. 1 – a) Electrocardiograph (ECG) registered sinus tachycardia, frequency of about 120/min, normogram, Q wave and ST elevation till +3.5 mm in D2, D3, aVF leads, ST segment denivelation of -2 mm in DI, aVL, V2 and V3 leads, without rhythm and conduction disorders; b) Right precordial leads with ST segment elevation in VR4 lead as a sign of right ventricle myocardial infarction.

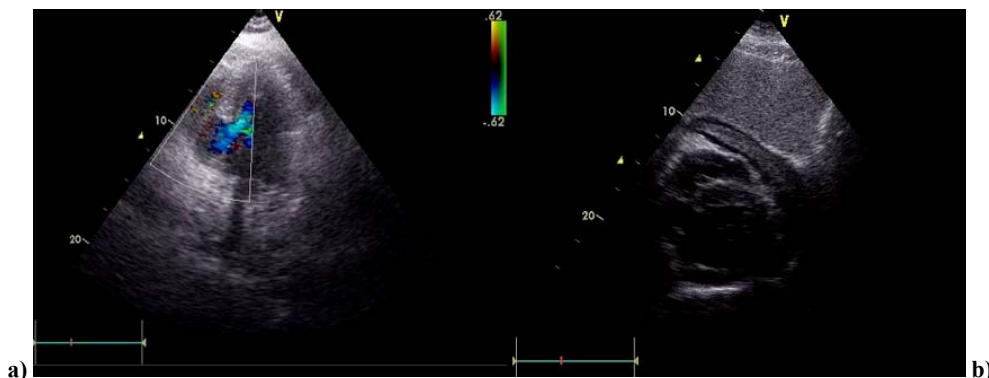


Fig. 2 – a) Doppler registered ventricular septum defect; b) Large thrombus in the pericardium.

Soon after admission due to acute left ventricular failure, hypoxemia was recorded in blood gases and the patient developed anuria and was intubated and mechanically ventilated. The patient was treated with double antiplatelet therapy, statins, and inotropes. Volume and acid-base status were also corrected according to blood gases. In order to adequately monitor the patient a central venous catheter was introduced (central venous pressure was 15 mmH₂O).

Immediately upon admission echocardiography was performed. There were normal left ventricular dimensions, with akinesia of all segments of the inferior and basal inferoseptal wall. In the region of the basal part of the inferoseptal wall

He indicated preparation of the patient for surgical intervention. During the entire hospitalization patient's condition was unstable and critical, and despite all the implemented measures cardiac arrest developed. Cardiopulmonary resuscitation did not restore spontaneous circulation and the patient died about 10 hours after admission.

Autopsy revealed the presence of 200 mL of blood and 250 mL of blood clot beneath the pericardium, which was smooth and shiny (Figure 3a).

The left ventricle was enlarged, with left ventricular thickening. In the upper third of the muscular part of the ventricular septum, a defect with torn edges, 2 cm in its larg-

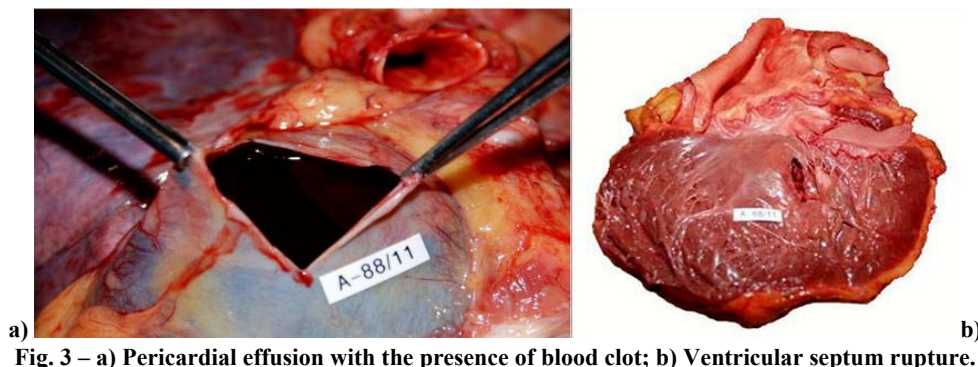


Fig. 3 – a) Pericardial effusion with the presence of blood clot; b) Ventricular septum rupture.

est diameter, was found (Figure 3b). At the posterior wall of the left ventricle, in the projection of the upper third of the ventricular septum, a small stellate defect, with blood permeated edges, 0.7 cm in its largest diameter that communicated with the left ventricular cavity was found (Figure 4).

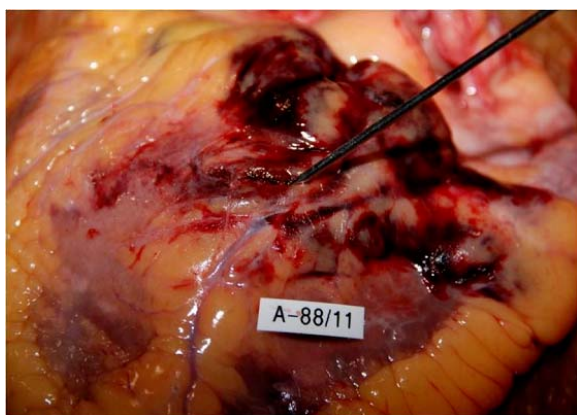


Fig. 4 – Left ventricle free wall rupture.

Histologically, in the cardiac muscle slices taken from the area of the septum and posterior wall of the left ventricle, the muscle fibers were torn, mainly without visible nucleoli. The cytoplasm of muscle cells was extremely acidophilic, homogeneous, without lines which are characteristic for muscle fibers. Interstitium was unevenly dilated, edematous and filled with neutrophils of which many were in close contact with the muscle fibers (Figure 5). Described areas corresponded to the coagulation type of necrosis, i.e. myocardial infarction.

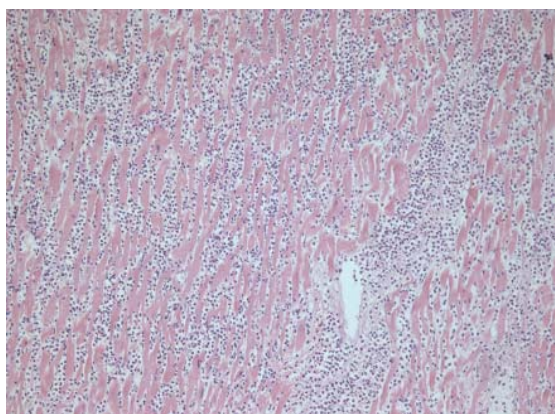


Fig. 5 – Myocardial infarction about four days old (HE, x10).

Coronary arteries had a narrowed lumen due to the presence of yellowish thickening of the intima, and serial sections of the right coronary artery found a fresh thrombus which completely closed the artery.

Macroscopic examination of other organs revealed significant atherosclerosis which mainly affected the thoracic and abdominal aorta and picture of shock in the kidneys. In the pleural cavities, on both sides, 200 mL of serous effusion was found, and examination of the lungs showed acute pulmonary edema.

Discussion

Despite the application of fibrinolysis and pPCI in the treatment of acute myocardial infarction, heart rupture remains a complication that is difficult to predict and heal. Many papers have studied the factors that may indicate the possibility of rupture in the era before pPCI and they were: one vessel coronary heart disease, previously myocardial infarction, older age, female gender and hypertension⁵⁻⁷.

In the era after PCI incidence of FWR decreased, and immediate PCI showed that can prevent development of abrupt rupture following acute myocardial infarction⁸.

In relation to the time of occurrence, rupture of the free wall of the left ventricle can be named as early rupture when it occurs within 48 hours. It is usually associated with delayed hospitalization, persistent chest pain and persistent ST segment elevation on ECG. Acute rupture of the free wall of the left ventricle is usually fatal within a few minutes due to the development of tamponade, and do not respond to standard cardiopulmonary resuscitation. Clinically, it manifests with cardiovascular collapse and pulseless electrical activity. In 30% of the cases rupture of the free wall of the left ventricle is late (subacute) and occurs after the second day⁹. The clinical picture includes recurrent chest pain and re-elevation of ST on ECG. Usually, this results in rapid deterioration of hemodynamic status of patients with permanent hypotension. Late rupture is thought to be less dependent on hypertension, but physical activity such as persistent vomiting and coughing can trigger it⁹.

Echocardiography can visualize the place of rupture of the free wall, which is accompanied by the presence of blood in the pericardium. Echocardiography cannot register this complication, and the presence of pericardial effusion is not diagnostically enough for the diagnosis of rupture. Typical finding in subacute rupture of the free wall of the left ventricle is echo mass in the pericardium, which indicates the

presence of blood clot, although it is not enough for a complete diagnosis without direct view of the rupture. This emphasizes the need for better education and best echo machines in intensive care units for better and faster diagnosis of acute myocardial infarction complications.

VSR as a mechanical complication after acute myocardial infarction in patients who did not receive reperfusion therapy occurs late, within five days, in contrast to patients who received fibrinolysis when it occurs within the first few days⁹. Risk factors for this complication are older age, infarction of anterior localization and one vessel coronary heart disease¹⁰.

Emergency operation in patients with this complication is vital. In the literature there are just single case reports which show successful operations¹¹⁻¹³. We have to emphasize that even when they are operated, mortality is extremely high⁸.

It should be noted that the operative mortality in patients with inferior infarction, as in our case, is much higher and reaches 58% as compared to patients with anterior infarction (25%)¹⁴.

DHR represents a serious and usually fatal complication, and often is primary found at surgery or autopsy in these patients. Risk factors for DHR are: older age, female gender, first myocardial infarction, anterior localization and hypertension¹.

DHS after acute myocardial infarction occurs rarely, so in the literature there are only single case reports. The only study on 10 patients with clinicopathological features of DHS is the study of Tanaka et al.¹. This is why each new case report is of some significance to this issue.

According to anamnesis, clinical findings and cardiac enzymes (creatin phosphokinase, myoglobin and troponin I) can be assumed that the myocardial infarction of inferior localization associated with right ventricular myocardial infarction in our example took about three days before admission, but that was misdiagnosed. In relation to this it was defined as a subacute rupture, as evidenced by the presence of blood clot in the pericardium, which probably in the first stage prevented acute tamponade. Elevation of ST segment on ECG and the presence of thrombus and small effusion in the pericardium was probably a sign of rupture of the free wall of the left ventricle. In our case reperfusion therapy (fibrinolysis or PCI) was not applied, myocardial infarction was three days old with hypertension, it was first myocardial infarction, and the autopsy showed one vessel coronary artery disease, so this patient had all the risk factors for myocardial rupture, which are described in the literature.

Conclusion

This case points out the need for early reporting after symptoms of acute myocardial infarction in order to prevent complications, the need for early reperfusion, and the necessity of detailed noninvasive tests in patients with acute myocardial infarction if the facilities are available. Echocardiography should be routinely performed in patients with acute myocardial infarction at admission to the intensive care unit and is always required in cases of hemodynamic instability of the patient.

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