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CASE REPORT

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Double cardiac rupture in the course of infarction

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Abstract

A 67-year-old man was admitted to hospital suffering from chest pains and electrocardiographic myocardial infarction with ST-segment elevation of the inferior wall, left and right ventricular infarction and cardiogenic shock. An emergency coronary angiography revealed total occlusion of the right coronary artery and absence of any collateral circulation. This was effectively recanalised with the implantation of a stent (TIMI 3 flow). Transthoracic echocardiography revealed a post-infarction ventricular septal defect with an essential left-to-right shunt. Standard pharmacotherapy without the possibility of connection to an intra-aortic balloon pump did not improve the haemodynamic parameters of the patient. After performing a delayed but successful recanalisation of the infarcted coronary artery within two hours there was a rupture in the free wall of the left ventricle at the border with the site of the infarct, which led to the sudden death of the patient. Correct diagnosis of the disputable double cardiac rupture was established by means of echocardiography. (Folia Cardiol. 2006; 13: 626–629)

Key words: double cardiac rupture, post-infarction ventricular septal defect, left ventricular free wall rupture

Introduction

A cardiac rupture is one of the most dramatic complications of myocardial infarction (MI). This mechanical MI complication may include the ventricular septum, the free wall of the left ventricle (LV) or the right ventricle (RV) and the papillary muscle [1].

The aim of the study is to present a case in which, after a post-infarction ventricular septal defect (PIVSD) and following infarct-related artery (IRA) recanalisation, an "acute" blow-out rupture of the free wall (FWR) of the LV occurred, which led to the sudden death of the patient.

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Case report

A 67-year old man was admitted to hospital with chest pains, which had been repeated over the previous three days, and in cardiogenic shock. On the day of hospitalisation the chest pain had lasted for 5.5 hours. The interview, which focused on arterial hypertension, lipid disorders and diabetes, gave negative results. The patient was an occasional smoker.

On the basis of the clinical symptoms and electrocardiographic features of the acute ST-elevation myocardial infarction (STEMI) of the interior wall, the LV interior wall and the RV and the elevated activity of troponin I and CKMB characterising the blood, the patient was qualified for urgent coronary angiography. However, it was impossible to put him on the intra-aortic balloon pump (IABP) before the invasive study, because the only pump was being used to treat another patient. Coronary angiography showed total coronary occlusion of the right coronary artery (RCA) in segment 2 without any collateral circulation (Fig. 1A) or significant changes in

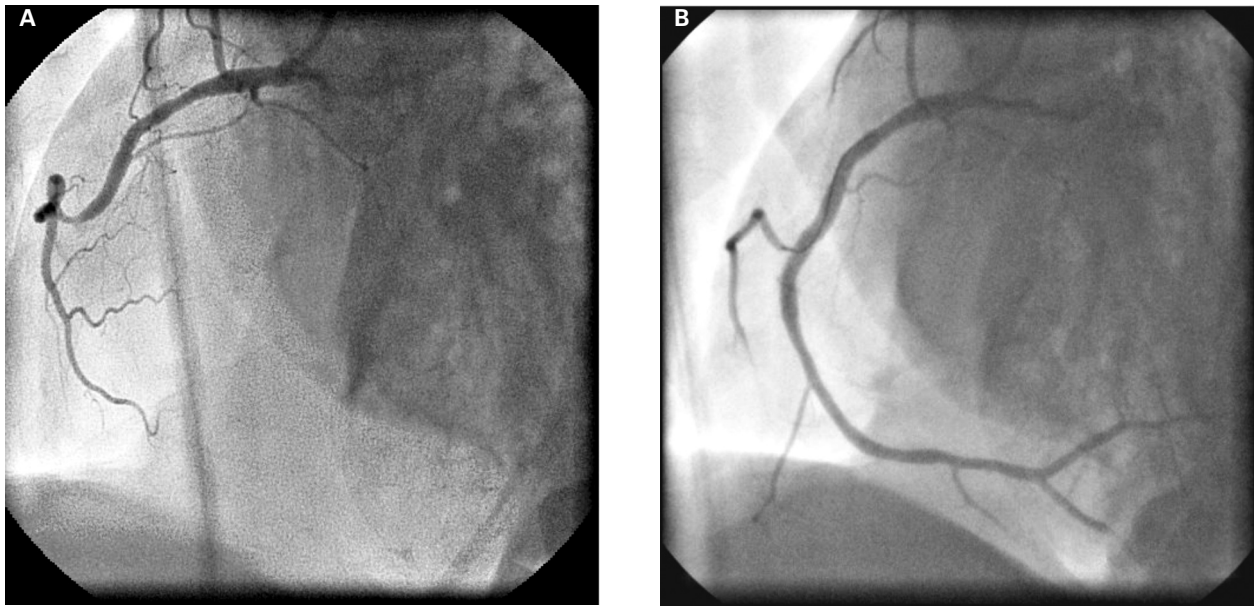

Figure 1. A. Angiography of the right coronary artery with total occlusion in segment 2. **B.** State after its recanalisation and stent implantation.

the remaining epicardial coronary arteries. At the same time a successful recanalisation of the RCA with the implantation of a stent Express 3.0/16 mm was carried out, achieving TIMI grade 3 flow (Fig. 1B) (A. Jachniewicz, MD, Haemodynamic Laboratory, L. Perzyna Provincial General Hospital, Kalisz).

After percutaneous coronary intervention the patient's state did not improve, and symptoms of cardiogenic shock remained together with a holosystolic murmur along the left edge of the sternum, which had also been heard at the beginning of his hospitalisation as well as a palpable mumble. In transthoracic echocardiography (TTE) with colour Doppler imaging the following was confirmed: the LV not enlarged; hypokinetic inferior and posterior wall motion, and EF = 35% (according to Simpson method), enlarged RV with hypokinetic free wall motion, left-to-right shunt through the oblique defect in the posterior-interior part of the ventricular septum (10 × 15 mm) (Fig. 2), Qp/Qs=1.94:1.0, moderate pulmonary hypertension and mitral (++) and tricuspid valve (+) insufficiency.

From the beginning of hospitalisation the patient was given liquids (dextran 40000, 0.9% NaCl), dopamine (3–5 μg/kg/min) *i.v.*, furosemide (2 amp.) *i.v.* and nitroglycerin (10 μg/kg/min) *i.v.* His general serious state worsened as a result of respiratory disorders, which required intubation and mechanical ventilation. His growing haemodynamic instability was the reason for the control TTE. This showed pericardial effusion (the maximum in the diastolic lateral wall being 25 mm, in the posterior 35 mm and at the tip 27 mm), diastolic falling back of the left atrium, RV free wall and left atrium, dilation of the inferior vena cava and, in a study made with Doppler of the tricuspid valve, variability of breathing flow, confirming cardiac tamponade without visualisation of FVR LV. Pericardiocentesis giving 210 ml of blood (haematocrit value complying

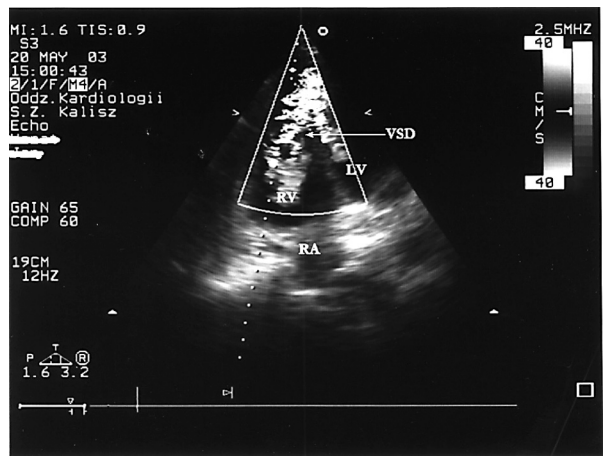

Figure 2. Echocardiographic examination. Apical four-chamber modified view. In examination with colour-coded Doppler an evident flow appears from the left ventricle (LV) to the right ventricle (RV) through the ventricular septal defect (VSD), right atrium (RA).

with circumference blood) showed no haemodynamic improvement. Resuscitation of the patient turned out to be ineffective. Two hours after recanalisation of the totally occluded coronary artery the patient's death was ascertained in the electromechanical dissociation mechanism.

The autopsy report stated that the direct cause of death was cardiac tamponade in the course of intramural rupture at the border of the healthy and necrotic changed LV posterior free wall in an area of 3 × 2 cm with a mural rupture of 1 cm (I type according to Perdigao [2]). The three-day MI ranged from the LV interior to the posterior wall and the RV wall. The oblique defect was located in the posterior-interior wall of the ventricular septum of the necrotic myocardium (1 × 1. 5 cm). In the RCA no acute thrombosis was noted in the implanted stent.

Discussion

The case presented, with PIVSD and LV FWR after successful but delayed recanalisation of the IRA, is a very rare one in which structural myocardial lesions have occurred together. Dudra et al. [3] described a patient with PIVSD and LV FWR directly after a successful percutaneous recanalisation of the IRA. Immediate cardiac surgery allowed the patient to live for over two more years.

In case of the patient described here PIVSD appeared on the day of hospitalisation and the diagnosis was made with the help of TTE. This examination with colour-coded Doppler helped to determine the area and size of the septum lesion as well as the magnitude of the interventricular shunt [4]. Oxymetric measurement with a Swan-Ganz catheter of blood and the increase in pulmonary artery oxygenation decided the diagnosis [5].

PIVSD in the case of this patient was connected with single-vessel disease without any collateral circulation and correlated with the MI localisation. This confirms the view that a cardiac rupture occurs more often in the case of single-vessel disease without the presence of any collateral circulation [6].

The three-day delay in hospitalisation was due to the fact that the patient described did not consult a doctor, which significantly increased the risk of FWR. The delayed myocardial reperfusion achieved on the third day of MI could, after recanalisation of the IRA by percutaneous angioplasty, have increased the LV FWR risk. The study by Yip el al. [6] confirms this observation. The authors demonstrated that there was a greater risk of FWR in patients who underwent delayed percutaneous recanalisation of the IRA. The LATE and GISSI I studies suggest a similar regularity on application of delayed fibrinolytic therapy in acute MI [7, 8]. The mechanism responsible for the ruptures in these cases may be conditional upon the post-reperfusion injury of cardiomyocytes and/or haemorrhage of the MI area.

The application of vasodilators (nitroglycerin and sodium nitroprusside) and positive inotropic drugs (dobutamine, dopamine and milrinone) in PIVSD therapy decreases afterload and increases myocardial contractility [4]. Putting the patient on IABP may additionally reduce the magnitude of the interventricular shunt and improve coronary perfusion [9]. The aim of standard pharmacotherapy is to provide haemodynamic stabilisation until percutaneous occlusion of the PIVSD with the occluder or corrective cardiac surgery has taken place. In the present case the pharmacological treatment did not improve the haemodynamic parameters. The impossibility of putting the patient on IABP would neither have reduced the magnitude of the interventricular shunt nor provided haemodynamic stabilisation.

Mortality in "acute" FWR amounts to nearly 100%. Patients with this rupture who qualify for successful cardiac surgery are rarely described [3, 10]. The acute cardiac LV FWR and instant increase in blood in the pericardial sac which occurred in the patient described here resulted in cardiac tamponade and ended with the patient's decease.

The correct diagnosis in this case was made on the basis of an echocardiographic study of the heart. Any deterioration of a patient's haemodynamic condition in acute MI is an indication for control echocardiography.

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