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Case Report

Ventricular septal rupture with hemodynamically important left-to-right shunt, right ventricular myocardial infarction, transient type III atrioventricular block and the development of left ventricular aneurysm as a complication of sub-acute myocardial infarction of the bottom wall accompanied by post-infarction unstable angina pectoris

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ABSTRACT

Ventricular septal rupture is a serious mechanical complication of myocardial infarction with important hemodynamic consequences. Without a rapid diagnosis and correction by surgical intervention, the short-term mortality of these patients is higher than 90%. We report the case of a patient with acute inferior myocardial infarction and a ventricular septal rupture with early diagnosis based on clinical examination and transthoracic echocardiography and postponed successful surgical correction.

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1. Introduction

Ventricular septal rupture (VSR) is a serious mechanical complication of myocardial infarction. The occurrence of RVS has significantly decreased due to the implementation of routine reperfusion treatment of acute coronary syndromes but it still is a complication responsible for approximately 5% of the causes of cardiogenic shock which is a complication of myocardial infarction [1–3]. Despite the availability of sophisticated diagnostic and particularly therapeutic methods, VSR-related mortality is still very high and the questions regarding optimum timing of indication and type of VSR closure and the way of usage of mechanical cardiac supports or intra-aortic balloon counterpulsation in this indication have not been quite clearly answered yet.

2. Case report

A 62-year-old male, smoker, otherwise so far without significant comorbidities except for five years of adequate treatment of arterial hypertension was chosen to undergo acute selective coronarography for intermittent quiescent stenocardias with progressing dyspnea within post-infarction unstable angina pectoris complicating acute Q-myocardial infarction of the bottom wall which had occurred approximately one week prior to hospitalization. The patient's physical examination on admission to hospital: no acute chest pain, no dyspnea, no palpitations, normal blood pressure and pulse rate, no pathological findings on auscultation of the heart—no murmur, regular, distinct heart sounds, with few bilateral basal pulmonary crackles; no pathological finding on palpation of the abdomen; no lower extremities edema, peripheral pulsation palpable. Based on coronarographic findings of the closure of the proximal segment of the right coronary artery (RCA) during quiescent stenocardias within post-infarction unstable angina pectoris, angioplasty was performed with direct implantation of one metallic stent (3.5×22 mm) into the culprit lesion. Subsequently, due to peripheral embolization, eptifibatide was administered as a bolus dose intravenously with a good effect. The intervention itself was performed successfully without complications; subsequently, however, transient hypotension occurred along with transient type III atrioventricular block, which, after a bolus dose of atropine, volume expansion and brief administration of noradrenalin in vivo, faded away spontaneously as did the hypotension. Subsequently, it is necessary to pharmaceutically correct systemic hypertension to reach normal values.

Clinical examination showed evident maximum intensity holosystolic murmur at Erb's point propagating toward the point. There was no audible congestion in the lungs. Blood pressure was generally within normal range and stroke frequency at a volumetric dose of betablocker (metoprolol succinate 25 mg daily) ranged between 80–90/minute.

Additionally, echocardiographic examination was complemented, with left ventriclar aneurysm found posteroseptally in the basal and medium segments with evident extensive VSR directed tangentially toward the apex of the heart. Color Doppler mapping showed apparent significant left-to-right shunt flow which was quantified through the ratio of flow-throughs in the pulmonary artery and the left ventricular outflow tract (Q_p/Q_s) of 3.9:1. Global systolic function of the left ventricle (LV) expressed by the parameter of left ventricular ejection fraction (LVEF) was extreme—50–55%. The right ventricle (RV) was significantly dilated (end-diastolic dimension with the base in apical 4-cavity projection was 48 mm). Moreover, longitudinal systolic dysfunction of the right ventricle was evident. Due to a planned surgical correction of VSR including a considered remodeling surgery, contrast cardiac magnetic resonance was performed.

With respect to hemodynamic stability, the patient was subjected to intensive monitoring (arterial blood pressure invasively, and further noninvasive monitoring), and it was decided to postpone the surgery, which had been indicated on the 20th day of hospitalization. The surgery was performed successfully and the post-surgical course including rehabilitation was uneventful (Figs. 1–5).

3. Discussion

Rupture of the free wall and ventricular septal rupture are among the most catastrophic complications of myocardial infarction and are etiologically responsible for approximately 5% of cardiogenic shocks with a mortality reaching up to 90% in the case of ventricular septal rupture. The incidence of both types of ruptures in the reperfusion era of myocardial infarction treatment is 1.4% [3]. Cardiogenic shock is initially present in 50% of VSR patients and the prognosis for individuals with rupture in posteroseptal localization is more serious in comparison to patients with apical defect [1–3,14,15]. Moreover, rupture of the papillary muscle with acute mitral regurgitation occurs in 1% of patients with acute myocardial infarction and in 8% of patients with cardiogenic shock complicating acute myocardial infarction [4].

Although according to The Global Registry of Acute Coronary Events (GRACE), for patients with acute coronary syndrome with ST-segment elevations (STEMI), the risk of rupture is higher in the group of patients with a longer time-to-reperfusion in comparison to patients treated conservatively, delayed reperfusion strategy has not been shown as an independent predictor of rupture risk. This paradox is probably explicable by selection bias which results in the fact that patients who have this relatively very rare complication very early were probably not included in the registry at all. STEMI or the premise of transmurality of necrosis, age and female gender are frequently cited as traditional risk factors for rupture. The occurrence of myocardial rupture is cited to be 1/111 in STEMI patients and 1/588 in NSTEMI patients which supports the assumption that transmurality of infarction represents a risk factor. On the contrary, the use of a beta blocker is probably connected to prophylactic actuation toward the rupture [3].

In the case of rupture of the free wall and papillary muscles, the clinical picture is so emergent that transferring the patient to the operating room including the necessary preparation prior to surgery (bedside pericardiocentesis, intubation, and artificial pulmonary ventilation, introduction of mechanical cardiac support) is normally inevitable. In the

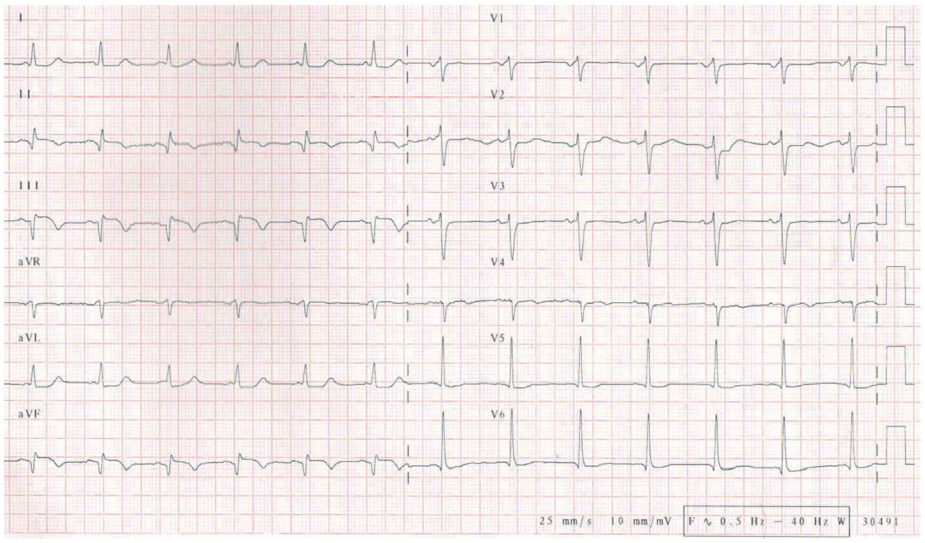

Fig. 1 – Baseline electrocardiography (ECG). **The baseline ECG curve shows already developed Q waves in leads II, III and VF, and simultaneously persisting elevation of the ST segment with symmetrically negative T waves as correlate of myocardial infarction of the bottom wall (Q-MI) in an outpatient.**

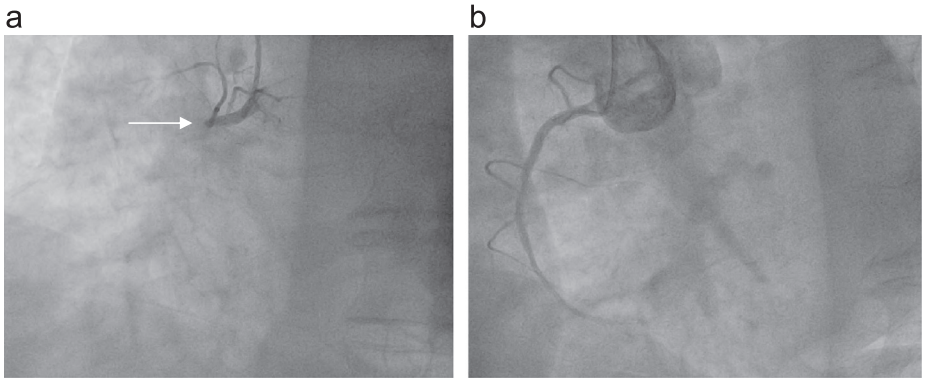

Fig. 2 – Selective coronarography (SCG). **During contrast agent application to the right coronary artery, occlusion of the proximal segment, which, due to post-infarction instable angina pectoris, was treated using angioplasty with direct stent implantation, is apparent.**

case of VSR, further patient management is significantly influenced particularly by hemodynamic (in)stability with a potential development of multi-organ failure on the one hand, and on the other, the necessity to create optimum scarring of the necrotic tissue of myocardium to perform as stable suture of the ventricular septum as possible. Due to the fact that there is no randomized study, there still remain unanswered questions regarding both the optimum timing for correcting the rupture and the method to be used (catheter vs. surgery), or the management of supportive pharmacological treatment and indicating mechanical cardiac supports in these patients [14,15].

The optimum **timing** for correcting the rupture is determined especially by the current hemodynamic status of the patient. In terms of ensuring a stable suture of the rupture, it is probably useful to postpone surgical management by approximately 2–3 weeks which is sufficient for scarring to occur in the area of the necrotic myocardium. On the other hand, the development of cardiogenic shock followed by a rapid development of multi-organ failure is a signal for urgent correction of VSR.

This is associated with another vital question regarding **the method of correcting a rupture**. Percutaneous closure of a rupture is, for its smaller invasiveness, considered an alternative to a surgical intervention. Nevertheless, experience with this approach is relatively limited to small sets or case reports. The result of the procedure is often not optimal and this intervention carries a lot of serious complications. In a prospective follow-up study of the results of a very early performed catheter closure of VSR using the AMPLATZER Septal Occluder in 29 patients with a routinely introduced IABK, 30-day survival amounted to 35%, and, in patients in cardiogenic shock (with a 55% occurrence in the set being monitored), mortality even reached 88%. In 14% of patients the attempt to close VSR resulted in failure. When the occluder was implanted successfully, a reduction of intracardial left-to-right shunt described by the ratio of Q_p/Q_s from original 3.3:1 to 1.4:1 was achieved. Serious complications such as absence of reduction of hemodynamic significance of the shunt, rupture of the left ventricle, or embolization of the occluder occurred in 41% of patients treated very early using

the catheter technique. Therefore, surgical suture is still considered the gold standard method and, as of now, there has not been any study reported in the literature that would prove superiority of catheter closure to surgical correction of a rupture. Nevertheless, serious comorbidities of the patient with a rapidly developing cardiogenic shock are the reason for individual preference of the catheter technique, which has been well attested by a lot of case studies showing positive results of this technique [7–13]. In addition to that, it is possible to carry out implantation of septal occluder in the place of the rupture in order to reduce left-to-right shunt, and based on the result, postponed surgical correction would possibly be complemented with revascularization surgery [7,10,11]. In this case it is appropriate to establish viability of the myocardium focused mainly on the perfusion territories of the expected revascularization [5,6,7,10,11].

Supportive therapy is again significantly determined by the patient's hemodynamic status. In the case of a completely stable patient, it is appropriate to modify hypertension probably using an intravenously applied beta blocker for an ultra short period of time, possibly in combination with nitrate or nitroprusside. When there is a progressive reduction in the left-sided cardiac output and systemic blood pressure, supporting the circulation with vasopressors or possibly with positively inotropic pharmaceuticals (dobutamin or levosimendan) is generally necessary. In the case of rapid deterioration of circulation, introduction of intra-aortic balloon counterpulsation (IABK) or mechanical cardiac support is indicated [13,14,15]. Although data from the SHOCK II randomized study do not support routine use of IABK in patients with acute myocardial infarction in cardiogenic shock, it is necessary to bear in mind that mechanical complications were among the exclusion criteria of this randomized study.

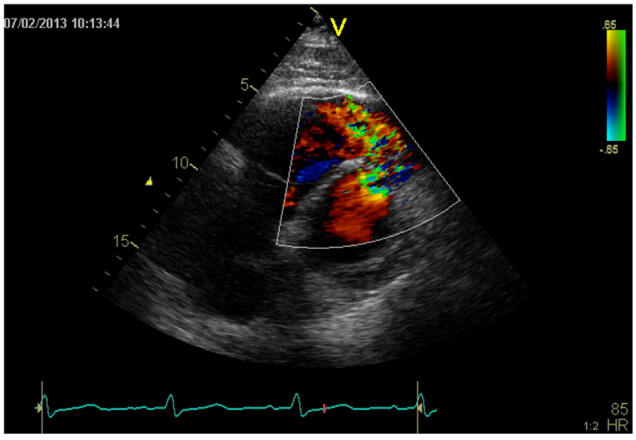

Fig. 3 – Echocardiographic examination. **Dyskinesia is apparent in the basal posteroseptal segment, with evident rupture tangentially directed toward the point. Left-to-right shunt was quantified by the ratio of the flow-through in the pulmonary artery and the outflow tract of the left ventricle (Q_p/Q_s) 3.9:1.**

4. Conclusion

Mechanical complications related to myocardial infarction are an emergency requiring very prompt echocardiographic

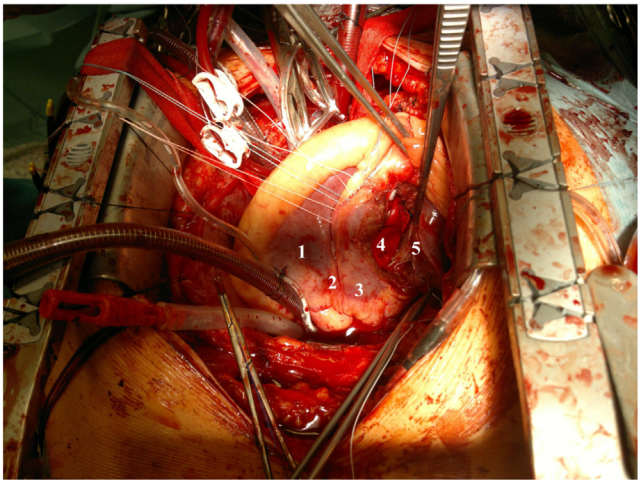

Fig. 5 – Surgical finding: **(1) the right ventricle, (2) the ventricular septum, (3) the left ventricle, (4) a patch, fixed to the mitral valve annulus, excluding the necrotic septum and covering the defect, and (5) the posteromedial papillary muscle.**

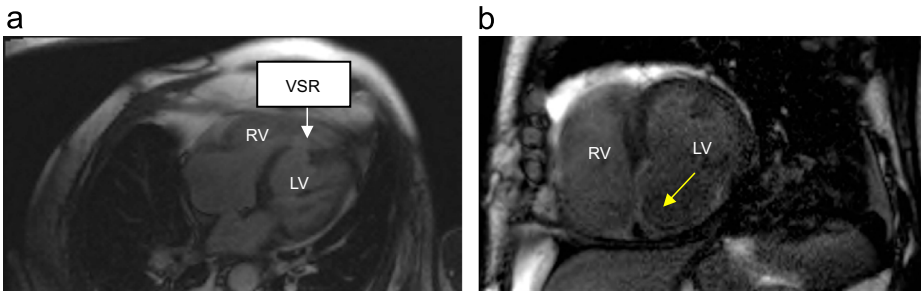

Fig. 4 – Cardiac magnetic resonance imaging. **A thinning of the myocardial wall in the posteroseptal segments of the left ventricle (LV) of as much as 2 mm with a clear formation of an aneurysm is apparent. The aneurysm is formed inferoseptally and inferiorly (3, 4) mainly in the basal segments, and inferoseptally and inferiorly (9, 10) in the middle segments. In the sequences showing late enhancement of the myocardium (yellow arrow), there is an apparent transmural scar in segments 3, 4, 9 and 10. In the basal segments, the right ventricular (RV) myocardium is created by a transmural scar and the right ventricle is dilated. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)**

diagnosis. The management of patients (the timing of correction and the technique of realization) with ventricular septal rupture is, at the time of diagnosis, determined especially by the current hemodynamic status. Instability of circulation is a signal for a very rapid escalation of treatment, particularly by using mechanical cardiac supports and considering urgent indication for correcting the rupture.

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