Case reports

Spontaneous closure of postinfarction ventricular septal rupture. A case report

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Spontaneous closure of a postinfarction ventricular septal rupture is extremely rare. We present such a case in which the postinfarction ventricular septal rupture closed spontaneously during follow-up. We postulate that the spontaneous closure of the ventricular septal rupture was probably due to thrombosis in the apical and septal aneurysm.

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Introduction

Ventricular septal rupture (VSR) is a rare but generally fatal complication of acute myocardial infarction. It has been reported that over 50% of such patients die within the first week of septal rupture, while 2 months later the mortality rate increases up to 87%[1,2]. An early diagnosis and prompt surgery are usually required. Spontaneous closure of a postinfarction VSR is extremely rare. To our knowledge, there is only one case published in the English literature[3]. We present another case of a postinfarction rupture of the ventricular septum which closed spontaneously during follow-up.

Case report

A 73-year-old Caucasian woman was admitted to our hospital for prolonged severe chest pain and dyspnea. At the time of admission her overall clinical status was poor; she had many comorbidites, including a right hemiparesis due to an ischemic stroke, rheumatic arthritis, severe anemia requiring blood transfusion, chronic renal insufficiency, and a lymphoangioma of the spleen. Her medical history included admission to the emergency department because of unstable angina 1 month previously. During the last days prior to the present hospitalization she had complained more frequently of chest pain but was unable to clearly describe the last episode and its duration. Physical examination documented a blood pressure of 180/100 mmHg and a mild and soft systolic murmur graded about 2/6 along the left sternal border. The creatine kinase (CK)-MB serum level at the time of admission was 155 ng/ml (the normal value for CK-MB is < 5.0 ng/ml at our Institution); electrocardiography was suggestive of the evolution of an acute extensive anterior myocardial infarction (Fig. 1). Transthoracic echocardiography confirmed the diagnosis of an apical myocardial infarction which had evolved to an apical aneurysm and mild mitral insufficiency. The left ventricular global systolic function was preserved, with a Simpson's biplane left ventricular ejection fraction (LVEF) of 59%. Because the exact time of onset of the myocardial infarction was not known, thrombolysis was not performed. The initial course was uncomplicated, with medical therapy rapidly relieving the chest pain and a stable clinical status. In view of the patient's poor clinical conditions, we decided not to submit her to coronary angiography. She was then discharged after 7 days with the following daily therapy: metoprolol 50 mg, enalapril 10 mg, furosemide 25 mg, isosorbide mononitrate 60 mg, acetylsalicylic acid 160 mg.

One week later, the patient was rehospitalized to our department because of the recurrence of angina and palpitations last-

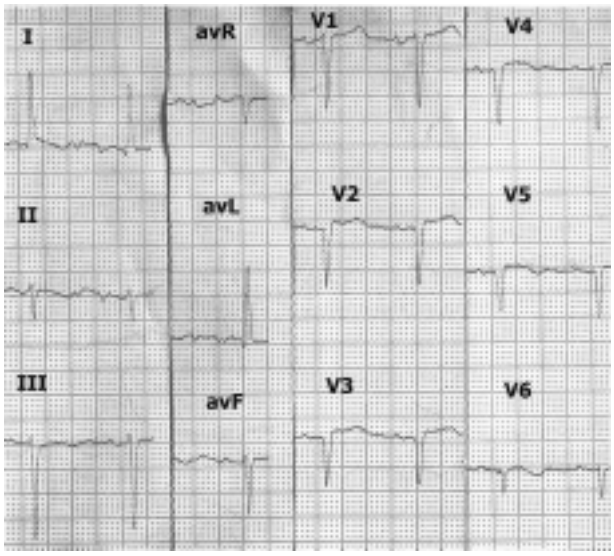

Figure 1. *Electrocardiogram on arrival showing signs of a subacute extensive anterior myocardial infarction. A deep and wide Q wave associated with ST-segment elevation may be seen on leads V_1-V_5.*

ing > 1 hour. Upon arrival, her blood pressure was 110/70 mmHg; a new precordial systolic 3/6 cardiac murmur was found. No dynamic change of the myocardial enzymes was detected. Electrocardiography revealed atrial fibrillation with a rapid ventricular rate. Electrical cardioversion was performed 2 hours after admission with restoration of sinus rhythm. A new echocardiogram was performed in order to evaluate the cause of the cardiac murmur. A large apical aneurysm was found; the distal part of the ventricular septum was thin and dyskinetic; it was dilated and similar to a septal aneurysm bulged towards the right ventricle during systole (diameters of the septal aneurysm about 1.8 × 1.8 cm). There was a 0.6 cm diameter myocardial tear at the center of the septal aneurysm; at color Doppler a turbulent jet with a maximal velocity of 5.3 m/s, originating from the left ventricle and entering into the right ventricle, was found across the defect (Fig. 2). LVEF was 60%. Mitral flow analysis revealed an impaired relaxation pattern, with an E/A ratio of 0.7. Surgical consultation was performed immediately. In view of the good response to medical therapy and the presence of other severe comorbidities, it was decided to continue with medical therapy and to submit the patient to serial echocardiography. The patient was asymptomatic and her clinical course uneventful. Cardiac catheterization, performed 1 month later, revealed total occlusion of the distal part of the anterior descending coronary artery and 60% stenosis of the right coronary artery. Ventriculography confirmed the presence of a large apical aneurysm without passage of the contrast medium from the left to the right ventricle. Despite the fact that ventriculography was suggestive of the VSR closure, a small left to right ventricular shunt was still identifiable at echocardiography and persisted until discharge; the last echocardiogram performed just before discharge revealed the presence of an apical mural thrombus inside the aneurysm.

Eight months after the infarction, the patient was rehospitalized because of a brady-tachycardia syndrome necessitating the implantation of a permanent pacemaker. Transthoracic echocardiography was repeated. The left-to-right shunt had completely disappeared; most of the chamber of the apical and septal aneurysm was filled with thrombus. LVEF was 25% (Fig. 3).

Follow-up included regular visits. At 3 years of follow-up the patient was still asymptomatic and even though the LVEF was still 25-30%, her clinical conditions were stable.

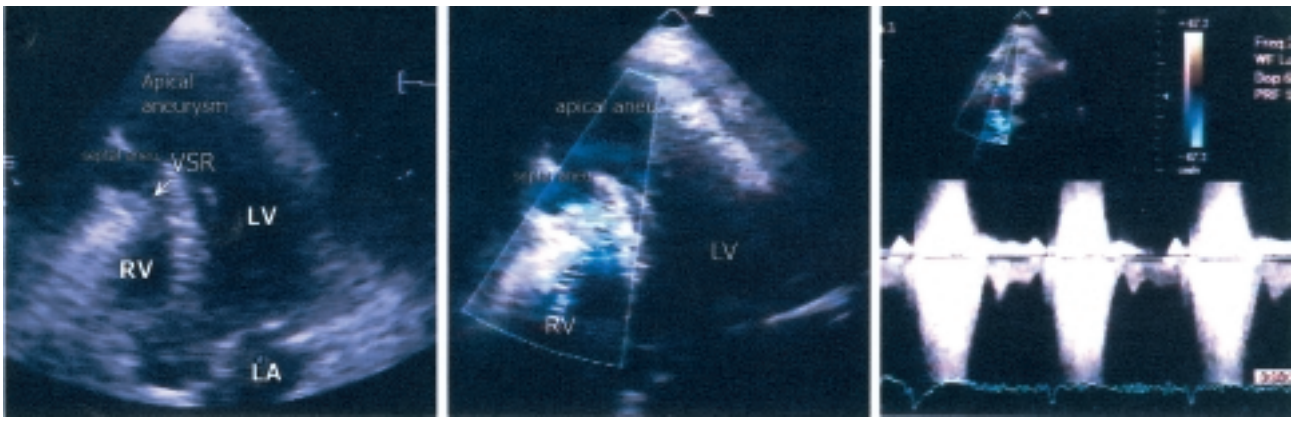

Figure 2. *Transthoracic echocardiography performed at the time of the second admission revealed that there was a rupture of the distal ventricular septum.* Left panel: *two-dimensional echocardiographic image of the apical 5-chamber view. A large apical aneurysm was shown; the distal part of the ventricular septum was thin and dyskinetic, it was dilated and, similar to a septal aneurysm, bulged towards the right ventricle (RV) during systole. There was a small* (arrow) *dehiscence of the myocardium at the center of the septal aneurysm.* Middle panel: *color Doppler echocardiography of the same view reveals a turbulent jet (*bright blue*) across the defect and entering into the RV.* Right panel: *continuous wave Doppler shows the systolic jet across the defect. LA = left atrium; LV = left ventricle; VSR = ventricular septal rupture.*

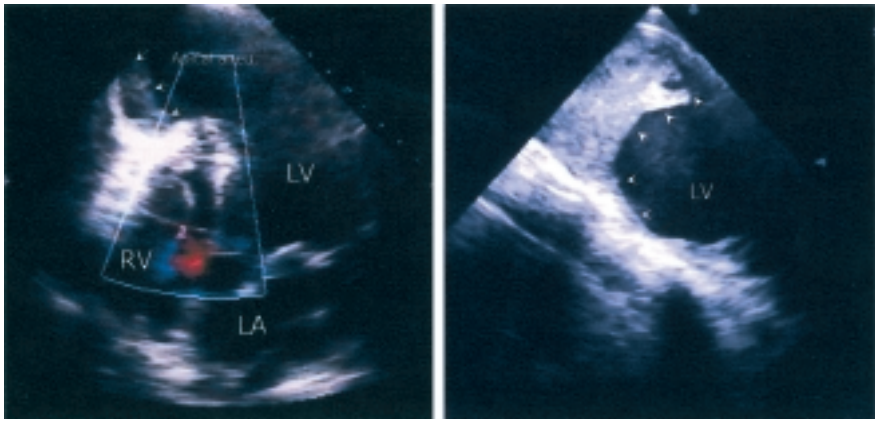

Figure 3. *Echocardiography was repeated 8 months after the infarction.* Left panel: *color Doppler echocardiography of the same view as in figure 2 shows that the left-to-right shunt had completely disappeared; a large apical mural thrombus inside the aneurysm is also evident* (arrow). Right panel: *a transesophageal echocardiographic image of the modified transgastric view shows that most of the chamber of the apical and septal aneurysm was filled by mural thrombus* (arrow). *LA = left atrium; LV = left ventricle; RV = right ventricle.*

Discussion

The prevalence of a postinfarction VSR is approximately 2%[4]. Although the mortality rate of acute myocardial infarction has decreased consistently in the last years, the prognosis of patients with a VSR is very poor, especially in case of conservative treatment. More than 5% of myocardial infarction-related deaths are due to a VSR[5]. Furthermore, more than 80% of patients with a postinfarction VSR die within 2 months of the rupture.

VSR usually occurs after a large transmural infarction, mostly within the first week. It is single in most cases; the most common site of the defect is often at the center of a septal aneurysm, and its size varies from 0.5 to several centimeters. The rupture may be a "through and through" hole, as in our case, or irregular and tortuous.

In most cases, an early diagnosis and timely surgery are mandatory for survival. A newly developed systolic murmur after a recent myocardial infarction, usually harsh and loud, and accompanied by shock and signs of congestive heart failure or frank pulmonary edema, requires prompt investigation. The diagnosis of a VSR must be considered and differentiated with other serious complications such as severe mitral regurgitation. Two-dimensional echocardiography combined with color Doppler is a reliable tool for a rapid diagnosis and follow-up. It allows an accurate determination of the location and size of the defect; it permits the evaluation of the regional and global heart function, the estimation of the right ventricular pressure, the assessment and differentiation with other possible associated complications and abnormalities such as valve regurgitation and the identification of thrombosis. Echo "drop-out" along the septum at the site of the tear at two-dimensional imaging and/or Doppler evidence of a left-to-right shunt at the site of rupture clarify the diagnosis. As compared with ventriculography, echocardiography seems more sensitive. In our case, although the ventriculographic result was negative, color Doppler still revealed a small interventricular shunt across the septal tear at 1 month after the initial rupture. Furthermore, it is noninvasive and repeatable and in view of the fact that it may be performed at the patient's bedside it is more suitable for the critically ill subject.

In view of the coexistence of numerous comorbidities, it was decided not to submit the patient to surgery. Furthermore, the negative result of ventriculography suggested that the dimensions of the shunt were minimal, even though it was still identifiable at echocardiography, and that there was a tendency towards a spontaneous closure of the septal rupture. On the other hand, the clinical course was uneventful. The patient remained asymptomatic even though the LVEF was very low at the last follow-up.

The small size of the defect could explain the relative hemodynamic stability after the acute event. In addition, spontaneous closure could have been facilitated by the small size of the defect. The thrombosis within the aneurysm may have also prevented the extension of the defect and may have even favored its spontaneous closure. However, spontaneous closure of a postinfarction VSR is a very rare occurrence in clinical practice. To our knowledge, there is only one similar case reported in the English literature[3]. There are some similarities between the two cases. As in our case, VSR had occurred as a consequence of an acute anteroseptal myocardial infarction. Other similarities included the small size of VSR and its location in the distal part of the interventricular septum, and the fact that the patient was hemodynamically stable, that the rupture was correctly diagnosed by means of transthoracic echocardiography and that, owing to the presence of comorbidities (severe chronic obstructive airway disease in the case described by Williams and Ram-

sey[3]), the patient was not submitted to surgery. Although the exact mechanism of spontaneous closure of a postinfarction VSR was not mentioned, the authors suggested that a septal rupture associated with an anterior infarction tends to be apical and has a better prognosis than rupture complicating an inferior infarction which involves the basal septum. This may have also been true for our patient.

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