

## Case Report

## Ventricular Septal Rupture After Acute Myocardial Infarction

IONUT DONOIU, OCTAVIAN ISTRATOAI, DAN-DOMINIC IONESCU

*Craiova University of Medicine and Pharmacy, Craiova Cardiology Center, Craiova, Romania*

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Ventricular septal rupture is a rare complication of acute myocardial infarction with important hemodynamic consequences. Spontaneous closure is extremely rare. 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 myocardial infarction and a ventricular septal rupture that was partially closed by the formation of a thrombus. Early diagnosis was obtained in the emergency room, based on clinical examination and transthoracic echocardiography.

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*Address:*  
Ionut Donoiu

*Department of  
Cardiology, University of  
Medicine and Pharmacy  
of Craiova, Craiova  
Cardiology Center  
1 Tabaci St., 200640  
Craiova, Romania  
e-mail: [i.donoiu@gmail.com](mailto:i.donoiu@gmail.com)*

**T**he occurrence of ventricular septal rupture (VSR) after acute myocardial infarction has become an uncommon complication in the reperfusion era; however, this condition is associated with a high mortality rate, even if immediate surgical repair is attempted. Our case emphasizes the risk factors and evolution of this condition.

### Case presentation

A 74-year-old hypertensive woman was admitted through the emergency department for epigastric pain and dyspnea that had started two days previously. The patient's condition worsened progressively, with decline of the cognitive state.

Physical examination revealed a regular pulse of 120 beats/min. The blood pressure was 120/70 mmHg and there was a harsh grade IV/VI systolic murmur best heard at the apex, radiating to the axilla. She had hepatomegaly with increased jugular venous pressure. Pulmonary rales were absent and there was no peripheral edema. There were no neurological signs of focal lesion.

The 12-lead electrocardiogram (Figure 1) showed sinus rhythm at 120 beats/

min, low QRS complex voltage in the limb leads, complete right bundle branch block, q waves and a 4 mm ST elevation in the anterior leads.

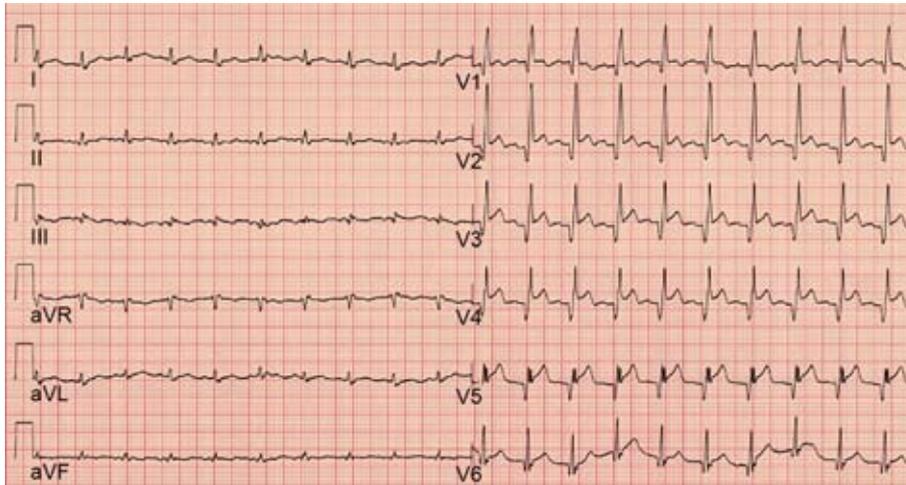
Serum troponin T level at admission was >2 ng/ml, with N terminal pro-natriuretic brain peptide (NT-proBNP) >3000 pg/ml.

Transthoracic echocardiography (Figure 2A & B) revealed a small rupture of the apical ventricular septum causing a VSR with left-to-right shunt. In the right ventricular side of the VSR there was a thrombus partially occluding the defect. The interatrial septum seemed to be intact. Transesophageal echocardiography was not performed.

Ultrasound examination of the lower limbs did not show any signs of deep vein thrombosis.

A diagnosis was made of acute anterior myocardial infarction complicated by a VSR and right ventricular thrombus.

In view of the late presentation, thrombolysis was not indicated. Coronary angiography was not performed because our center does not have primary PCI capabilities. Closure of the VSR was not undertaken as the patient's family refused her transfer to a cardiovascular surgery center. Therefore,



**Figure 1.** Electrocardiogram at admission.

she received only supportive medical treatment. The hemodynamic status worsened gradually requiring inotropic support with dopamine and dobutamine.

Repeated echocardiography examinations during the following days showed the persistence of the thrombus near the VSR, excluding the possibility of a thrombus in transit.

On day seven, the family requested that she be discharged, contrary to recommendations.

### Discussion

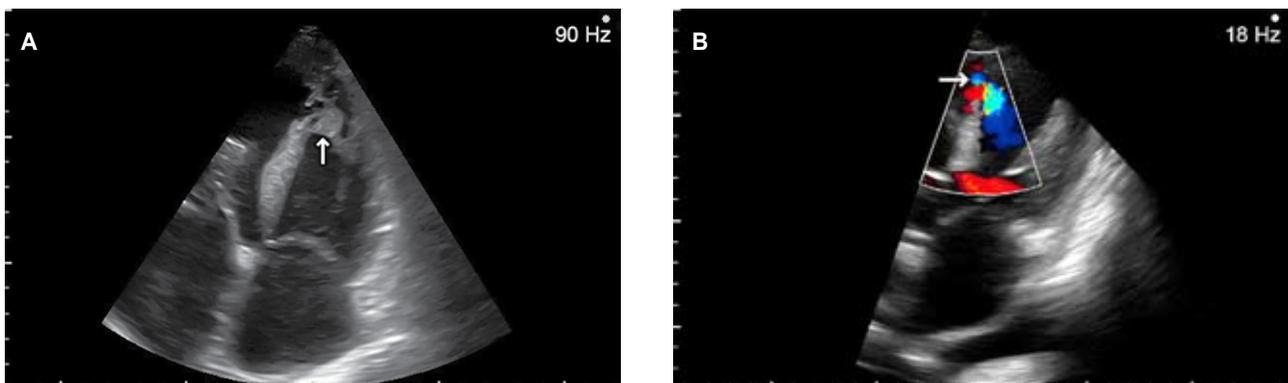
VSR is a rare but serious complication of acute myocardial infarction that is, in almost all cases, fatal without early surgical intervention. It had an incidence of 1-3% in the era before reperfusion therapy, decreasing with the introduction of thrombolytic therapy.<sup>1,2</sup> VSR is more infrequent than a rupture of the ventricular free wall. Women are affected more commonly than

men. Other risk factors are age, non-smoking and hypertension.<sup>3</sup>

VSR usually occurs 2-8 days after the infarction and often precipitates cardiogenic shock. There are a few reported cases of silent myocardial infarction followed by an asymptomatic VSR or presenting as chronic congestive heart failure.<sup>4</sup> The size of the defect determines the magnitude of the left-to-right shunt and consequently the hemodynamic deterioration, which affects survival. Complete spontaneous closure of such an acquired defect is extremely rare.<sup>5</sup>

There are three types of VSR (the original classification made by Becker and van Mantgem was for free-wall rupture): in type I there is an abrupt tear in the wall without thinning; in type II, the infarcted myocardium erodes before rupture and is covered by a thrombus; and type III represents the perforation of a previously formed aneurysm.<sup>6</sup>

The blood supply to the septum originates from



**Figure 2.** A. Transthoracic echocardiogram in the apical four chamber view. The ventricular septal rupture is shown towards the apex, with a 12 mm diameter thrombus (arrow) in the right ventricle. B. The left to right shunt (arrow).

branches of the left anterior descending coronary artery, the posterior descending branch of the right coronary artery, or the circumflex artery when it is dominant. VSR has equal frequency in anterior and non-anterior infarctions.<sup>7</sup> Anterior MI is associated with rupture of the apical septum, in inferior MI, it often occurs at the base of the heart. An MI associated with a VSR is usually extensive. Early treatment of MI with thrombolysis may reduce the incidence of VSR, influence the time to septal rupture and improve the outcome.

Development of thrombi in the left ventricle after myocardial infarction is not uncommon. In contrast, right ventricular thrombi are rare. Right heart thrombi may also develop *in situ* as a result of blood stagnation in patients with a cardiomyopathy or with catheter or pacemaker leads. Floating right heart thrombi in most cases are seen by accident, in transit from deep veins to the pulmonary artery. If a patent *foramen ovale* or atrial septal defect is present, a thrombus can cross from the right atrium directly into the left atrium with paradoxical embolism. The direct demonstration of paradoxical embolism by imaging a thrombus traversing the *foramen ovale* is unusual.<sup>8,9,10</sup>

Our case illustrates the typical risk factors of VSR: an extensive acute myocardial infarction (anterior, with right bundle branch block) in an old hypertensive, non-smoking woman, with no history of angina. We describe a rare situation in this rare condition – the partial occlusion of the defect by a thrombus – which may have slowed the evolution.

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