Ventricular Septal Rupture After Myocardial Infarction*  
Detection by Transesophageal Echocardiography  

David Harpaz, M.D.; Pratima Shah, M.D.; Gian Paolo Bezante, M.D.; and Richard S. Meltzer, M.D., Ph.D.

Acute rupture of the interventricular septum is a relatively unusual complication following acute myocardial infarction. The echocardiographic features depicted by transthoracic echocardiography are well described. However, transesophageal echocardiographic description of a ruptured septum has not been previously reported. This brief report illustrates the transesophageal features of such a defect.

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Ventricular septal rupture during the course of acute myocardial infarction is relatively rare, occurring in fewer than 1 percent of patients. The diagnosis should be considered with the appearance of a new, harsh, loud pansystolic murmur in the clinical setting of hypoperfusion and shock accompanied by signs of congestive heart failure or frank pulmonary edema. This usually happens several days after a large myocardial infarction. Noninvasive diagnosis is made by two-dimensional echocardiography with Doppler ultrasound, demonstrating echo “dropout” along the septum at the site of the tear and/or Doppler evidence of a left-to-right shunt at the site of the rupture. Although transesophageal echocardiography of congenital ventricular septal defects and free cardiac ruptures after cardiac surgery have been described, postinfarct ventricular septal defect (VSD) has not been explicitly described with this technique, although one report mentions a single such case in a series of 30 critically ill patients.

Case Report

A 79-year-old man with a history of hypertension was transferred to our hospital on the fourth day following an acute inferior myocardial infarction. He was considered for possible permanent pacemaker implantation due to late advanced atrioventricular block.

Physical examination on admission revealed hypotension and fulminating pulmonary edema. A harsh grade 4/6 pansystolic murmur was evident along the entire precordium.

The ECG was consistent with a Q-wave inferior myocardial infarction. Complete heart block and a ventricular rate of 56 beats per minute were recorded.

An urgent bedside two-dimensional transthoracic echocardiogram revealed akinesis of the posterior wall and dyskinesis of the inferior wall with a large posteroseptal aneurysm that encompassed the entire inferior septum. A large (1.2 x 1.0 cm) defect was visible in the midportion of the inferior septum adjacent to the site of the aneurysm (Fig 1). A prominent left-to-right shunt was observed with both color and spectral Doppler echocardiography. The right-sided chambers were enlarged, and the right ventricle was severely hypokinetic. The estimated pulmonary artery pressure, calculated from the peak tricuspid systolic gradient, was 62 mm Hg. Severe mitral regurgitation was noted with a structurally normal mitral valve.

The patient was stabilized medically, and an intra-aortic balloon and a temporary atrioventricular sequential pacemaker were inserted. Coronary angiography revealed total obstruction of the midportion of the right coronary artery. The left coronary system was normal. The patient’s condition initially improved with medical therapy, and he was operated on 72 h after transfer.

At surgery the right atrium was dilated. The right ventricle was dilated and hypokinetic. Right ventricular infarction was noted, as well as recent infarction of the inferior left ventricular wall.

Transesophageal echocardiographic monitoring during the operation revealed dyskinesis of the inferior septal wall, akinesis of the posterior wall, and lateral wall hypokinesis. A perforation was noted

Figure 1. Parasternal short-axis transthoracic echocardiographic view. Arrow indicates VSD in the inferior septum, next to the aneurysmal region. LV = left ventricle; RV = right ventricle.

Figure 2. Transgastric short-axis transesophageal echocardiographic view. Arrow indicates irregular serpiginous defect in the inferior septum, posterior to the posteromedial papillary muscle.

*From the Cardiology Unit, Department of Anesthesiology (Dr. Shah), and the Center for Biomedical Ultrasound (Drs. Harpaz, Bezante, and Meltzer), University of Rochester Medical Center, Rochester, NY.

Reprint requests: Dr. Meltzer, Cardiology, Box 679, University of Rochester Medical Center, Rochester, NY 14642-9679

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in the inferior septum at the level of the midportion of the papillary muscle; the perforation was irregular and serpiginous (Fig 2). The site of the rupture was 0.5 cm posterior to the insertion of the posteromedial papillary muscle. Color flow Doppler (Fig 3) demonstrated three mosaic turbulent signals in the right ventricle, originating in the left ventricle and visibly moving through the ruptured ventricular septum. Moderately severe mitral and severe tricuspid regurgitation were also noted.

A patch of glutaraldehyde-treated pericardium was used to close the septal defect. A second pericardial patch was used to close the inferior left ventriculotomy site.

Intraoperative images obtained after cardiopulmonary bypass showed a minimal leak through the septal defect repair greatly reduced in extent since the bypass.

The postoperative course was complicated by multiorgan failure, and the patient died 4 days after the operation.

Discussion

The combination of a first transmural infarction in a territory supplied by a single diseased coronary vessel that has no septal collaterals is found in a high proportion of cases of acute rupture of the interventricular septum as a complication of acute myocardial infarction. The septal rupture may occur at the center of a septal aneurysm. The perforation is single in most cases, and its size varies from one to several centimeters. During systole, the septal aneurysm might bulge toward the right ventricle, causing the defect to widen. Others have demonstrated bulging of a large septal aneurysm adjacent to the site of the tear (“ventricular aneurysm septal tear”), as was the case in this patient. The perforation may be a “through and through” hole or irregular and serpiginous, as in our case.

Defects with a serpentine course through the septum or with ragged edges are more difficult to visualize by echocardiographic imaging. Reasons for nonvisualization of such defects are multiple small defects and prominent right ventricular trabeculation.

These limitations might be overcome by detection of a right-to-left shunt by contrast echocardiography or a left-to-right shunt by Doppler or intraoperative transesophageal echocardiography. Systolic flow acceleration on the left side of the defect is usually obtainable by pulsed and color Doppler. The right ventricular pressure can be estimated from the difference between the arterial pressure obtained by the cuff method and the peak transventricular systolic gradient obtained by continuous-wave Doppler.

Color flow imaging is able to demonstrate the site of the defect, depicting a left-to-right mosaic turbulent signal with the most prominent portion in the right ventricle. These signals are highly sensitive and specific.

There is an advantage of the increased resolution and improved images from transesophageal echocardiography over transthoracic echocardiography when searching for specific information related to the site of the septal rupture. The involvement of the papillary muscle and submural apparatus can be readily assessed, allowing the surgeon to better estimate the need for concomitant mitral surgery before institution of cardiopulmonary bypass. Further, in some cases transthoracic echo allows neither direct visualization of the defect nor adequate Doppler examination. In such cases, transesophageal echocardiography provides an additional acoustic window. Intraoperatively, it provides a tool to detect early leakage, although it should be noted that a slight residual left-to-right shunt is frequently seen and is not a cause for alarm. Transesophageal echocardiography can be safely performed in critically ill patients, which is usually the case in patients with acute septal rupture.

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References


