

Transatrial Repair of Ventricular Septal Rupture under Preoperative Localization by Transesophageal Echocardiography

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We report about a 71-year-old woman with postinfarction ventricular septal rupture who was successfully treated by transatrial closure under preoperative localization by transesophageal echocardiography. In an attempt at transatrial repair of the ventricular septal rupture, the most important thing is preoperative localization of the defect in the septum, which is located high and posterior, where it is smooth with relatively few trabeculations and can be readily exposed by retraction of the tricuspid valve. (Ann Thorac Cardiovasc Surg 2001; 7: 180–2)

Key words: postinfarction ventricular septal rupture, transatrial repair, transesophageal echocardiography, preoperative localization of the defect

Introduction

Postinfarction ventricular septal rupture (VSR) is an uncommon yet serious complication of acute myocardial infarction. This is also often fatal unless surgically treated. The standard approach for closure of postinfarction VSR is ventriculotomy through the infarct zone. Repair of the defect in an inferobasal position through the zone of infarction can be technically difficult, requiring reconstruction of the posterior ventricular wall with prosthetic material. Some investigators have reported the transatrial approach for septal repair offers a safe and reliable repair of postinfarct VSR in selected cases.¹⁻⁸⁾ The advantages of this approach is avoiding the need for ventriculotomy with the attendant risks of myocardial damage, hemorrhage and ventricular arrhythmias. In an attempt at transatrial repair of the VSR, the most important thing is preoperative localization of VSR. The following case report describes a patient with preoperative localization of the VSR by transesophageal echocardiography who

underwent a successful operation for the VSR through a right atrial approach.

Case Report

A 71-year-old woman with rheumatoid arthritis under steroidal and immunosuppressive drug therapy was admitted with acute inferior myocardial infarction to a local hospital. Medical therapy consisted of diltiazem, nitroglycerin and aspirin in addition to her other rheumatoid arthritis medications. She was stable until eight days after myocardial infarction, when a pansystolic murmur at the left sternal border and pulmonary edema developed. She was transferred to our hospital for treatment of refractory congestive heart failure. She was classified as New York Heart Association Functional Class IV and controlled on a regimen of dopamin, norepinephrine, nitroglycerin, nicardipine and furosemide, administered intravenously. Chest X-ray showed an increase in pulmonary markings and enlargement of the heart silhouette with a cardio-thoracic ratio of 70%. A transthoracic echocardiography with colour flow mapping showed left-to-right shunt and left-sided overload. With stabilization of the vital signs, cardiac catheterization was undertaken; it demonstrated two-vessel disease with complete occlusion of the right coronary artery and high-grade stenosis of the circumflex artery. Pulmonary artery pressures were 33/13 mmHg. The pulmonary-to-systemic flow ratio (Qp/

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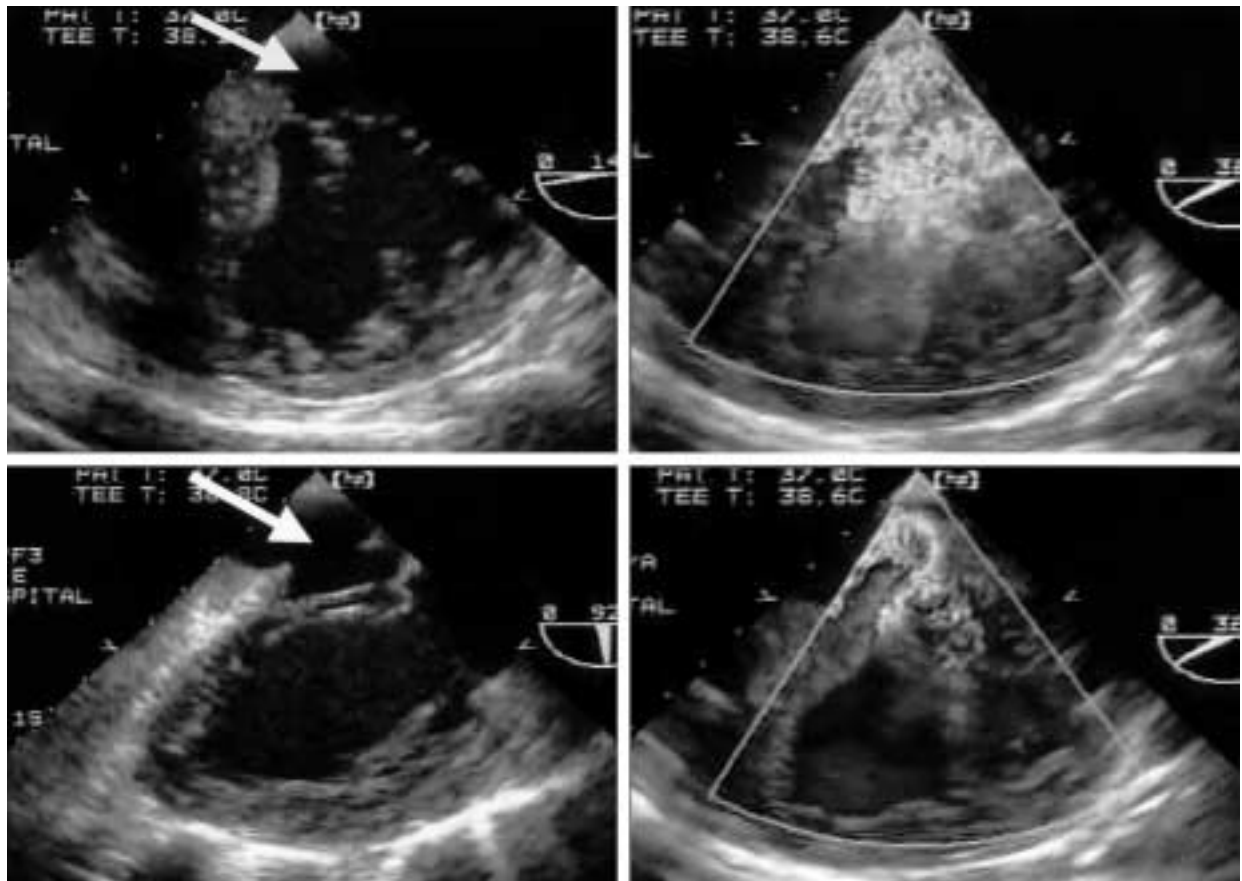


Fig. Intraoperative transesophageal echocardiogram: 2-dimensional image demonstrates an area of discontinuity in the ventricular septum (maximal width 20 mm).

a: Short axis view, c: long axis view. Color Doppler image demonstrates mosaic color signals indicative of turbulent blood flow moving from the left ventricle through the posterior ventricular septum into the right ventricle. b: Short axis view, d: long axis view. Arrows indicate the discontinuity in the ventricular septum.

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|---|---|
| a | b |
| c | d |

Qs) was 4.2:1. Without an intraaortic balloon pump, her hemodynamic condition was maintained. Thus, nineteen days after infarction, we performed operative closure of the VSR. Intraoperative transesophageal echocardiography showed that the VSR was located posteriorly, beneath the septal leaflet of the tricuspid valve (Fig.). Because the VSR was located high and posterior in the septum, we decided to undertake an attempt at transatrial repair of the VSR. In the operation, a median sternotomy was performed. The right ventricle was found to be grossly dilated and there was a small infarct of the inferior aspect of the left ventricle, but no aneurysmal change. Cardiopulmonary bypass with separate caval cannulation and a left ventricular vent placed via the right superior pulmonary vein was instituted, and hypothermia of 28°C was induced. After antegrade and retrograde blood cardioplegic arrest was obtained, exposure was obtained via the right atrium, with retraction of the tricuspid valve. The VSR could be seen through the tricuspid valve in-

sinuating under its septal leaflet in a posterior basal location, quite the same as preoperative transesophageal echocardiographic findings. The VSR was approximately 20 mm in diameter with an irregular border. The VSR was closed with a bovine pericardial patch. The patch was placed on the right side of the septum by taking interrupted mattress sutures of 3-0 Teflon-butressed Ticron first from within the defect toward the right ventricle and then through the patch. All sutures in the septum were brought out as far away from the defect as possible to tie over viable muscle. A saphenous vein bypass of the posterolateral branch was done. There were no difficulties in weaning the patient from cardiopulmonary bypass. Her postoperative recovery was uneventful. At twenty-seven days after surgery, cardiac catheterization showed normal pulmonary and right-sided pressure and normal LV end-diastolic pressure. The coronary angiogram revealed no stenosis or occlusion of the saphenous vein graft. The ventriculogram showed aneurysmal change in the infe-

rior basal, but no residual left-to-right shunt. Nine months postoperatively, she is alive and well. We are carefully following her conditions by echocardiography, but there are no changes in size and shape of the aneurysm.

Comment

Ventricular septal rupture is a rare complication of acute myocardial infarction and is usually fatal if not surgically repaired. The standard operative techniques are infarctectomy and reconstruction of the ventricular septum and free walls with patches or infarct exclusion of the left ventricle by an endocardial patch through a left ventriculotomy or through the infarcted free wall where possible. The disadvantages of this approach is the attendant risks of myocardial damage, hemorrhage and ventricular arrhythmias. When the VSR is associated with extensive inferior infarction, exposure and repair of defects in this position through the standard approach are technically very difficult. Since the first successful transatrial repair of postinfarction VSR by Filgueira et al.,¹⁾ some surgeons have described the operative technique and reliability, in delayed and acute settings, of this approach.^{2,5-8)} In selected cases, this approach can offer a safe and reliable repair of postinfarction VSR, however, there are potential problems with this approach. The dense and irregular trabeculations of the right ventricle can make the exposure and exact identification of the VSR difficult. The patch repair on the right side of the septum may predispose the septum to residual or recurrent defect because of the left-to-right pressure gradient during systole. The placement of sutures through the tricuspid valve may make tricuspid valve damage. However, since some investigators have described the technical solutions to these problems,^{2,7,8)} the most important thing is the location of the VSR.^{4,7)} If the VSR is located high and posterior, where it is smooth with relatively few trabeculations, it can be easy to expose and exactly visualize the VSR and the transatrial repair can offer the potential for successful closure and avoidance of residual defect. However, to make a decision of the indication in an attempt at transatrial repair of the VSR preoperatively, the most important thing is preoperative exact localization of the VSR. In most cases, the patient is critically ill. The left ventriculogram is not good for preoperative localization of the VSR, because it may acutely depress ventricular function. Two-dimensional and color doppler surface echocardiography are non-invasive procedures and are sensitive for preoperative lo-

calization of the VSR. But their main limitation is the presence of small acoustic windows in some patients, especially critically ill patients. The technique of transesophageal echocardiography has circumvented this limitation to a large extent. Although transesophageal echocardiography is considered a semi-invasive procedure, it is safe even in seriously ill patients after anesthesia. It has the advantage of high resolution imaging, and the use of a biplane technique allows evaluation of the entire ventricular septum. We believe that the best examination for preoperative localization of the VSR is the transesophageal echocardiography.

In conclusion, the right atrial approach to the VSR is an alternative technique of septal repair in selected cases. If the accurate preoperative localization of the VSR, which is located high and posterior in the septum, has been made by transesophageal echocardiography, safe and reliable transatrial repair of the VSR may be applicable.

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