

Post-Infarction Ventricular Septal Defect Treated During the Acute Phase by Transcatheter Closure With an Amplatzer Septal Occluder

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A 75-year-old man had a large anterior myocardial infarction complicated by a ventricular septal defect, which was treated in the first 48 h by transcatheter closure using the Amplatzer septal occluder. Treatment was successful (with only mild residual post-procedure shunting) and coronary angioplasty with stent implantation at the point of occlusion of the middle left anterior descending artery was performed in a later intervention. However, the patient died 7 days after the procedure as a result of sepsis and ventricular failure.

Key words: *Ventricular septal defect. Myocardial infarction. Coronary angioplasty.*

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Comunicación interventricular postinfarto de miocardio tratada en fase aguda mediante cierre percutáneo con el dispositivo Amplatzer

Presentamos el caso de un paciente de 75 años afectado de un infarto anterior extenso complicado con una comunicación interventricular muscular apical que se trató mediante la implantación percutánea de una prótesis de tipo Amplatzer dentro de las primeras 48 h de evolución del infarto. Con este tratamiento se consiguió cerrar de forma eficaz el defecto interventricular (ligero shunt residual posprocedimiento), y posteriormente se practicó con éxito una angioplastia con implantación de stent en una oclusión de la descendente anterior media. Sin embargo, el paciente falleció 7 días después del procedimiento por sepsis y fallo ventricular.

Palabras clave: *Comunicación interventricular. Infarto de miocardio. Angioplastia coronaria.*

INTRODUCTION

Ventricular septal defects (VSD) are a rare complication (1%-2%) in acute myocardial infarction and are associated with mortality rates of more than 80% with medical treatment. Surgery is the standard procedure to treat this condition, but when it has to be performed during the acute stage (<2 weeks), the mortality rate is very high (30%-50%), and the incidence of significant residual defects is more than 40%.¹

Recently, the transcatheter closure of congenital muscular VSD using Amplatzer devices (AGA, Medical Corporation, MN, USA) has shown promising results.² We report a case of successful transcatheter closure of a post-infarction VSD during the acute stage, although the patient died subsequently from sepsis and ventricular failure.

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CLINICAL CASE

A 75-year-old patient was admitted to another center due to chest pain that lasted more than 12 h. He was diagnosed with extensive anterior myocardial infarction and therefore did not receive fibrinolytic treatment. On the second day, a systolic murmur was detected and Doppler echocardiography revealed an apical muscular VSD (Figure 1A), which motivated transfer to our center. Upon arrival, the patient was in a state of cardiogenic shock, and given the high risk associated with surgery—i.e., an elderly patient in a state of shock—we opted for the alternative transcatheter approach. After obtaining informed consent from the patient, we performed the procedure (<12 h after diagnosing VSD) via the right internal jugular and femoral artery. The process was monitored with transthoracic Doppler echocardiography. The patient received 100 U/kg heparin and a dose of cefuroxime (750 mg). Initially, a counterpulsation balloon was implanted via the left femoral artery. A contrast left ventriculography showed an apical muscular ventricular septal defect 15 mm in diameter (Figure 2A). The VSD was

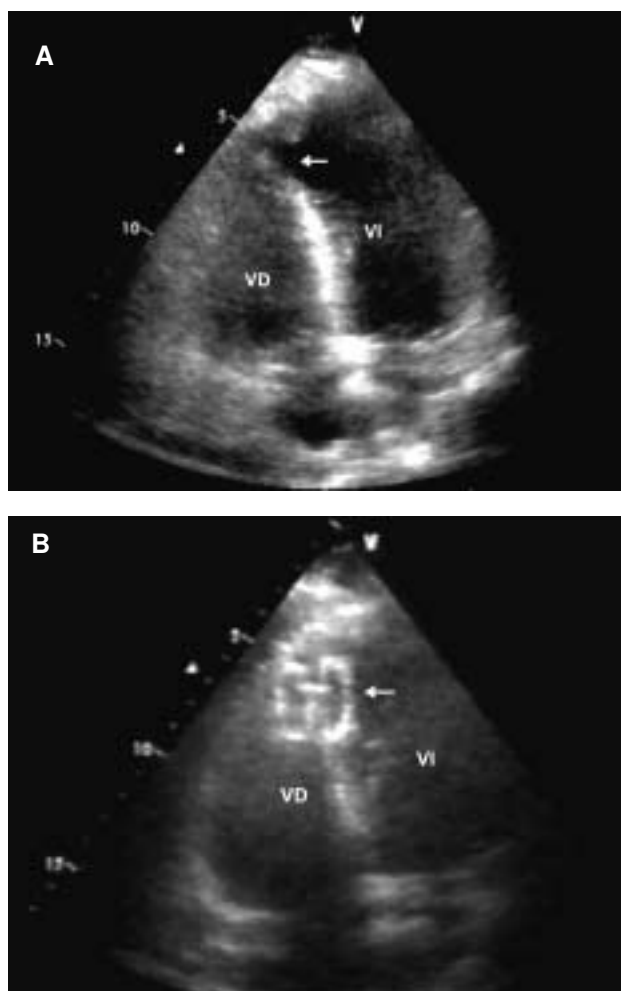
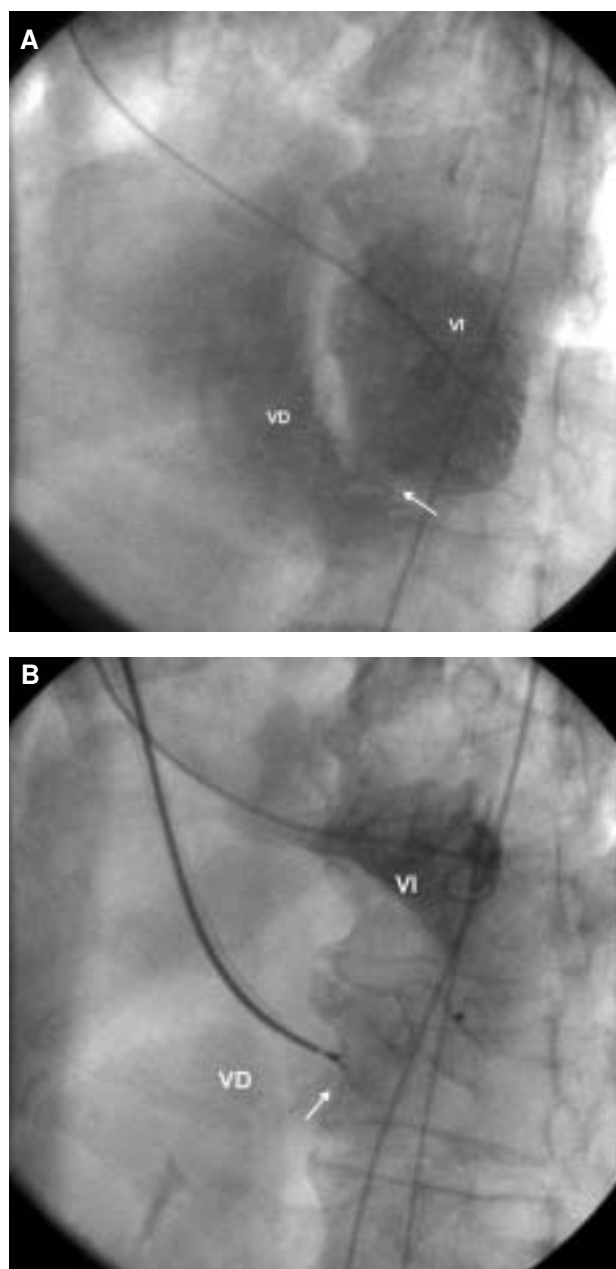


Fig. 1. A: ultrasound image of apical muscular ventricular septal defect (VSD) (arrow). B: ultrasound image of Amplatzer device (arrow) implanted in the VSD. RV indicates right ventricle; LV, left ventricle

crossed from the LV using a guide, to create an arteriovenous loop as described in previous publications.² From the vein end of the guide we introduced into the left ventricle a Mullins-type 10 Fr catheter. Then an Amplatzer 20-mm septal occluder was advanced through it. This device is made up of a fine mesh of nitinol with dacron patches, and consists of two discs (left and right) linked by a 4-mm waist. The left disc was expanded first, and after checking by echocardiography that its placement in the ventricular septum was correct, the central stent and the right disc were expanded. Subsequent echocardiography and contrast ventriculography showed their correct placement and a very slight residual shunt between the lower edge of the device and the apex of the ventricle (Figure 2B), and so the device was finally deployed (Figures 1B and 2C). Later, angioplasty with a stent for an occlusion in the middle left



anterior descending artery was performed. In the following days the patient showed hemodynamic improvement and the echocardiograms confirmed the correct placement of the device, with a slight residual shunt between its lower end and the ventricular apex. The ejection fraction was 35%, and the value of Qp/Qs obtained by oximetry was 1.2. Four days after the procedure, the patient developed sepsis of unknown origin—blood culture yielded negative results—which proved impossible to control with the use of wide-spectrum antibiotics. The patient died 10 days after the infarction from septic shock and ventricular failure.

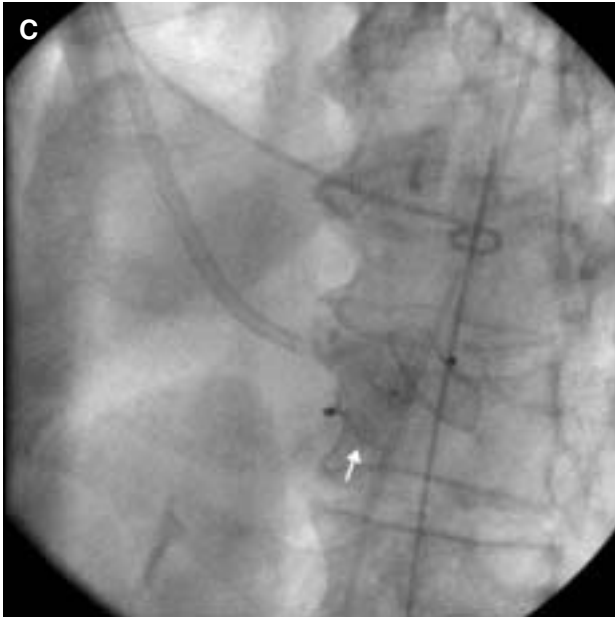


Fig. 2. A: contrast left ventriculography reveals an apical ventricular septal defect (VSD) of 15 mm diameter (arrow). B: amplatzer device (20 mm) implanted in the VSD (arrow), just before deployment. Contrast ventriculography shows a slight residual shunt mainly within the device. C: x-ray of the Amplatzer device after being deployed. Note the two discs (left and right) linked by a connecting waist placed in the VSD. RV indicates right ventricle; LV, left ventricle.

DISCUSSION

Experience in the treatment of VSD that arises as a complication of myocardial infarction is limited.³⁻⁹ In most published cases, a VSD occluder was successfully implanted, but variations have been reported regarding residual shunt. In the study with the largest sample available (12 patients), an Amplatzer device was successfully implanted in 83% of the patients. The incidence of residual shunt was higher than 30% and hospital mortality reached 25%.⁹ In fact, in those cases that reported the time span between the onset of complications and transcatheter closure, survival was restricted to either patients treated in the subacute stage (>15 days postinfarction) or to those whose VSD was residual after surgical repair.³⁻⁸ The high rate of early mortality has been attributed to secondary ventricular failure from extensive infarction, or associated co-morbidity, as in the septic process that occurred in the patient we present.

For logistical reasons, the Amplatzer device implanted in our patient was designed for the closure of atrial septal defects. An Amplatzer occluder specially designed for VSD would probably have been more suitable, because it has a longer connecting waist (7 mm), such that it would have provided a better fit to the ventricular septum.² In any case, we think that the use of an oversized device to support the discs on healthy myocardial tissue (as done in our case) is a key factor to reduce residual shunts and prevent possible embolism caused by the device.

In conclusion, postinfarction VSD can be treated with a transcatheter closure procedure. Although surgical intervention is still the treatment of choice for this kind of complication, the transcatheter approach could be considered an alternative in high-risk patients.

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