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Transcatheter Closure of Traumatic Ventricular Septal Defect: An Alternative to Surgical Repair?

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A 19-year-old man with multiple-system injuries including a serious head injury and two poorly tolerated traumatic ventricular septal defects, was admitted to our hospital. Transcatheter closure of the cardiac defects was attempted instead of surgical repair because the required anticoagulation for cardiopulmonary bypass could precipitate intracranial bleeding. The two ventricular septal defects were successfully closed with Amplatzer devices, but the patient remained in hemodynamically unstable condition and subsequently died. Transcatheter closure of traumatic ventricular septal defect is an alternative to surgical repair, although it remains a hazardous procedure and requires experienced anesthesia management.

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Traumatic ventricular septal defect (VSD) is a rare complication of cardiac trauma and may require surgical repair without delay in the case of poor hemodynamic tolerance [1, 2]. However, in patients with multiple-system injuries, cardiopulmonary bypass with anticoagulation may be contraindicated when there is a head injury with intracranial bleeding. We report transcatheter closure of traumatic VSDs in a patient who sustained a serious head injury.

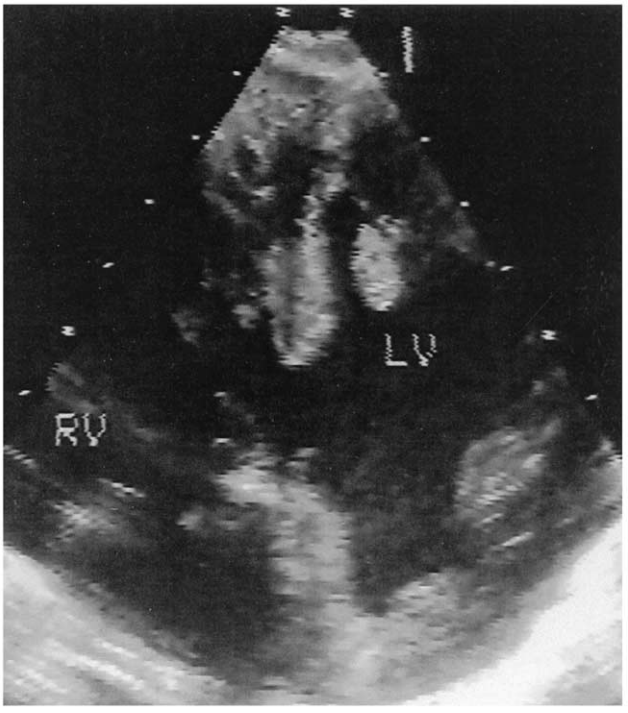

*Fig 1. Transesophageal echocardiographic view of large midmuscular ventricular septal defect. (*LV = *left ventricle;* RV = *right ventricle.)*

A 19-year-old man was admitted to the intensive care unit after a motorcycle crash in which he was not wearing a helmet. He lost consciousness (initial score on the Glasgow coma scale, 7) and was intubated. He had multiple injuries, including head trauma and crush injuries to the anterior chest wall, trunk and legs. Radiography of the skull, head, and chest revealed a fracture of the right clavicle. There was also a shaft fracture of the left leg. Head tomodensitometry showed diffuse cerebral contusions with meningeal bleeding.

After the initial evaluation, the patient became hypotensive, and auscultation of the chest revealed a loud systolic murmur at the left sternal edge. Transesophageal echocardiography demonstrated a large mid muscular VSD (Fig 1). Systemic arterial systolic blood pressure was 80 mm Hg, whereas pulmonary arterial systolic pressure was 40 to 45 mm Hg through a Swan-Ganz catheter. An important left-to-right shunt was documented, with an oxygen saturation step-up of 31% from the superior vena cava to the pulmonary artery (65% to 96%). Inotropic support (dopamine hydrochloride, 10 $\mu g \cdot kg^{-1} \cdot min^{-1}$) and an attempt to increase pulmonary vascular resistance by lowering the inspired oxygen fraction associated with mild hypoventilation failed to reduce the left-to-right shunt. An intraaortic balloon pump was placed without further hemodynamic stabilization.

Surgical repair of the VSD under cardiopulmonary

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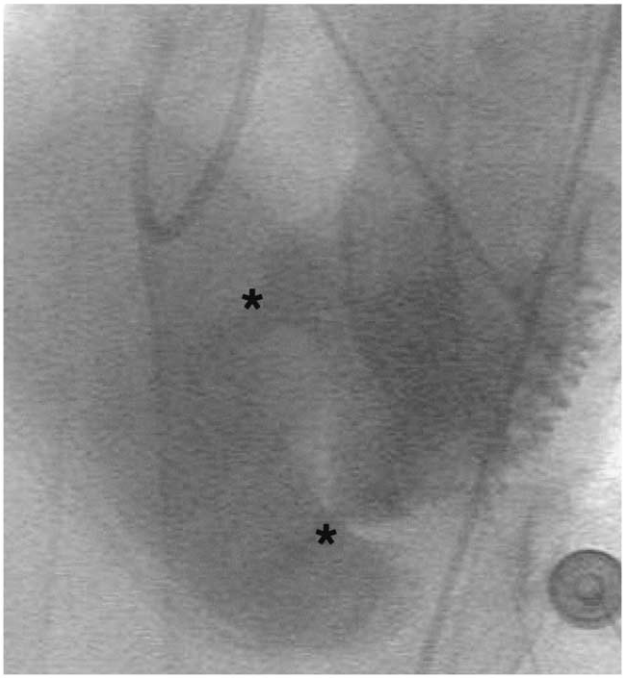

Fig 2. Long-axial oblique left ventriculogram showing two ventricular septal defects (*) in midmuscular and apical positions.

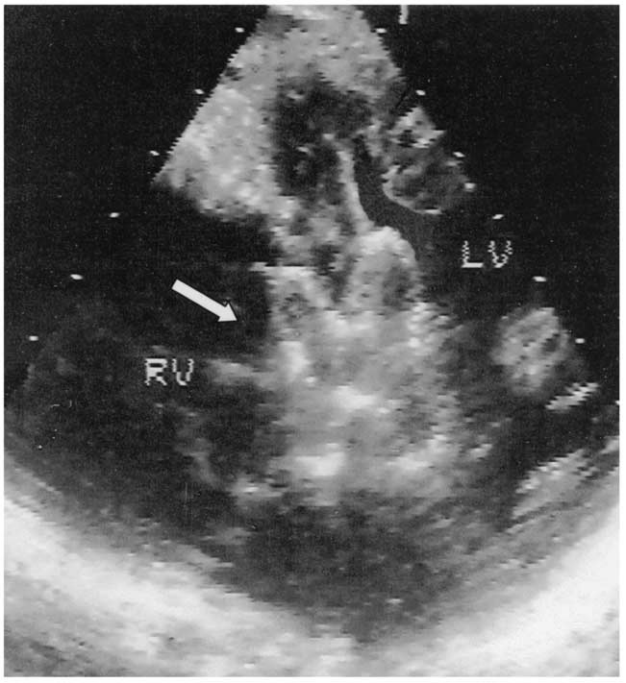

Fig 3. Transesophageal echocardiographic view of midmuscular ventricular septal defect closed with a 22-mm Amplatzer atrial septal defect occluder (arrow). (LV = *left ventricle;* RV = *right ventricle.)*

bypass was contraindicated because the required anticoagulation could precipitate meningeal and brain bleeding. It was decided to attempt transcatheter closure of the VSD in the catheterization laboratory. A long-axial oblique left ventriculogram showed a large midmuscular VSD and an additional smaller apical VSD that was not previously recognized by transesophageal echocardiography (Fig 2). Under fluoroscopic and transesophageal echocardiographic guidance, the two VSDs were closed with two Amplatzer devices (atrial septal defect occluder, 24 mm and 22 mm) (Figs 3 and 4). After release of the second device to close the midmuscular VSD, a complete atrioventricular block occurred and required temporary transvenous pacing. The patient recovered sinus rhythm, and the cavopulmonary difference in oxygen saturation fell to 10% after the procedure. However, the condition of the patient did not improve, and severe hypotension with multiorgan failure developed. He died 3 days after admission.

Comment

A traumatic VSD occurs either because of heart compression between the sternum and the spine or because of injured coronary arteries with myocardial infarction [1, 2]. Ideally, surgical repair is performed after a period of several days or weeks of stabilization [1]. In the case of poor hemodynamic tolerance, use of an intraaortic balloon pump associated with ventilatory manipulation of the pulmonary vascular resistance to reduce the left-to-right shunt may acutely stabilize the condition of the patient. When such supportive measures fail to improve the hemodynamic status, surgical repair under cardiopulmonary bypass is required, although the necessary anticoagulation may precipitate bleeding from other injuries [2].

Transcatheter closure of a VSD was first reported by Lock and in associates [3] in 1988. More recently, successful closure of VSDs have been accomplished with a device designed specifically for this purpose: the Amplatzer VSD occluder. However, the device is not suitable

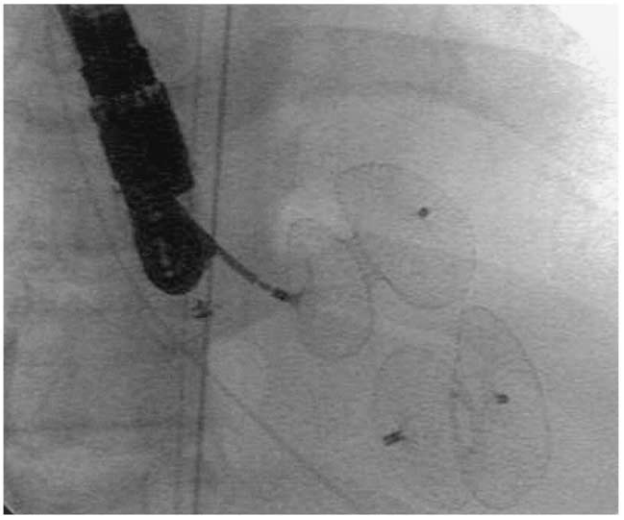

Fig 4. Fluoroscopic image of two Amplatzer atrial septal defect occluders (22 and 24 mm) positioned through ventricular septal defects.

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for VSDs larger than 12 mm [4]. In our patient, the size of the midmuscular VSD, measured by transesophageal echocardiography, was 18 to 20 mm. It was necessary to use a 22-mm atrial septal defect occluder to close it. The smaller apical VSD was closed with a 24-mm atrial septal defect occluder, as the position of the device was optimal.

The technique of transcatheter closure of a VSD has been detailed previously [3, 4]. Briefly, a retrograde arterial catheter is passed from the left ventricle to the right ventricle. An exchange guidewire is advanced through this arterial catheter into the pulmonary artery and is snared from a percutaneous jugular or femoral vein approach. Then, the catheter is removed, and a long transseptal sheath is advanced over the wire from the jugular or femoral vein to the left ventricle. After transesophageal or balloon sizing or both of the VSD, the correct device size is selected (equal to the measured diameter) and screwed to the tip of a delivery cable. After being collapsed into a loader, the device is advanced by pushing the delivery cable into the sheath and deployed under transesophageal and fluoroscopic guidance in the left ventricle. The device is released when its position is optimal and interference with the valvar structure is excluded.

The procedure requires initial heparinization (100 U/kg), and it can be associated with hemodynamic instability, although the success of the procedure and the outcome are usually satisfactory. In a retrospective review of 70 VSD closures, hypotension occurred in 40% and dysrhythmias in 28.5% [5]. General anesthesia with endotracheal intubation is required, and the procedure is guided by transesophageal echocardiography. Our patient was in hemodynamic unstable condition before the procedure. His status was further compromised by a transient complete atrioventricular block after device closure of the midmuscular VSD.

In conclusion, even though our patient died, we believe that transcatheter closure of a traumatic VSD is an alternative to surgical repair. However, it is accomplished using a prolonged procedure with potential hemodynamic instability, and it requires experienced anesthesia management.

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Echocardiographic Evidence of Right Ventricular Remodeling After Transplantation

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Right ventricular (RV) failure is a significant source of mortality after cardiac transplantation. The use of RV assist devices (RVAD) as a bridge to recovery has been reported. However, early changes of RV structure and anatomy after RVAD implantation have yet to be described. We report a case of RV failure after transplantation requiring RVAD implantation. After 3 weeks of gradual weaning of inotropic and RVAD support, the device was explanted successfully. Transesophageal echocardiography documents RV hypertrophy and remodeling between RVAD implantation and removal, suggesting a rapid adaptive response of the right ventricle in the presence of pulmonary hypertension.

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Right ventricular (RV) failure remains a leading cause of early death after orthotopic heart transplantation (OHT). Usually RV failure is related to persistent recipient pulmonary hypertension associated with a history of chronic congestive heart failure. Failure of the donor's right ventricle to adapt to increased pulmonary vascular resistance (PVR) leads to hemodynamic collapse. Strategies to improve ventricular mechanics include reducing PVR with vasodilators and increasing RV contractility with inotropic agents [1, 2]. Use of a right ventricular assist device (RVAD) is indicated when refractory RV failure is present despite maximal pharmacological therapy. RVAD support is effective as a bridge to RV recovery, particularly if implanted before end-organ injury becomes irreversible [3]. However, echocardiographic evidence of adaptive changes of the right ventricle to elevated PVR after RVAD implantation has not been described previously. We present a patient in whom transesophageal echocardiography demonstrated ana-

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