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Case report / Olgu Sunumu

Transcatheter closure of post-myocardial infarction ventricular septal defect with the amplatzer septal occluder

Myokard infarktüsü sonrası gelişen ventriküler septal defektin amplatzer septal okluder ile transkateter yoldan kapatılması

Abdullah Erdem,[1] Celal Akdeniz,[1] Cenap Zeybek,[1] Numan Ali Aydemir,[2] Ahmet Çelebi[1]

[1]Department of Pediatric Cardiology, Dr. Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital, İstanbul, Turkey;
[2]Department of Cardiovascular Surgery, Dr. Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital, İstanbul, Turkey

The ventricular septal defect (VSD) is an uncommon complication of post-myocardial infarction (MI) in 0.2% of the patients. Medical therapy alone results in a considerably poor prognosis with a one year survival of 3-7%. Until recently, urgent or early surgical defect closure has remained the only therapeutic option. However, surgical defect closure is associated with high mortality rate and residual shunt in up to 20%. The Amplatzer devices provide a novel therapeutic option for transcatheter treatment for this complication. There is a limited data on transcatheter closure of post-MI VSD. In this article, we present a 55-year-old male case with post-MI VSD which was closed by Amplatzer septal occluder designed to close secundum atrial septal defects.

Key words: Amplatzer; myocardial infarction; ventricular septal defect.

Ventriküler septal defekt (VSD) miyokard enfarktüsü (Mİ) sonrası hastaların %0.2'sinde gelişen nadir bir komplikasyondur. Yalnızca medikal tedavi ile takip edildiklerinde prognoz oldukça kötü olup, bir yıllık sağkalım oranı %3-7 arasındadır. Son yıllara kadar defektin cerrahi olarak acil veya erken dönemde kapatılması tek tedavi seçeneği idi. Ancak bu defektlerin cerrahi kapatılması yüksek ameliyat mortalitesi ve %20'ye varan rezidü şant oranlarıyla birliktelik gösterir. Amplatzer tipi cihazlar, bu komplikasyonun transkateter tedavisi için yeni bir seçenek oluşturmaktadır. Miyokard enfarktüsü sonrası gelişen VSD'lerin transkateter yoldan kapatılması ile ilgili veriler henüz yetersizdir. Bu yazıda Mİ sonrası gelişen VSD'nin sekundum atriyal septal defektleri kapatmak için geliştirilmiş Amplatzer septal okluder ile kapatıldığı 55 yaşında erkek bir olgu sunuldu.

Anahtar sözcükler: Amplatzer; miyokard enfarktüs; ventriküler septal defekt.

A ventricular septal defect (VSD) is a rare but lethal complication of a myocardial infarction (MI)[1,2] that occurs between two and 14 days after the infarction and often precipitates cardiogenic shock. The prognosis for patients with a VSD is extremely poor, with reports indicating only 3-7% survival rate after one year.[1-4] To decrease the high morbidity and mortality associated with this disorder, patients should undergo emergent surgery.[4-6]

Primary transcatheter closure of post-MI VSDs may be an alternative to surgery for patients with suitable anatomy of the defect,[2,7-9] and the Amplatzer devices provide new therapeutic options for transcatheter treatment of this complication.[9,10] In this report, we present post-MI VSD closure in a 55-year-old male patient using the Amplatzer septal occluder (ASO) (AGA Medical Corporation, Plymouth, Minnesota, USA), a device originally developed for transcatheter closure of secundum atrial septal defects (ASD).

CASE REPORT

A 55-year-old male patient was referred to our clinic for surgical treatment of post-MI VSD due to a large

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Correspondence: Abdullah Erdem, M.D. Dr. Siyami Ersek Göğüs, Kalp ve Damar Cerrahisi Eğitim ve Araştırma Hastanesi, Çocuk Kardiyolojisi Kliniği, 34668 Haydarpaşa, İstanbul, Turkey.
Tel: +90 505 - 770 58 05 e-mail: drabdullaherdem@hotmail.com

anterior MI. When he was transferred to the intensive care unit (ICU), he was in a comatose state and was on mechanical ventilatory support. He had suffered a previous ventricular fibrillation attack and had been defibrillated before he was transported. A coronary angiography performed before transfer revealed critical stenosis of the left anterior descending artery (LAD) and a totally occluded circumflex artery. At our clinic, percutaneous balloon angioplasty was unsuccessful, so he received thrombolytic treatment with streptokinase. On the fourth post-infarction day, he developed tachycardia, hypotension, oliguria, pulmonary edema, and a new pansystolic murmur; therefore, intravenous inotropes, and intra-aortic balloon counter pulsation (IABP) were initiated. Transthoracic echocardiography (TTE) showed anteroseptal akinesia and an apical 14x17 mm VSD (Figure 1). Transcatheter closure of the VSD was performed on the fifth day of infarction under TTE and fluoroscopy since it was thought that the risk of surgical mortality was high for this clinical situation.

Under general anesthesia with continuous TTE monitoring, right and left heart catheterization was performed via a 5 French (Fr) sheath in the right femoral artery and a 6 Fr sheath in the right femoral vein. In addition, an 8 Fr sheath was placed in the right internal jugular vein. A hemodynamic study revealed that the pulmonary artery pressure was mildly elevated with a main pulmonary artery pressure of 46/29 mmHg and a mean of 36 mmHg. An angiogram of the left ventricle (LV) in the hepatoclavicular projection [35º left anterior oblique (LAO), 35º cranial] revealed the presence of a large, muscular VSD in the anterior apical location. Transthoracic echocardiography and cardiac catheterization (Figure 2) showed a VSD of 20 mm in diameter with a 2.4:1 left-to-right shunt. The VSD was crossed from the LV side using a 5 Fr partly cut pigtail catheter. Then a hydrophilic 0.035-inch guide was advanced via the catheter and manipulated to the main pulmonary artery. After this was completed, the catheter was advanced over this wire to the main pulmonary artery. Next, the hydrophilic guidewire was exchanged for a 0.035-inch J-tipped Noodle guide wire (AGA Medical Corporation, Plymouth, Minnesota, USA). This was snared in the main pulmonary artery and exteriorized out the right internal jugular vein to form an arteriovenous wire loop. Over this wire, an 8 Fr long delivery sheath was advanced from the right internal jugular vein to the right ventricle (RV), the VSD, and then to the LV. Following this, a 22 mm ASO device was loaded and advanced from the internal jugular vein until it reached the tip of the sheath, It was then withdrawn into the cavity of the LV. Afterwards, the LV side of the device disc was deployed in the LV. Then the whole assembly (device, cable, and delivery sheath) was withdrawn until the disk was against the ventricular septum. Deployment of the RV side disc was also accomplished under TTE. A repeat LV angiogram and TTE confirmed the appropriate device position (Figure 3), and the device was detached by a counterclockwise rolling movement. The following

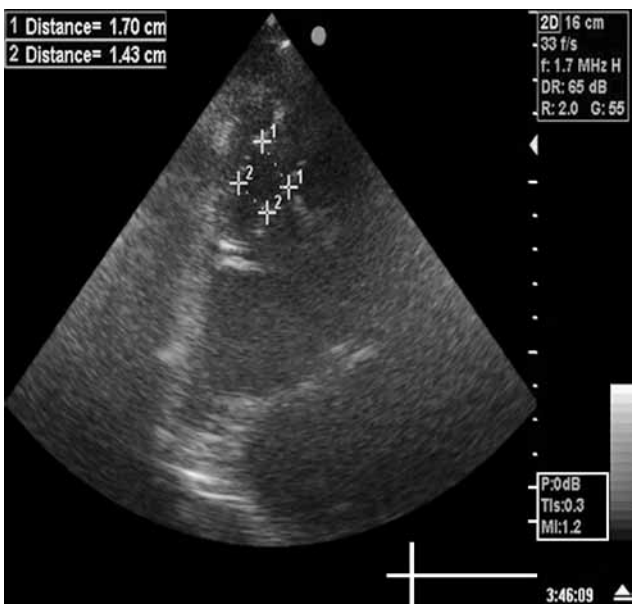

Figure 1. Transthoracic echocardiographic two-dimensional view of the post-infarction ventricular septal defect.

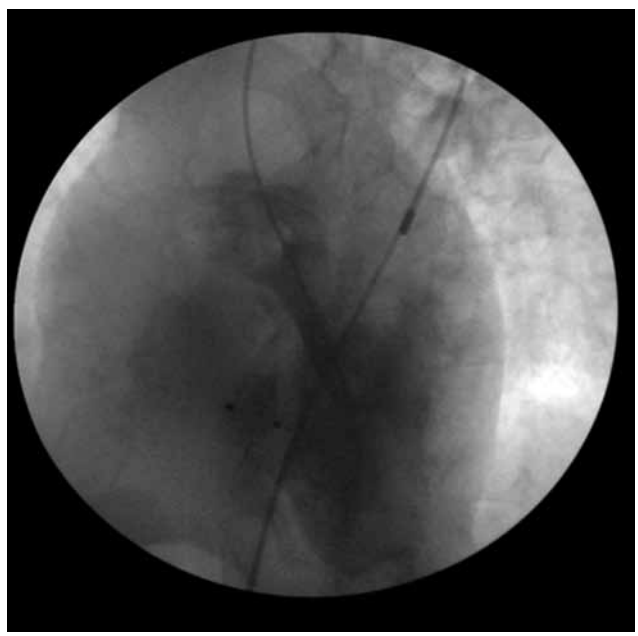

Figure 2. Left ventriculography after deployment of the device.

day, repeat TTE demonstrated that the device was in a good position with minimal residual shunt, but LV function was still poor with an LV end-diastolic dimension of 62 mm. The patient's clinical condition partially improved after the percutaneous procedure; however, it did not allow for him to be weaned from the IABP and inotropic support. Although the procedure was successfully performed and the volume overload was abolished, the depressed LV function led to impaired peripheral organ perfusion, and the patient eventually died seven days after the procedure.

DISCUSSION

Although surgical techniques, myocardial protection, and perioperative mechanical and pharmacological supportive measures have improved, surgery for post-MI VSD still yields 50% mortality and 20% postoperative residual shunt.[2,7]

Since surgical repair of a ruptured myocardium is challenging, percutaneous closure of the post-MI VSD presents an alternative form of treatment.[2] However, the percutaneous procedure also poses significant problems. First, it is extremely difficult to place a closure device on a fragile septum. Additionally, residual shunt is highly probable. Since the ASO is self-centering and has a waist part, it is easier to place on the ruptured septum with minimal or no residual shunt.[9,10] On the other hand, the post-MI VSD could be very large or multiple in nature and have the possibility of increasing in size over time. The defect edges are also very fragile. Theoretically in these cases even the oversized devices may not only embolize itself but also cause scar tissue embolization.

In the presented case, we hypothesized that the patient's clinical condition was critical and chose to perform an early surgical closure of the VSD. We accepted the risk of residual shunting and decided that even a partial closure of the defect would be of great benefit by reducing the left-to-right shunt. Indeed, the patient's clinical condition slightly improved after the percutaneous procedure but not enough for him to be weaned from IABP and inotropic support. Although the procedure was successfully performed and the volume overload was abolished, the depressed LV function led to impaired peripheral organ perfusion and the patient's death.

We believe that in selected patients with suitable anatomy of the defect, transcatheter closure with ASO devices could be beneficial for post-MI VSD. However, larger devices should probably be used to prevent residual shunting.

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