Transcatheter device closure of a residual postmyocardial infarction ventricular septal defect

Miyokart enfarktüsü sonrası gelişen ventriküler septal defektin transkateter yolla kapatılması

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Summary – Postmyocardial infarction ventricular septal defect (VSD) carries a high mortality and, even after successful surgery, residual defect is common. A 75-year-old woman was admitted with the diagnosis of hyperacute anterior myocardial infarction. Primary percutaneous intervention was performed by stenting of a totally obstructed segment in the proximal left anterior descending artery. The patient's condition deteriorated on the second postprocedural day with a 3/6 pansystolic murmur at the mesocardium. Echocardiography revealed an apical anteroseptal VSD and moderate pulmonary hypertension. She underwent surgical VSD closure with a Gore-Tex patch and coronary artery bypass grafting to the left anterior descending and circumflex arteries. The patient's condition continued to be unstable due to septicemia and hemodynamically significant residual VSD. After medical management of septicemia, the residual defect was successfully closed using a 10-mm Cardio-O-Fix septal occluder under fluoroscopic and transesophageal echocardiographic guidance. The clinical condition of the patient was then stabilized and there was no significant residual shunt on echocardiography on the third postprocedural day.

Özet – Miyokart enfarktüsü sonrası gelişen ventriküler septal defekt (VSD) mortalitesi yüksek olan bir hastalıktır ve başarılı cerrahi tedaviden sonra bile oldukça yüksek sıklıkta defektin devam ettiği görülür. Yetmiş beş yaşında bir kadın hasta, hiperakut anteriyor miyokart enfarktüsü tanısıyla hastanemize yatırıldı. Primer perkütan girişimle, sol ön inen arter proksimalinde görülen tam tıkalı segmente stent yerleştirildi. Girişimin ikinci gününde hastanın genel durumu bozuldu. Muayenede mezokardiyak odakta 3/6 dereceli pansistolik üfürüm duyuldu. Ekokardiyografide apikal anteroseptal VSD ve orta derecede pulmoner hipertansiyon saptandı. Defekt Gore-Tex yama kullanılarak cerrahi yolla tamir edildi ve aynı seansta sol ön inen arter ve sirkumfleks artere baypas greft uygulandı. Cerrahi sonrası gelişen septisemi ve hemodinamik olarak önemli VSD kalıntısı nedeniyle hastanın durumunda düzelme olmadı. Sepsisin medikal tedavisi sonrasında, VSD defekti 10 mm Cardio-O-Fix septal tıkayıcı ile floroskopi altında ve transözofageal ekokardiyografi eşliğinde başarıyla kapatıldı. Girişim sonrasında hastanın klinik durumu düzeldi ve girişimin üçüncü günü yapılan ekokardiyografide önemli şanta rastlanmadı.

Ventricular septal defect due to acute ventricular septal rupture following myocardial infarction carries a high mortality.[1,2] Surgical repair also presents a high mortality risk, but progressive deterioration in hemodynamic status makes surgical intervention often the only realistic option. On the other hand, despite successful surgery, residual VSD is common,[1,2] which may cause significant hemodynamic disturbance requiring reintervention.

Transcatheter closure is an established method of treating congenital VSDs,[3] but clinical experience in transcatheter closure of postmyocardial infarction VSDs is limited.[4-6] We report on successful transcatheter closure of a residual post-MI VSD that persisted after surgical patch closure.

Abbreviations:

Post-MI *Postmyocardial infarction*
VSD *Ventricular septal defect*

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CASE REPORT

A 75-year-old woman was admitted with anginal chest pain. Electrocardiography showed ST-segment elevation in all precordial leads. The patient was transferred to the catheterization laboratory immediately for primary percutaneous intervention with the diagnosis of hyperacute anterior myocardial infarction. Coronary angiography showed total obstruction in the proximal left anterior descending artery just after the first diagonal branch. The obstructed segment was stented by a Gazelle bare-metal stent, 3.25 mm x 18 mm in size. Postprocedural echocardiography at 24 hours revealed severe hypokinesia at apical, mid-anterior, and anteroseptal walls and the patient developed dyspnea, hypotension, and sinus tachycardia approximately at 36 hours. Auscultation revealed a 3/6 pansystolic murmur maximally heard at the mesocardium. Echocardiography was repeated and an apical anteroseptal VSD was visualized with a left-to-right intracardiac shunt. Estimated pulmonary artery systolic pressure using tricuspid regurgitation jet of moderate severity was 55 mmHg. Because of rapid deterioration in clinical condition, intra-aortic balloon pumping was initiated to stabilize hemodynamics of the patient.

She underwent surgical VSD closure with a Gore-Tex patch and coronary artery bypass grafting to the left anterior descending artery and circumflex artery on the fourth postprocedural day. Intraoperative transesophageal echocardiography showed no residual defect (Fig. 1a). However, the patient's condition remained labile despite intra-aortic balloon pumping and inotropic support. A residual VSD was suspected on transthoracic echocardiography on the second postoperative day. Transesophageal echocardiography confirmed the residual defect. The diameter was around 4 mm, and the pulmonary-to-aortic flow ratio was calculated as 1.5 (Fig. 1b). The patient's clinical condition deteriorated overtime with intervening pulmonary infection and septicemia. Follow-up echocardiographic examination showed both enlargement of the residual defect (6.5 mm) and increase in systemic-to-pulmonary shunt ratio (Qp/Qs 1.9) (Fig. 1c).

On the 21st postoperative day, transcatheter closure of the recurrent VSD was planned. It was thought to be less invasive compared to a redo surgical procedure. Under general anesthesia, the right femoral artery and vein and the right internal jugular vein were cannulated. Intravenous heparin was administered. The VSD was crossed using a retrograde arterial approach with a 6 Fr end-hole catheter guided by a hydrophilic guide wire. The catheter was advanced into the left pulmonary artery over the hydrophilic guide wire, which was then exchanged for a 0.035 inch exchange length guide wire. The proximal end of the ex-

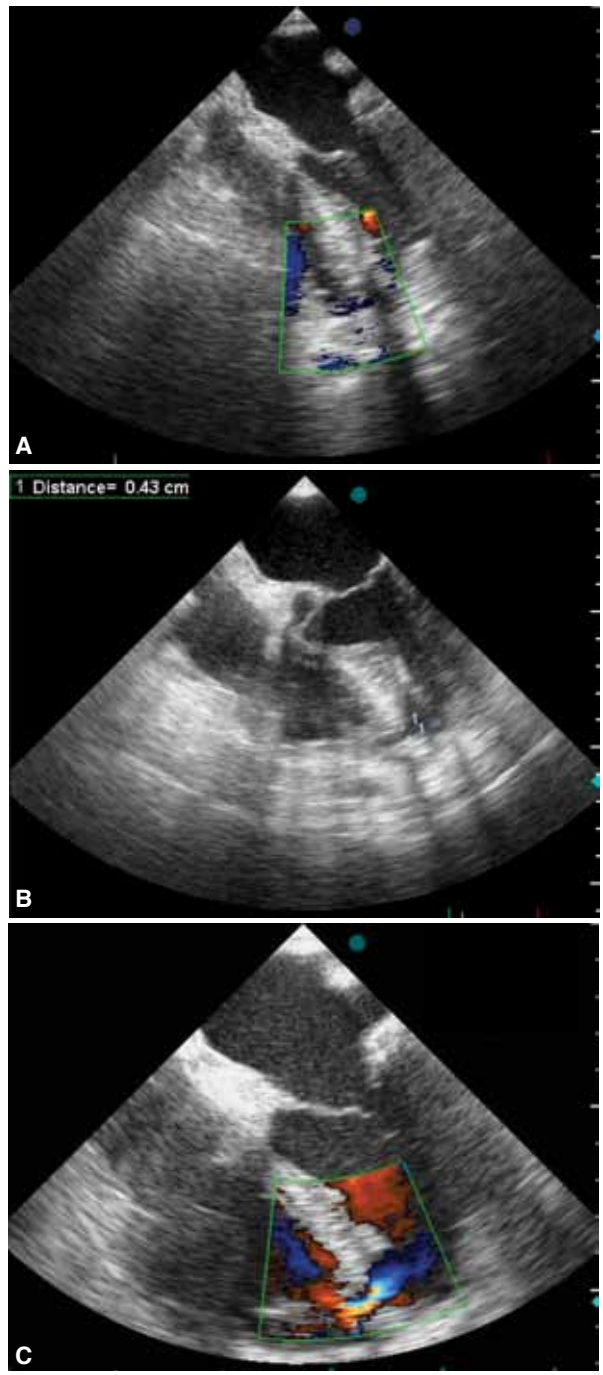

Figure 1. **(A)** Intraoperative transesophageal echocardiography shows no residual shunt. **(B)** Transesophageal echocardiography on the second postoperative day shows a residual defect about 4 mm in diameter. **(C)** Follow-up echocardiography shows enlargement of the residual defect to 6.5 mm with increased pulmonary-to-systemic flow ratio.

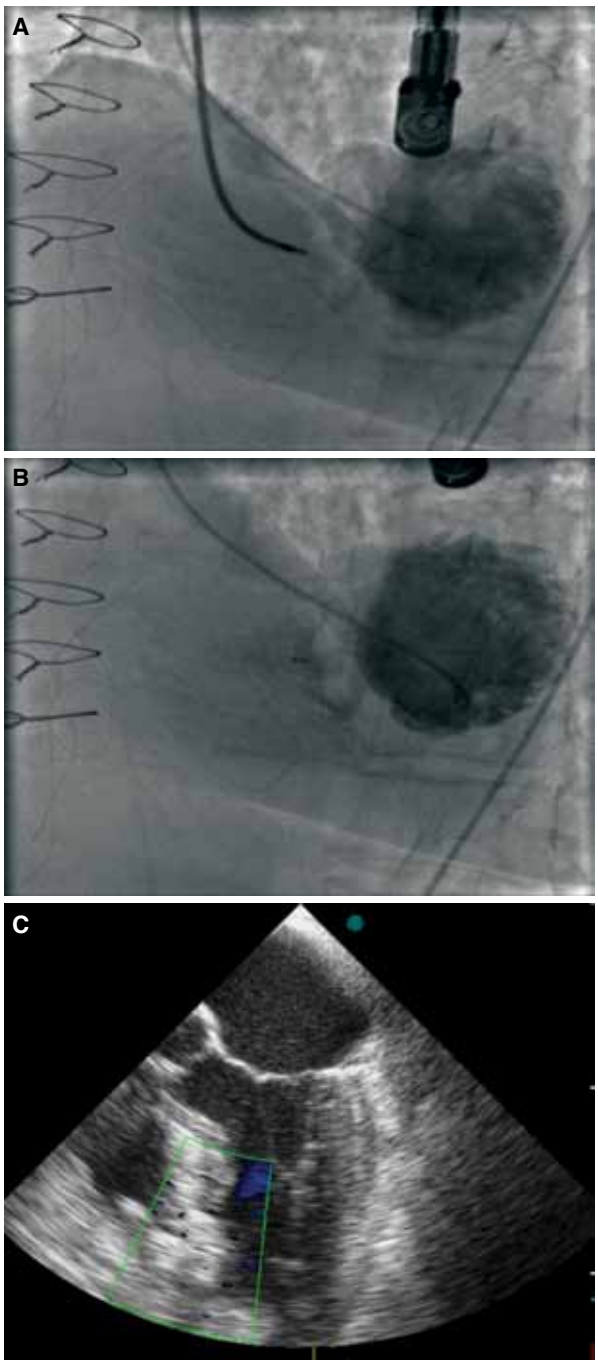

Figure 2. Control angiograms **(A)** before and **(B)** after the release of the device show minimal flow through the device. **(C)** Postprocedural transesophageal echocardiography shows only trivial left-to-right shunt through the device.

change guide wire was then snared in the pulmonary artery with a Goose-neck snare and then extruded via the right internal jugular vein, thereby creating an arteriovenous guide-wire loop. A 25-mm sizing balloon was then introduced over the exchange guide wire into the left ventricle. The balloon was inflated with dilute contrast and the waist was measured. The stretched diameter was 8.3 mm. A 9-Fr long sheath was advanced from the internal jugular vein into the left ventricle. A 10-mm Cardio-O-Fix septal occluder was loaded and introduced through the long sheath. The distal disc was opened and pulled back onto the left ventricular side of the septum under echocardiographic guidance. After confirmation of septal alignment, the proximal disc was deployed. The device was then released by counterclockwise rotation of the delivery wire (Fig. 2 a, b). Transesophageal echocardiography showed only trivial left-to-right shunting through the device (Fig. 2c). Transthoracic echocardiography on the following day showed a small residual shunt. She was weaned from the ventilator, inotropic support was stopped, and she was discharged from the intensive care unit in one week. No intracardiac shunt was detected after 36 days of the procedure.

DISCUSSION

Early surgical closure of post-MI VSD may improve survival, but long-term outcome depends on the presence or absence of residual shunting and left ventricular function.[2,7] Sutures may tear out easily from the acutely infarcted myocardium, resulting in life-threatening suture-line rupture and patch dehiscence. This residual defect may cause significant hemodynamic disturbance or hemolysis requiring re-intervention.

Transcatheter post-MI VSD closure may provide short-term hemodynamic stabilization either as an interim measure to allow myocardial strengthening by scarring or an alternative to primary or redo surgery.[4-6,8,9] In our patient, a significant residual shunt remained after surgery with preserved inferior and posterior left ventricular contractility. Because of her unstable hemodynamic condition that increased the risk for a redo surgery, transcatheter closure of the residual defect was planned and undertaken under multiplane transesophageal echocardiography guidance.

It has been suggested that estimation of balloon-stretched diameter of the defect is necessary to select device size[6,10] as measurements by echocardiography may underestimate defect size. However, this is controversial especially in the acute setting, since it is not uncommon to cause enlargement of the ruptured defect even with a very compliant sizing balloon. In our case, the acute period was over and we felt comfortable to use a sizing balloon in order to have a better estimation of the defect size.

In conclusion, transcatheter closure of post-MI VSDs offers an important therapeutic option in patients who are poor candidates for surgical closure and have hemodynamically significant residual shunts. More experience is needed to assess its value as a primary closure technique or bridge to surgery in acute ventricular septal rupture.

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Anahtar sözcükler: Kalp kateterizasyonu; kalp septal defekti, ventriküler/etyoloji/komplikasyon/tedavi; miyokart enfarktüsü/komplikasyon; ventriküler septal yırtık/etyoloji/tedavi.