Transcatheter closure of an aortico-left ventricular tunnel using Amplatzer® Vascular Plug II

Olgu Sunumu

Aortiko-sol ventriküler tünelin Amplatzer® Vascular Plug II kullanılarak transkateter kapatılması

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ABSTRACT

An aortico-left ventricular tunnel results from abnormal communication between the ascending aorta and the left ventricle, bypassing the aortic valve and causing unrestricted diastolic run-off to the left ventricle. The standard approach involves surgically closing the tunnel immediately following diagnosis, even in asymptomatic patients. Although a few reports exist regarding closure of the aortico-left ventricular tunnel with device, to our knowledge, this is the first article to report on the successful closure of aortico-left ventricular tunnel using an Amplatzer® Vascular Plug II.

Keywords: Aortico-left ventricular tunnel; transcatheter closure; vascular plaque.

ÖZ


Anahtar sözcükler: Aortiko-sol ventriküler tünel; transkateter kapama; vasküler plak.

An aortico-left ventricular tunnel (ALVT) results from abnormal communication between the ascending aorta and the left ventricle (LV), bypassing the aortic valve and causing unrestricted diastolic run-off to the LV.[1,2] Patients with this condition primarily suffer from chronic volume overload of LV, LV dysfunction and aortic valvar insufficiency. The standard approach involves surgically closing the tunnel immediately following diagnosis, even in asymptomatic patients.[3] Although a few reports exist regarding device closure of the ALVT, to our knowledge, this article is the first to report on the successful device closure of ALVT using an Amplatzer® Vascular Plug II (AVP II).[2,4,5]

CASE REPORT

A 14-year-old male patient was referred to our center for evaluation of a murmur. He complained of shortness of breath during exercise. Physical examination revealed a loud “to-and-fro” murmur all over the precordium, and peripheral pulses were bounding. The initial transthoracic echocardiographic examination revealed tunnel-like communication between the ascending aorta and LV, originating superior to the right coronary cusp, traversing between the aorta and pulmonary artery, and ending in the left ventricular outflow tract (Figure 1). A Color Doppler examination showed an unrestricted diastolic regurgitant flow through the tunnel into the LV. No aortic insufficiency was observed. There was significant left ventricular dilation with end-diastolic dimension (LVEDd) measuring 72 mm (Z-score: +4.5). The LV ejection fraction (EF) and fractional shortening (FS) were 45% and 22%, respectively. The tunnel measured 7.7 mm in diameter and 27 mm in length at the narrowest point.
The patient was admitted to the catheter laboratory with the aim of device closure of the tunnel using transesophageal echocardiography (TEE) guidance. Bilateral femoral artery access was obtained, and 100 units/kg of heparin were administered. An ascending aortogram performed in the right anterior oblique and lateral views using a 6F pigtail catheter revealed a sigmoid-shaped tunnel measuring 5.8 mm at the aortic orifice, 7.8 mm in the middle section and 11 mm at the left ventricular opening (Figure 2). After crossing the defect with 0.018” Pointer® guidewire through a 6F right Judkins catheter (Medtronic Inc., Minneapolis, MN, USA), balloon interrogation with an 8x30 mm Tyshak® balloon was performed to delineate the tunnel’s size and course. The defect showed a loose indentation on the balloon at its aortic opening. Then, an appropriate guiding catheter was advanced into the LV over the wire. A 12 mm AVP II (St. Jude Medical, Inc., St. Paul, Minnesota, USA) was utilized initially and placed in the middle section of the defect. However, it could not be released due to the significant residual regurgitant flow seen on the control aortogram. A 14x12 mm Amplatzer duct occluder (ADO) then was implanted into the tunnel. The device occluded the defect completely but caused significant aortic insufficiency (AI). Ultimately, it was only possible to occlude the defect with a 16 mm AVP II without significant AI on TEE.

After releasing the device, the control angiogram showed complete occlusion of the tunnel (Figure 3, 4). The coronary arteries were patent, and there was no demonstrable aortic regurgitation. The following day, echocardiography also showed complete occlusion of the defect with trivial AI (Figure 5, 6). The patient was discharged 24 hours after the procedure and started aspirin at a dose of 5 mg/kg/day. Eight months after the device closure, echocardiogram showed that the device was positioned well with no residual shunt. There was trivial aortic regurgitation with normal LV end diastolic dimension and systolic function (LVEDd: 52 mm, LVEDd Z-score: +1.27, EF: 58%, FS: 29%).

DISCUSSION
Aortico-left ventricular tunnel is a very rare congenital defect. Aortico-left ventricular tunnel is usually diagnosed during infancy with symptoms of heart failure; however, it may not be symptomatic until the second decade or even later in life.[3,5,6] The defect causes unrestricted diastolic flow from the ascending aorta to the LV, so progressive LV dilation with or without systolic dysfunction invariably occurs with large defects. Although surgical correction has been accepted as the treatment of choice,[7] there have been a few reports of successful transcatheter device closure of ALVT, especially with ADOs.[2,4,5]

Major concerns about the transcatheter device closure include proximity of the defect to the coronary arteries, compression and deformation of the aortic valve, and incomplete closure that can cause significant hemolysis.[3,4,8] However, we believe that device closure of ALVT using an AVP II has several advantages over surgery. The procedure eliminates
the need for sternotomy and cardiopulmonary bypass and shortens hospital stay. It also precludes surgery-related complications, such as impingement of the right coronary cusp, distortion of the aortic valve and left bundle branch block that occurs due to sutures placed in the aortic and ventricular openings. Regardless of surgical technique, researchers have reported an incidence of AI in patients with ALVT after surgical closure up to 60%. On the contrary, the AVP II may act as a supporting tissue to the right coronary cusp by filling the gap created by the tunnel, thus avoiding future aortic valve dysfunction.

The AVP II is a cylindrical self-expanding device made of nitinol wire mesh and indicated for peripheral arterial and venous embolization. It has a central lobe surrounded by two shorter occlusion discs. These three lobes are all of the same diameter and create six planes of occlusion. The manufacturer recommends choosing an AVP II size 20 to 50% larger than the target vessel diameter for complete occlusion. However, in their study on 178 pediatric patients with patent ductus arteriosus, Garay et al. needed an AVP II size 2.6 times the ductus’ narrowest diameter for satisfactory occlusion.

Figure 3. After releasing the device, complete occlusion of tunnel is shown in the anteroposterior angiogram.

Figure 4. After releasing the device, complete occlusion of tunnel is shown in the left lateral angiogram.

Figure 5. Transthoracic parasternal long axis view revealed complete occlusion of tunnel.

Figure 6. Transthoracic parasternal short axis view revealed complete occlusion of tunnel.
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occlusion. In our patient, a 12 mm AVP II could not completely occlude the tunnel, and the size of final implanted device was 2.7 times the narrowest tunnel diameter. Therefore, we believe that larger-than-recommended sizes of AVP II may be required in these distensible defects.

Use of AVP II in ALVT has several advantages over ADO I and other devices with a retention disc larger than the main body. The larger retention disc in these devices may cause hemolysis due to shear stress on the erythrocytes when remaining in the left ventricle.[4] As seen in our patient, the retention discs may distort the aortic valve and result in significant aortic insufficiency when left inside the tunnel. In addition, the proximal retention skirt on a double disc device may obstruct the coronary artery entries when left on the aortic side. In our patient, the AVP II, with its flexible and soft nature, perfectly conformed to the shape of the ALVT. Due to its design with three lobes of the same diameter, the AVP II also eliminated the risk of protrusion of the retention skirts into the left ventricular cavity or ascending aorta.

In conclusion, our report suggests that it is feasible to close an aortico-left ventricular tunnel via a transcutaneous procedure, depending on the size and morphology of the tunnel and the patient age. Amplatzer® Vascular Plug II may be preferable in transcatheter closure of aortico-left ventricular tunnel because of its soft structure and strong occlusive characteristics. However, the dimensions of aortico-left ventricular tunnel measured on echocardiography and angiography may be misleading due to the complex morphology and distensibility of the defect; therefore, larger than predicted devices may be required.

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REFERENCES