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IMAGES IN INTERVENTION

Left-to-Right Interventricular Shunt as a Late Complication of Transapical Aortic Valve Implantation

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An 86-year-old man was referred to our institution for a symptomatic severe aortic stenosis (New York Heart Association [NYHA] functional dyspnea class III; indexed effective orifice area [EOA]: 0.3 cm^2/m^2; mean gradient [MG]: 54 mm Hg; left ventricular ejection fraction [LVEF]: 56%). He had a history of dyslipidemia, prostate cancer, and coronary artery bypass graft (saphenous vein graft on left anterior descending coronary artery [LAD] and right coronary artery) 26 years ago.

A transcatheter aortic valve implantation (TAVI) was proposed, because this patient was considered a high-surgical-risk candidate (Logistic EuroSCORE 28%, Society of Thoracic Surgeons Score 7%). A transapical approach was chosen, because both the iliofemoral and subclavian arteries were not suitable for transarterial access. The aortic annulus measured by transesophageal echocardiography was 22.6 mm.

The procedure was performed under general anesthesia, through a left minithoracotomy, as previously described (1). A 26-mm Edwards-Sapien valve (Edwards Lifesciences, Inc., Irvine, California) was implanted under fluoroscopy and transesophageal echocardiography guidance, with good procedural outcome. The post-implantation period was marked by an increase of cardiac troponin I (peak: 12.9 ng/ml) and a transient renal failure. At discharge (Day 10), the patient was in NYHA functional class II, and heart sounds were normal. Transthoracic echocardiography (TTE) showed LVEF: 63%, prosthesis MG: 11 mm Hg, indexed EOA: 0.91 cm^2/m^2, stable mild paravalvular leak, and pulmonary artery systolic pressure: 47 mm Hg.

Clinical and TTE data were similar at 1-month and 6-month follow-up. At 1-year follow-up, the patient declined in NYHA functional class III. He didn't describe any chest pain since his last visit. Clinical examination revealed a loud continuous systolic and diastolic murmur. There was no change of the electrocardiogram. The TTE revealed a moderate enlargement of the right ventricular cavity. The apical part of the interventricular septum appeared less echogenic than the rest of the myocardium (Fig. 1). Color Doppler showed a continuous left to right flow through the apical part of the interventricular septum with a Doppler peak systolic gradient of 89 mm Hg (Figs. 2A and 2B). The LVEF, prosthetic mean gradient, and indexed EOA were 63%, 11 mm Hg, and 0.90 cm^2/m^2, respectively. Pulmonary artery systolic pressure was 65 mm Hg. An echocardiographic contrast agent (Sonovue, Bracco Imaging, Milan, Italy) failed to reach the apex of the right ventricle. It seemed to be "washed" by the shunt flow (Fig. 3, Online Video 1). After a few cardiac cycles, contrast agent appeared into the left cardiac cavities and at last into the right ventricle apex through the septal communication.

After considering the age and will of the patient, a moderate cognitive impairment, a progressive prostate cancer, and despite the worsening of his functional status, we decided against any invasive treatment.

Various transapical TAVI complications have been described, mostly observed during initial procedure or short-term follow-up (2,3).

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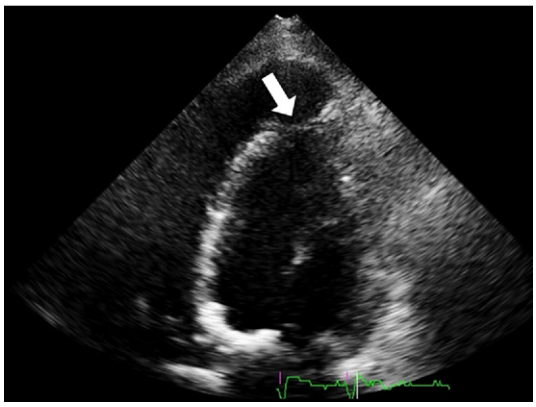

Figure 1. 2-Dimensional Transthoracic Echocardiography Apical View of the LV and RV

The echogenicity of the apical segment **(white arrow)** of interventricular septum is less pronounced than the basal and medial segments. The right ventricle (RV) apex is enlarged. LV = left ventricle.

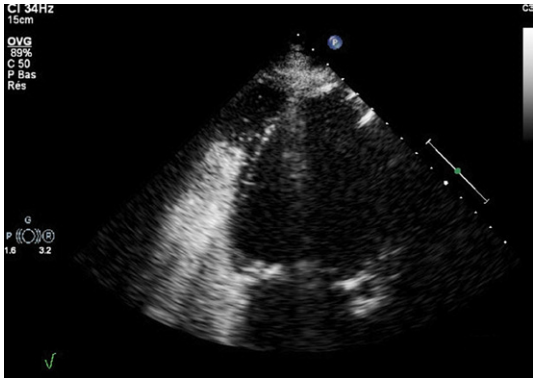

Figure 3. Intravenous Injection of an Echocardiographic Contrast Agent

During the first cardiac cycles, microbubbles cannot reach the right ventricular apex because of "washing effect" of left-to-right flow; (Online Video 1). GP = maximal pressure gradient; Vit = peak velocity.

In this case, we can reasonably argue that puncture through the septum at the time of the procedure did not cause this late septal defect, because we can expect that it would have been observed before discharge or during initial follow-up. It is likely that this complication was caused by late rupture of scar tissue due to periprocedural injury. The potential mechanisms of this injury could be:

1. Occlusion of the distal LAD or a side branch by the purse string suture, resulting in a periprocedural myocardial infarction.
2. A pressure necrosis caused by an apical puncture and then an insertion of the sheath too close to the distal septum.

To our best knowledge, this is the first report of late apical interventricular communication after transapical TAVI. Considering the possible causes discussed in the preceding text, periprocedural echocardiographic guidance of the left ventricle apical puncture and subsequent sheath insertion could be proposed to prevent this complication. Particular attention should also be given to the location of the distal LAD before making the purse string suture.

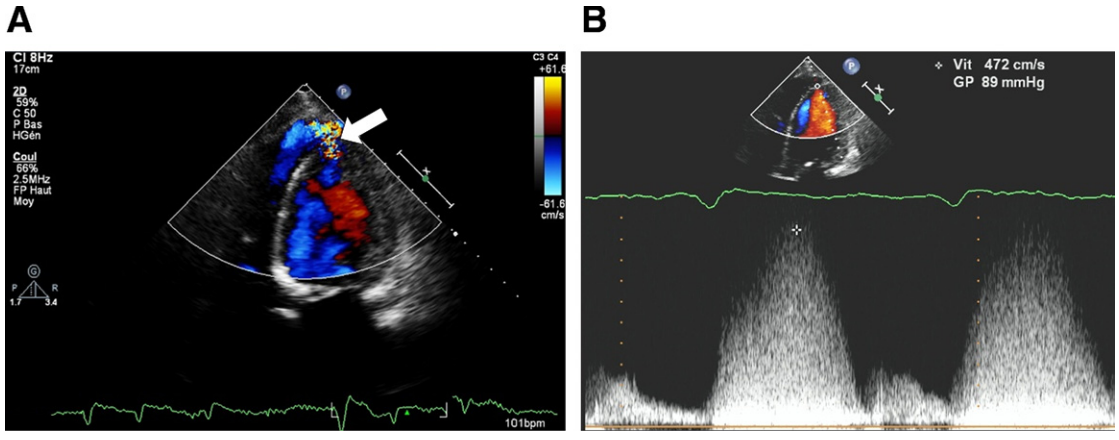

Figure 2. Color-Doppler Imaging of the Left-to-Right Flow

(A) Color-Doppler imaging of the left-to-right flow through the apical part **(white arrow)** of the interventricular septum. **(B)** Continuous-wave Doppler of the left-to-right flow through the apical part of the interventricular septum. 2D = 2-dimensional; coul = color Doppler; GP = maximal pressure gradient; Vit = peak velocity.

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This case emphasizes the role of systematic regular follow-up to detect late complications after such procedures. It might also improve our awareness of the wide range of potential complications of TAVI.

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APPENDIX

For supplementary videos, please see the online version of this article.