

Figure 2. Left: aortography showing a normal aortic coalctation or interruption (5). Right: aortography after stenting, showing a normal aortic calibre (6).

acute heart failure. Percutaneous treatment with dilation of the coartation and stenting was decided upon, with good results.²

In our area, more than 90%–95% of heart failure cases are due to ischemic heart disease, hypertension, arrhythmias and valvular disease, but on occasion, as with this patient, the unexpected may happen. In this case, and contrary to all statistics and initial data, the sound of hoofbeats were those of a zebra and not of a horse.

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Available online 18 December 2010

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doi:10.1016/j.rec.2010.06.003

Transapical Technique as an Alternative Approach to Paravalvular Leak Closure

Vía transapical como alternativa para el cierre del leak paravalvular

To the Editor,

Paravalvular leaks occur in between 3% and 7% of cardiac valve prostheses. As many patients in whom closure is indicated are at high surgical risk, it is often necessary to resort to percutaneous techniques. Such approaches are complicated by the lack of specifically designed materials. Nevertheless, results continue to improve as a result of advances in imaging techniques and the development of new devices.

Anterograde or retrograde approaches can be used according to the location of the prosthesis and the leak. Despite the use of stiff guidewires or looped guidewires to establish arteriovenous loops, advancing the introducer for the dispensing catheter through the leak is usually the most complicated step.

We present a case in which a transapical technique^{1,2} was used when both anterograde and retrograde approaches failed. This new technique allowed rapid and straightforward access to the leak, as well as providing good support for advancement of the introducer.

The patient had aortic and mitral mechanical valve prostheses and had been admitted to hospital three times for acute pulmonary edema. A paravalvular leak estimated by transesophageal echocardiography (TEE) to be 8 mm high, 6 mm long, and 4 mm wide was observed in the posteroseptal region and was associated with severe mitral insufficiency and signs of hemolysis. As the patient had a logistic EuroSCORE of 32%, a decision was taken to attempt percutaneous closure. The first attempt employed an anterograde approach via transeptal puncture. Despite the use of stiff guide wires, no catheters could be introduced that would allow positioning of the device. In the same procedure, a retrograde approach was also attempted. When the catheter was advanced to the left ventricle through the aortic valve prosthesis, one of the discs of the prosthesis was repeatedly blocked, leading to severe hemodynamic deterioration. A decision was therefore taken to attempt a transapical procedure.

Forty-eight hours later, the procedure was done under general anesthesia with TEE guidance. After localization of the apex by transthoracic echocardiography, a left anterior minithoracotomy

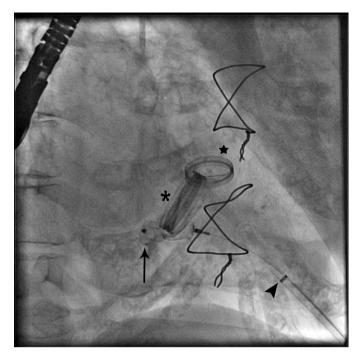


Figure 1. Angiogram showing the device (arrow), introduced through the dispensing catheter via a transapical approach (arrowhead), along with the mitral valve prosthesis (asterisk) and the aortic valve prosthesis (star).

was performed and a tobacco pouch suture prepared. The apex was punctured with a trocar and a standard "J" 0.035 mm guidewire (Bolton Medical Inc. Sunrise, FL, USA) was advanced to place an 8 French introducer (Terumo Medical Corp, Tokyo, Japan). A straight, hydrophilic, 0.035 mm guidewire (Terumo Medical Corp, Tokyo, Japan) was advanced through the leak until it reached the left atrium and then a Vista Brite tip JR 3.5 catheter (Cordis, Miami, FL, USA) was advanced over it. The guidewire was then replaced with a stiff guidewire (Amplatzer Super Stiff, AGA Medical, Golden Valley, MN, USA) that allowed the 8 French catheter for the device to be advanced. A Vascular Plug III (AGA Medical. Golden Valley, MN, USA) occlusion device measuring $12 \text{ mm} \times 5 \text{ mm}$ was then advanced with the dispensing catheter in the left atrium and when only the distal segment had been opened it was withdrawn towards the left ventricle until it reached the leak, without releasing it. The mitral insufficiency disappeared but the device interfered with one of the discs and a decision was taken to remove the entire system. Another Vascular Plug III occluder measuring $12 \text{ mm} \times 5 \text{ mm}$ was advanced and, this time, it was opened completely in the left atrium before inserting it into the leak (Fig. 1). The device did not interfere with any of the discs but left slight residual paravalvular insufficiency. After performing pushing maneuvers and confirming that it was well supported, the device was released (Fig. 2). Finally, the tobacco pouch suture was closed.

During follow-up, echocardiography and direct visualization did not reveal morphologic or functional changes compared with the results obtained immediately following implantation.

The patient was discharged and continued to show clinical improvement.

The transapical technique may speed up, facilitate, and simplify procedures for closure of paravalvular leaks and should be considered in complex cases or in those situations in which percutaneous approaches have failed.

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Available online 15 December 2010

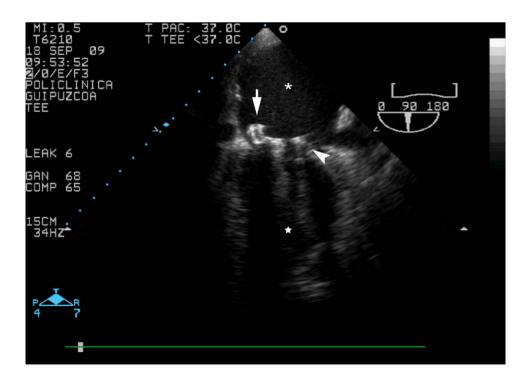


Figure 2. Transesophageal echocardiogram. The final position of the Vascular Plug III occlusion device (arrow) can be seen along with the mitral valve prosthesis (arrowhead), the left atrium (asterisk), and the left ventricle (star).

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doi:10.1016/j.rec.2010.06.006

An Unusual Complication of Exercise Echocardiography

Una rara complicación de la ecocardiografía de esfuerzo

To the Editor,

A 53-year-old woman was referred for stress echocardiography. Her clinical record included a history of high blood pressure and VVIR pacemaker implantation secondary to atrioventricular nodal ablation for symptomatic refractory paroxysmal atrial fibrillation. During the previous 3 months, she reported episodes of precordial oppression and dyspnea triggered by emotionally stressful situations. Exercise echocardiography was indicated because of recurrent pain and the pacemaker rhythm in the electrocardiogram (ECG).

Baseline echocardiography showed concentric left ventricular hypertrophy (12 mm) and a left ventricular ejection fraction (EF) of 67%, with interventricular asynchrony due to pacemaker stimulation without alterations in segmental contractility. At the end of the fifth minute of the Bruce protocol (heart rate 105 bpm [63% of maximum], blood pressure 180/85 mm Hg), the patient experienced intense chest pain. Echocardiography showed septoapical and inferoapical akinesis.

Given the persistent pain, the patient was referred to our center for urgent coronary angiography. The coronary arteries appeared normal. Ventricular angiography showed akinesis of the middle segments of the anterior and inferior wall, apical hypokinesia, basal segment hypercontractility and an EF of 45%–50% without an intraventricular gradient (Fig. 1).

The patient was hospitalized in the coronary unit, where treatment with atenolol was begun. She experienced no new episodes of chest pain. The troponin-T level reached a maximum of 0.19 ng/mL (normal <0.1 ng/mL) in 24 h and normalized at 48 h (0.07 ng/mL); the creatine kinase level was within the normal range. At 24 h, the ECG revealed a negative precordial T wave (Fig. 2). At 1 week, the echocardiogram showed no alterations in left ventricular segmental contractility and the EF had recovered to 65%. Consequently, the patient was discharged with a diagnosis of Tako–Tsubo syndrome.

Tako–Tsubo syndrome—or stress-induced cardiomyopathy—is a clinical entity characterized by the presence of chest pain in the context of a stressful situation, an ECG that mimics ST-segment elevation myocardial infarction, normal coronary arteries, and transitory left ventricular dysfunction due to midapical akinesis or dyskinesis.¹ In acute coronary syndromes,² the estimated prevalence is 0.7%. Knowledge of the pathophysiology is quite limited although it has been suggested that dysfunction of the coronary microcirculation occurs due to the release of catecholamines into plasma triggered by stress.³ However, in some cases, no trigger mechanism has been found.⁴ In our patient, the only trigger was the physical and emotional stress of undergoing a test for ischemia. The baseline echocardiogram was normal and akinesis appeared during the test. Ventricular angiography performed shortly after this suggested Tako–Tsubo syndrome. In our patient, the dysfunction mainly affected the middle segments, a location that

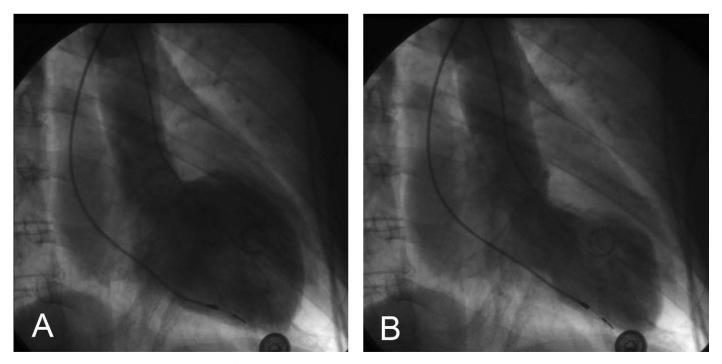


Figure 1. Ventricular angiography. Akinesis of the middle segments, apical hypokinesia, and baseline hypercontractility. Diastole (A) and systole (B).