

LOOPING AGAINST TAVI LEAKS. THE HIDDEN HOOK

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HISTORY AND PHYSICAL

We present a 61 years old man with diabetes and dyslipidemia. In 2011 a degenerative aortic valve disease, associated to a moderate aortic stenosis with a hypertrophic left ventricle and a normal ejection fraction was diagnosed which was stable in periodic transthoracic echocardiograms (TTE) studies.

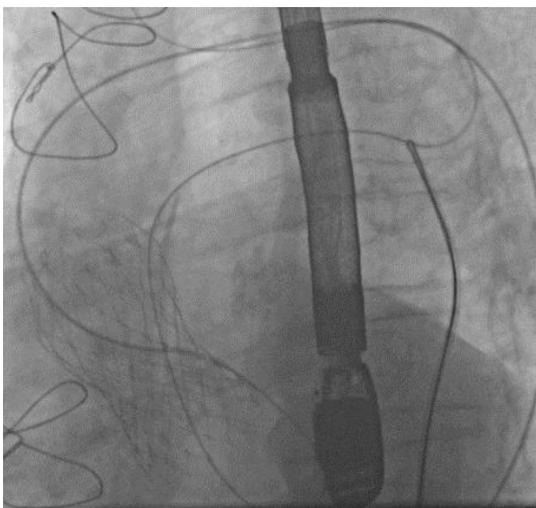
In 2014 our patient presented with progressive dyspnea, reaching NYHA grade III. Transaortic gradients in TTE were similar to the previous ones. We performed a catheterization study, invasive gradients were measured in the cath lab (peak to peak aortic gradient 52 mmHg and mean gradient of 35 mmHg); coronary arteries were normal. Taking in to account the possibility of a low gradient severe aortic stenosis a stress TTE was performed in 2015. In the basal images a severe stenosis gradient was detected (maximum of 68 mmHg and mean of 43) increasing significantly in the immediate post-stress phase.

The patient was presented during the heart team session and accepted for aortic valve replacement surgery. In February 2016 surgery was planned, nevertheless when the surgeons started the intervention, after sternotomy, they found a extended calcification of the aortic root and ascending aorta. Because it was impossible to make an effective aortic clamp, they decided to finished the procedure and present the patient during the heart team session again. At this time patient was jointly accepted for a transcatheter aortic valve implantation procedure.

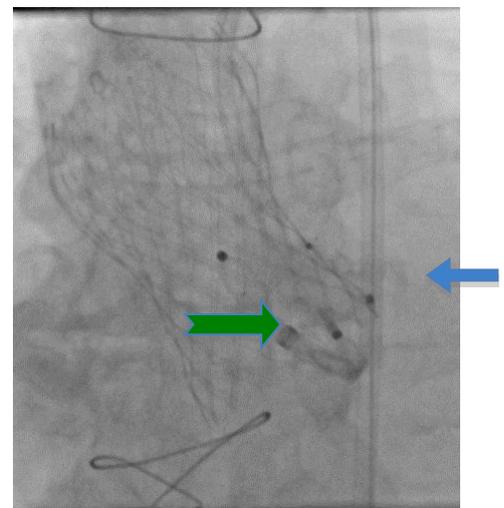
CT-scan confirmed extensive calcification of aortic valve and aortic root. We also use CT images to measured aortic ring (30x26 mm), area (60 mm²) and perimeter (88.1 mm)

In April 2016 a percutaneous aortic valve bio-prosthesis Corevalve 31 mm was implanted. After the procedure, a severe aortic regurgitation was documented, related with two periprosthetic leaks (a posterior predominant one of 15 x 4 mm and a lateral of 6 x 3 mm). In de following months the patient developed symptoms of heart failure that persisted regardless of appropriate optimization of medical treatment.

IMAGING



Detail of the arterioarterial formation, capturing the guide with the tie



Delivery of the AVP device in the posterior leak (green arrow) and AVP device positioned in the lateral leak (blue arrow)

INDICATION FOR INTERVENTION

The functional repercussion that the severe aortic regurgitation was causing was the main reason to try to close the multiple leaks, in order to reduce aortic regurgitation, improve the symptoms and probably life expectancy.

INTERVENTION

We performed the procedure with propofol sedation and continuous transesophageal echocardiographic monitoring (TEE). Through the left femoral artery, using a multipurpose catheter, we advanced a long hydrophilic Terumo guide of 260 cm through the corevalve struts and the lateral leak reaching in a retrograde way the left ventricle. Then we move the guide forward in an anterograde way, through the natural lumen of the Corevalve n° 31 reaching ascending aorta. We capture the guide with a PFM tie of 30 mm that was passed in a retrograde way through the right femoral artery, and then we externalize the guide. In this way, we made our arterioarterial loop with its origin in the left femoral artery, through the descending aorta- ascending aorta- struts and lateral leak- left ventricle- ascending aorta- descending aorta and right femoral artery.

Once the arterioarterial loop was made we progress a 7 F sheath/dilator, crossing retrograde through the valve to the left ventricle and then anterograde to the lateral leak. We remove the guide and progress an AVP device 6 x 3 mm (St Jude Medical), then we release the device confirming the occlusion efficacy by fluoroscopy and Doppler.

Using the same strategy and sequence, we approached the posterior leak. An arterioarterial loop was made and a 8 F sheath/dilator was advanced through the aortic valve to the left ventricle and then anterograde through the posterior leak, between the aortic root and the valve struts.

Once the sheath was positioned we removed the dilator and the guide and advanced a second device AVP III 14 x 5 mm (St Jude Medical). The device was correctly released, confirming once again a correct position and satisfactory occlusion.

The procedure was finished without any incidence, and a control TEE performed before discharge confirmed a total occlusion of the leaks.

At 6-month follow up our patient is stable in NYHA grade and the TTE control confirmed absence of aortic regurgitation, with a normal left ejection fraction.

LEARNING POINTS OF THE PROCEDURE

Paravalvular aortic regurgitation is one of the most common complications after percutaneous aortic valve implantation, with a negative impact in medium term life expectancy. Severe calcification is one of the predictors of leaks.

In self-expanding valves the disposition of the struts may complicate crossing of the sheath. The arterioarterial loop allows passing the sheath retrogradely to the left ventricle and anterogradely to the leak, helping to place the AVP device in the right position.

On the other hand, the design of AVP devices offers an excellent alternative to achieve the total occlusion of the leaks.