Another failed surgical bioprosthetic valve: a second transcatheter valve-in-valve solution

Outra falha de bioprótese valvar cirúrgica: uma segunda solução transcateter valve-in-valve

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ABSTRACT - Transcatheter mitral valve-in-valve replacement has recently emerged as an increasingly common alternative for high surgical risk patients. We report a case of a successful transseptal transcatheter mitral valve-in-valve replacement for the treatment of a bioprosthetic mitral valve degeneration and severe regurgitation, in an 86-year-old patient who had undergone transcatheter aortic valve-in-valve procedure 6 years ago. This case emphasizes the crucial role of a careful preoperative assessment using multimodality imaging to plan the procedure, in a patient with higher risk of left ventricular outflow obstruction due to the previous transcatheter aortic valve-in-valve procedure.

Keywords: Mitral valve; Aortic valve; Heart valve prosthesis implantation; Prosthesis failure


Descritores: Valva mitral; Valva aórtica; Implante de prótese de valva cardiaca; Falha de prótese

INTRODUCTION

Bioprosthetic heart valves (BHV) are increasingly chosen as the favourite valve due to their favourable hemodynamic profile and low thrombogenicity. However, BHV are prone to structural valve degeneration (SVD), which has become a major challenge for long-term prognosis, resulting in limited long-term durability and often requiring redo valve replacement. Most patients who develop SVD are older and have a prohibitive surgical risk. To meet the needs of high-risk patients, transcatheter mitral valve-in-valve (TMViV) replacement has emerged in the past years and changed the paradigm of treatment of these patients.

This case report aimed to emphasize the crucial role of a careful preoperative assessment using multimodality imaging to plan the procedure, in a patient with higher risk of left ventricular outflow obstruction, due to the previous transcatheter aortic valve-in-valve (TAViV) procedure.

CASE REPORT

An 86-year-old male patient, with permanent atrial fibrillation (AF) and history of degenerative valvular disease, underwent simultaneous aortic and mitral valve replacement (MVR) by surgical bioprosthesis (Carpentier-Edwards 23; Edwards Li...
fesciences, Irvine, California, United States and St Jude Medical-Biocor 27; Inc, St Paul, Minnesota, United States, respectively) in 2002, due to severe aortic and mitral stenosis. In 2013, due to symptomatic severe bioprosthetic aortic valve regurgitation, he successfully underwent TAViV replacement (Edwards Sapien 3), with no residual leak. His regular medication was vitamin K antagonist, furosemide and ramipril.

In December of 2019, the patient was admitted to emergency department due to the self-perception of a thrill in the precordium, noticed in the previous week, associated with the progressive worsening of dyspnea in the last three months. He is currently in New York Heart Association (NYHA) class III. He denied chest pain, palpitations or syncope. At physical examination he was afebrile, with a blood pressure of 110/60mmHg, a 6/6 holosystolic murmur in the precordium, crackles at the lower lobes and well perfused extremities. Electrocardiogram showed AF with a controlled ventricular response and left ventricular hypertrophy criteria. Laboratorial investigation showed increased prohormone of brain natriuretic peptide level (2,564pg/mL, reference <125pg/mL) and slightly elevated c-reactive protein (18mg/L, reference <3mg/L). Chest X-ray showed cardiomegaly and bilateral interstitial infiltrates. Transthoracic echocardiography (TTE) showed a bioprosthetic mitral valve degeneration and an eccentric severe transvalvular regurgitation, with a mildly reduced left ventricular ejection fraction (LVEF; 45%), moderate resting pulmonary hypertension, in addition to a normally functioning aortic bioprosthesis. Transoesophageal echocardiography (TEE) was performed to clarify mitral regurgitation (MR) mechanism and assess atrial septum anatomy. It revealed a small perforation and flail of the posterior leaflet of mitral bioprosthesis, causing a severe eccentric transprosthetic mitral regurgitation, with two visible jets and Coanda effect (Figure 1 and Videos 1 and 2). Additionally, TEE estimated the mitral annulus-to-interventricular septum (IVS) distance in 27mm, and excluded any interatrial septum anomaly. In the second day since admission, inflammatory markers decreased and became negative. Serial blood cultures were negative. Coronary angiography excluded epicardial coronary disease.

Due to patient's frailty and high surgical risk, as assessed using the EuroSCORE II and Society of Thoracic Surgeons (STS) score, calculated as 18.8% and 9.2%, respectively, it was decided in a Heart Team meeting to perform a transseptal TMViV replacement.

The choice of the ideal valve size for TMViV procedure was determined based on the preoperative assessment by the cardiac computed tomography angiography (CTA), which showed a previous aortic valve-in-valve with normal function and assessed surgical mitral bioprosthesis and left ventricular outflow tract (LVOT) dimensions. It was simulated a three-dimensional virtual implant of a 26-mm Edwards Sapien 3 transcatheter heart valve into the patient’s mitral annulus, showing a low risk of LVOT obstruction (LVOTO), with neither any LVOT narrowing, nor significant calcification or elongation of the mitral bio-

![Figure 1](image_url). Transoesophageal echocardiography at zero (A) and 10° (B) showing the perforation (white arrow) and flail of the posterior leaflet of surgical mitral bioprosthesis, causing an eccentric severe mitral transprosthetic regurgitation with two visible jets: a shorter jet directed to the interatrial septum (blue arrow), in relation to the perforation, and a longer jet directed to the posterior wall of the left atrium (red arrow), exerting a Coanda effect. White arrow indicates perforation of the mitral bioprosthesis posterior leaflet; blue arrow indicates jet direction of severe mitral transprosthetic regurgitation.
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Video 1. Transoesophageal echocardiography at 10º showing an eccentric severe mitral transprosthetic regurgitation with two visible jets: a shorter jet directed to the interatrial septum (in relation to the perforation) and a longer jet directed to the posterior wall of the left atrium, exerting a Coanda effect. No paravalvular leak was seen.

Figure 2. Preoperative evaluation of cardiac computed tomography angiography. (A) Maximum intensity projection reconstruction displaying three chamber view, showing the previously implanted transcatheter aortic valve-in-valve and the surgical bioprosthetic mitral valve (St Jude Medical-Biocor 27; Inc, St Paul, Minnesota, United States), excluding any left ventricular outflow tract narrowing or significant calcification of the anterior mitral bioprosthesis leaflet; (B) axial multiplanar reformation or reconstruction (MPR) exhibiting the internal mitral bioprosthesis annulus dimensions; (C) axial multiplanar reformation or reconstruction showing left ventricular outflow tract area, using the 3Mensio structural heart module software (Pie Medical imaging, Bilthoven, The Netherlands); (D) simulation of the mitral valve-in-valve implantation with assessment of aortic-mitral angle, using the 3Mensio structural heart module software (Pie Medical imaging, Bilthoven, The Netherlands).
prosthesis anterior leaflet. The bioprosthesis in mitral position measured 26x24mm, the LVOT area was calculated in 637mm², and aorto-mitral angle was estimated in 76º (Figure 2). A 26mm Edwards Sapien 3 transcatheter heart valve was chosen for the procedure.

The procedure was conducted under general anesthesia and TEE guidance. The bilateral common femoral veins were accessed under ultrasound guidance. The transvenous pacemaker (Abbott Medical, United States) was positioned through the left femoral vein. The delivery system was through the right femoral vein (14F eSheath; Edwards LifeSciences). A dose of 5,000 units of heparin was given systemically. A transseptal puncture was performed, followed by dilation of the interatrial septum with a 14mm balloon (Abbott Medical) to facilitate the passage of the delivery system. The balloon was removed and a Safari guidewire (Boston Scientific, United States) was advanced across the mitral valve and into the left ventricular apex. A 26mm Edwards Sapien 3 transcatheter heart valve was advanced into the degenerated mitral bioprosthesis using both fluoroscopy and TEE guidance to facilitate crossing of the septum. The inflation was slowly performed, under rapid pacing (180/minute), trying to keep the valve 10% above the sewing ring. The valve alignment was very challenging, and the valve position was considered too much on the ventricular side; however the TMViV final position was accepted, considering the good functional result assessed by intraoperative TEE (Figures 3A and 3C; Videos 3 and 4).

**Figure 3.** (A and B) The deployment of the balloon expandable 26mm Sapien 3 valve into the surgical mitral bioprosthesis under fluoroscopy and transoesophageal echocardiography guidance; (C) the final fluoroscopic image showing the transcatheter aortic valve-in-valve and the transcatheter mitral valve-in-valve in place after the transcatheter mitral valve-in-valve procedure.

**Video 2.** Transoesophageal echocardiography at 0º showing the perforation and flail of the posterior leaflet of the mitral bioprosthesis, causing the eccentric severe magnetic resonance.
Video 3. The deployment of the balloon expandable Sapien 3 valve into the degenerated surgical mitral bioprosthesis.

Video 4. The final fluoroscopic image showing the transcatheter aortic valve-in-valve and the transcatheter mitral valve-in-valve in place after the transcatheter mitral valve-in-valve procedure.
Video 5. Post-procedure transoesophageal echocardiography, at 10° and perpendicular plan, showing the final result of the transcatheter mitral valve-in-valve replacement.

Video 6. Post-procedure three-dimensional transoesophageal echocardiography showing the final result of transcatheter mitral valve-in-valve replacement, with no transvalvular or paravalvular regurgitation.
Following valve deployment, TEE showed resolution of mitral regurgitation with no paravalvular leak, a peak and mean mitral transprosthetic gradients of 8 and 4mmHg (Videos 5 and 6) and the absence of LVOTO (post-procedure peak LVOT gradient increased from 6 to 9mmHg). The mean aortic transprosthetic gradient did not increase after the procedure (mean gradient of 21mmHg). Post-procedure fluoroscopic image with both TAViV and TMViV in place was taken (Figure 3C; Video 4). Two Proglide (Abbott Medical) devices were used for hemostasis of the vascular accesses. In the immediate postoperative period, the patient remained on low molecular weight heparin and switched to warfarin on the third day. Symptoms subsequently reduced and he was discharged on the sixth day in NYHA class II. At nearly three-year follow-up, patient remained stable without further hospitalizations.

**DISCUSSION**

Mitral regurgitation is the second-most frequent valvular heart disease in Europe, and valve replacement surgery remains the gold-standard treatment. More than 200 thousand heart valve replacement surgeries are performed annually worldwide – in that, 20% with mechanical valves and 80% with bioprosthetic valves, and these numbers will probably triple in 2050. Bioprosthetic heart valves are increasingly chosen as the favourite valve due to their favourable hemodynamic profile and low thrombogenicity, often in young patients, with the aim to avoid anticoagulant treatment. They are recommended when good quality anticoagulation is unlikely or contraindicated, because of high bleeding risk, and in those patients whose life expectancy is lower than the presumed durability of the bioprosthesis.

Consequently, the proportion of patients at risk for SVD will constantly rise in near future. However, while surgical reintervention has always been considered the gold-standard treatment for SVD, the introduction of transcatheter heart valves is currently changing the paradigm of bioprosthesis degeneration treatment, mainly among high-risk patients.

Bioprostesis SVD is a complex multifactorial process caused by mechanical stress, lipid-mediated inflammation, and immune rejection processes, which leads to deterioration of leaflets or subvalvular structures of the prosthesis. As a result, thickening, calcification, tearing, or disruption of the prosthetic valve occurs, causing, in most cases, a critical drop in hemodynamic efficacy of the bioprosthetic valve due to stenosis and/or regurgitation. Prosthetic valve thrombosis and infective endocarditis are not included in the definition of SVD, but these complications, even if treated successfully, may subsequently lead to SVD.

Structural valve degeneration is common and frequently occurs more than 7 to 8 years after the initial valve replacement. In mitral valve SVD has been reported in approximately 25 to 30% of cases at 10 years, and 50 to 70% at 15 years. In the mitral position, bioprosthesis are expected to degenerate faster when compared to aortic valve, due to the higher gradients exposure. For this reason, a higher age limit for bioprosthesis in mitral position has been recommended by the latest guidelines (age >70 years old versus 65 years old for the aortic position).

The definition of SVD is built in stages to reflect the process of bioprosthetic degeneration, which is typically gradual, taking place over the years. The stages of SVD are based on the state of the implanted valve and not on the patient’s clinical status. The stage 1 includes the early morphological leaflet changes, with no hemodynamic compromise. The stage 2 corresponds to morphological abnormalities of leaflets with hemodynamic sequelae, being divided according to the type of dysfunction (e.g. stage 2S, in case of moderate stenosis), since clinical implications and pace of deterioration are likely different between both failure modes.

This clinical case illustrates a stage 3 SVD, the most severe stage of SVD, which is characterized by the development of severe stenosis and/or regurgitation. Indeed, valves at this stage should be approached similarly with a reintervention as the recommended treatment when the patient is symptomatic, as occurred with our patient.

Although redo MVR has been considered the gold-standard treatment for bioprosthetic SVD, patients who develop SVD had frequently a prohibitive surgical risk and the TMViV replacement has emerged as a common alternative for these patients. In fact, recent studies have demonstrated that patients with advanced age, male gender, prior heart valve surgery, previous coronary artery bypass graft, preoperative severe left ventricular systolic dysfunction, severe pulmonary hypertension, renal failure on dialysis, non-elective surgery, and very high EuroSCORE-II or STS-score represent extremely high-risk patients, who might benefit from less-invasive transcatheter mitral procedures.

Mortality at 30 days and one year following TMViV replacement has been reported as zero to 8% and 11 to 16%, respectively, with a procedural success rate >95%.

Recent data showed that transseptal access has been associated with lower mortality compared to transapical access, and it is an independent predictor of lower mortality at one year.

Complications of TMViV procedure include LVOTO, valve migration or embolization, elevated postprocedural gradients, residual MR, and valve thrombosis. The methodical assessment of candidates is a key step for conducting a successful transseptal TMViV replacement. Those with a small LVOT and a long calcified anterior MV leaflet may not be good candidates, due to the increased risk of LVOTO, a preventable and potentially life-threatening complication, which occurs in zero to 6% of patients after TMViV.

Irrespective of access choice, the Achilles heel of ViV-transcatheter mitral valve replacement (TMVR) is the iatrogenic LVOTO. ViV-Valve-in-valve transcatheter mitral valve replacement-induced LVOTO, defined as an LVOT peak gradient...
increase of ≥10mmHg post-TMVR, occurs when the metal frame of the transcatheter valve pushes the anterior leaflet of the previous bioprosthesis toward the IVS, that may protrude into the LVOT and causes LVOTO.  

The risk of LVOTO after TMVR may be higher in patients with pre-existing aortic valve prosthesis, because patients with prior aortic stenosis often have left ventricular hypertrophy or small LV cavity, and the frame of the aortic prosthesis can extend into the LVOT. Furthermore, the anchoring mechanism of TMVR may interfere with the proper functioning of the aortic prosthesis.  

In fact, given the complexity of our patient who had undergone previous TAViV procedure, the risk of LVOTO was even higher, and appropriate multimodality imaging assessment was crucial to have a successful procedure. Indeed, preoperative computed tomography is mandatory to plan the procedure and to assess the potential risk for iatrogenic LVOTO. CT allows determining the true inner dimensions of the prostheses, assessing the ideal site for transseptal puncture, and the best fluoroscopy angles for valve deployment.  

In our case, the simulation of a virtual valve allowed us to estimate the risk of LVOTO during the ViV procedure, by analysis of several predictors of such complication, including the aortomitral-annular angle, which is the angle between the annular planes of both valves. If the angle were obtuse, there would be a higher risk of LVOTO, since the struts of the prosthesis would encroach on the LVOT. It also allowed assessing the degree of septal hypertrophy and LV size.  

Transoesophageal echocardiography should be performed to identify the mechanism for bioprosthetic failure, assess the anatomy of the interatrial septum to evaluate the procedure feasibility, and also predict the risk of LVOTO. The mitral annulus-to-IVS distance, assessed by TEE, has shown an excellent predictive value for LVOTO, providing potential adjunctive role to CTA.  

Regarding outcomes, there is a paucity of data in the literature directly comparing redo-MVR with TMViV replacement. Kamioka et al. compared the clinical and echocardiographic outcomes between both procedures, and there were no statistical differences in mortality at 30 days and at one year. Nevertheless, TMViV was associated with a much lower rate of major bleeding and atrial arrhythmias, as well as a shorter hospital stay.  

Although TMViV is at an earlier phase of development when compared to TAViV, it is rapidly evolving as a technology that is currently becoming a solution for patients with degenerated mitral bioprosthesis at high-risk for conventional redo-MV replacement.  

This case describes an unusual clinical presentation of a severe mitral bioprosthesis degeneration, which was successfully treated with TMViV, which is a complex procedure, mainly because of the risk of LVOTO. In this case, it was even more challenging in a patient who underwent a TAViV procedure six years ago. This successful non-simultaneous double ViV aortic and then MVR, emphasize the potential of ViV procedures as an alternative for bioprosthesis degeneration in high surgical risk patients. Preoperative planning by multimodality imaging plays a key role for a safe and successful procedure, mainly in challenging cases with higher risk of complications.  

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The authors declare there are no conflicts of interest.

**CONTRIBUTION OF AUTHORS**  
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