MINIMALLY INVASIVE CARDIAC SURGERY TO REPLACE TRANSCATHETER IMPLANTED AORTIC BIOPROSTHESIS AFTER EARLY DEGENERATION. CASE REPORT

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ABSTRACT

Surgical replacement of a transcatheter implanted aortic valve prosthesis (TAVR) that presented early degeneration (in less than 24 months after its implantation) does not commonly occur in a short period of time and may be related to a worse prognosis and complications. Minimally invasive cardiac surgery can minimize surgical stress in situations where the individual undergoing it is fragile. The purpose of this article is to review the topic and describe a case of a frail 84-year-old patient who presented with valve dysfunction after TAVR and underwent surgical replacement of the bioprosthesis, through minimally invasive surgery.

KEYWORDS: POSTOPERATIVE COMPLICATIONS; AORTIC VALVE STENOSIS; THORACIC SURGERY; TRANSCATHETER AORTIC VALVE REPLACEMENT.

INTRODUCTION

Aortic stenosis is an abnormality characterized by narrowing of the aortic valve orifice, resulting in obstruction of the left ventricular outflow tract. The etiology is related to the age group of the affected population. In individuals over 70 years old, it is related to calcification, and in younger populations, it is related to the bicuspid valve. In terms of prevalence, it is present in 0.5% of the general population. [1]

As aortic stenosis treatment, surgical valve replacement (SVR) is the first-line therapy for patients with valve disease. The dysfunctional native valve is replaced with an artificial one, which can be mechanical or bioprosthetic. Additionally, transcatheter aortic valve replacement (TAVR) has emerged as a valid option for patients with severe symptomatic aortic stenosis who are appropriately selected. TAVR is an alternative to open surgery through sternotomy for patients with severe symptomatic aortic stenosis who are appropriately selected. Several randomized studies have established the superiority of TAVR for treating patients who present a prohibitive and high risk of surgical mortality and as a reasonable alternative for elderly patients with an intermediate risk of surgical mortality. [1, 2, 3, 4]

The durability of TAVR is less well-defined than SAVR. In the literature, studies on the durability of TAVR extend only to five years, a period shorter than the expected time for deterioration of valves used in SAVR. The durability of TAVR is even less defined in the population of patients with bicuspid aortic valves (BAV). [3] Valve replacement can be with a mechanical or bioprosthetic prosthesis, with each option having advantages and disadvantages in terms of durability and anticoagulation, for example. The main disadvantage of a bioprosthesis is durability, related to structural valve degeneration (SVD), a condition that eventually requires reoperation for valve replacement, a major surgical intervention. The probability of SVD is very low in the first 10 years after valve replacement in the elderly, with a gradual increase in incidence after that period. [2, 3, 5]

In the clinical case reported in this study, the insertion of a bioprosthesis through TAVR was performed on a patient who was appropriately selected according to the criteria found in the literature. The patient presented degeneration of the aortic bioprosthesis in a significantly shorter period than previously reported in the literature. This case report was approved by the Research Ethics Committee of the Hospital de Urgências de Goiás under CAAE: 38630920.7.0000.0033.

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CASE REPORT

Patient is an 84-year-old female, 1.43 m tall, weighing 55 kg, with a medical history of systemic arterial hypertension, aortic stenosis, left ventricular hypertrophy, diffuse atherosclerosis, pulmonary hypertension, peptic disease (gastritis), intestinal polyp, diverticular disease of the colon, hypothyroidism, neurogenic bladder, and difficult-to-control heart failure (pulmonary congestion and low cardiac output). She is taking Brasart HCT®, Selozok®, Trezor®, AAS®, Marevan®, Euthyrox®, Uniprost®, and Venaflon®. The patient underwent TAVR for implantation of an aortic valve bioprosthesis (Sapien S3® 20 mm) on 03/11/2021, due to severe and symptomatic aortic valve stenosis. On 22/06/2023, the patient was admitted to the emergency department with complaints of asthenia, malaise, abdominal discomfort, exertional dyspnea, upper gastrointestinal bleeding (melena), and normocytic, normochromic anemia without hemodynamic compromise, requiring transfusion. The patient had an elevated INR and Marevan® was suspended upon admission due to upper gastrointestinal bleeding.

The patient had a previous transthoracic echocardiogram performed on 23/05/23, with the following findings: aortic root diameter (Ao): 25 mm, left atrium (LA): 40 mm, right ventricular diameter (RV): 24 mm, left ventricular end-diastolic diameter (LVEDD): 43 mm, left ventricular end-systolic diameter (LVESD): 25 mm. Presence of a biological aortic endoprosthesis, with inadequate visualization of its leaflets on transthoracic examination, but presenting severe stenosis and mild to moderate intraprosthetic regurgitation (peak systolic gradient of 126 mmHg and mean of 78 mmHg, time to peak aortic flow acceleration (TA): 137 ms, TA/Left ventricular ejection time (LVET): 0.37, Doppler velocity index (DVI): 0.23, effective orifice area estimated by the continuity equation at 0.57 cm2, indexed at 0.40; aortic regurgitation parameters: vena contracta: 3.6 mm and half pressure decay time = 309 ms). Mild mitral and tricuspid valve regurgitation. High echocardiographic probability of pulmonary hypertension. On a transesophageal echocardiogram performed on 20/06/2023, the patient presented: Ao: 25 mm, LA: 36 mm, RV: 22 mm, LVEDD: 44 mm, LVESD: 27 mm. Doppler of the biological aortic prosthesis: mean gradient: 72 mmHg, peak gradient: 117 mmHg, peak velocity 5.4 m/s, DVI: 0.21, TA: 124 ms, estimated area 0.51 cm2. Turbulent flow in the right atrium, with peak velocity of 3.3 m/s, turbulent flow in the left ventricular outflow tract consistent with central aortic insufficiency. Moderate left ventricular diastolic dysfunction.

Once the dysfunction of the aortic valve bioprosthesis was evidenced - severe aortic stenosis and mild to moderate intraprosthetic regurgitation secondary to valve degeneration, associated with a history of difficult-to-control heart failure (pulmonary congestion and low cardiac output), the patient was evaluated by the cardiac surgery team and the hemodynamics team. Percutaneous treatment was considered unfeasible, and therefore, open aortic valve replacement surgery was indicated, with a minimally invasive approach (figures 01 and 02).

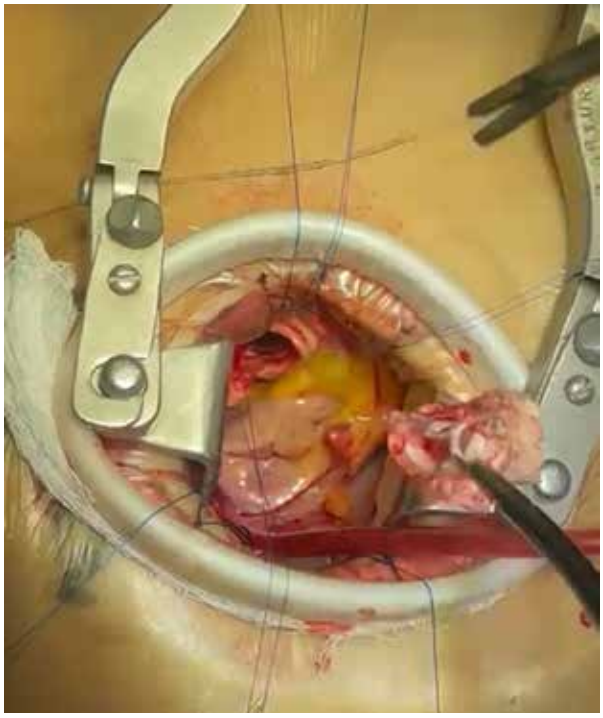

Figure 01. Minimally invasive surgery for removal of the anterior aortic valve bioprosthesis.

Figure 02. Appearance of the degenerated aortic valve bioprosthesis after its extraction.

The patient underwent successful cardiac surgery on 20/07/2023 for the implantation of aortic valve bioprosthesis no. 23 (Crown PRT®), enlargement of the aortic ring and aorta with bovine pericardium, through anterolateral/right parasternal minithoracotomy, with no complications (figure 03).

Figure 03. Implantation of the new aortic valve prosthesis through minimally invasive surgery.

The patient was monitored with a cardiac monitor, pulse oximeter, temperature probe, non-invasive blood pressure cuff, capnograph, gas analyzer, and Conox. A radial artery catheter was inserted for invasive blood pressure monitoring, a central venous catheter was placed in the subclavian vein, and a peripheral venous access was established with a 20-gauge catheter. A urinary catheter was also inserted. The patient underwent balanced general anesthesia, with intravenous induction and maintenance with an inhaled anesthetic. She was maintained on controlled mechanical ventilation in a closed circuit system with gas reabsorption.

The duration of cardiopulmonary bypass (CPB) was 156 minutes, and the aortic cross-clamp time was 119 minutes. The patient received a blood transfusion (3 units of packed red blood cells in the operating room and 8 units of cryoprecipitate). An autotransfusion system (Cell Saver) was used. A right-sided pleural effusion was identified, and thoracic drainage was performed. The patient was then transferred to the ICU while still intubated, on mechanical ventilation, hemodynamically stable, and receiving sodium nitroprusside (0.76 mcg/kg/minute). She remained stable, had effective awakening, and was successfully extubated without complications. She continued to be stable, with adjustment of antihypertensive medication, weaning off and discontinuation of sodium nitroprusside. The chest tube was removed. On 21/07/2023, she was discharged from the ICU.

A follow-up transthoracic echocardiogram on 22/07/23 showed a good surgical outcome: mild dilation of the left atrium (indexed volume = 41 ml/m2), normal dimensions of the left ventricle (LV) with preserved systolic function and moderate diastolic dysfunction. The biological prosthesis in the aortic position was functioning normally, without signs of stenosis (mean gradient = 6 mmHg, peak gradient = 11 mmHg), and no regurgitation. The mitral valve showed slight calcification of the annulus and mild regurgitation. Mild tricuspid regurgitation was also present. There was moderate dilation of the aortic root (48 mm, post-surgical enlarge ent).

DISCUSSION

Aortic stenosis is characterized by the narrowing of the aortic valve, which obstructs the blood flow from the left ventricle of the heart to the aorta. Symptoms of aortic stenosis include fatigue, heart murmur, chest pain or tightness, heart palpitations, shortness of breath, and feeling faint or dizzy with exertion. Complications of aortic stenosis can include heart failure, stroke, blood clots, endocarditis, and sudden death. [6]

In terms of pathophysiology, aortic stenosis appears to be mediated by an inflammatory process, similar to atherosclerosis, and calcification deposition can occur in the final stage of the scarring process, similar to coronary atheroma. As a result of the deposition and thickening of the valve, obstruction of the left ventricular outflow tract occurs, leading to wall hypertrophy. The myocardium becomes less compliant due to the increased diastolic pressure in the left ventricle and impaired relaxation. Pre-syncope and syncope can occur in situations of high cardiac demand, vasodilation, and arrhythmia. In severe cases, angina can occur due to increased left ventricular mass, poor coronary filling, and reduced coronary flow reserve. The risk of sudden cardiac death is proportional to the severity of the disease. [1, 6]

The use of bioprosthetic valves has been steadily increasing in the last decade, surpassing mechanical valves, with aortic valve prostheses being the most common. This trend is likely multifactorial, explained by the better hemodynamic performance of aortic bioprostheses, patient lifestyle preferences, the absence of a need for prolonged systemic anticoagulation, and the aging of the target population. [3, 4, 5] TAVI is a minimally invasive procedure in which a prosthetic valve, which will replace the damaged valve, is inserted through a catheter via access routes such as the femoral artery, subclavian artery, or common carotid artery. [6, 7]

The guidelines for reporting the outcomes of bioprosthetic valves classify the related factors affecting durability and promote valve dysfunction into BVD and non-BVD. The pathophysiology corresponds to calcific degeneration as a result of repetitive mechanical stresses. There is significant variability in the definition of BVD, still lacking

a universal definition. [3, 4] Non-BVD refers to secondary processes involving the valve, such as patient-prosthesis mismatch, valve leaflet thrombosis, endocarditis, pannus ingrowth, or paravalvular leak. BVD and non-BVD are not exclusive processes. Non-BVD mechanisms, such as patient-prosthesis mismatch, leaflet thrombosis, and paravalvular regurgitation, have been associated with accelerated BVD due to valvular hemodynamic alteration and mechanical stress. [4]

Bioprosthetic Valve Degeneration (BVD) is defined as the degeneration or intrinsic dysfunction of prosthetic valve materials. Previous studies have defined BVD as the need for reoperation due to the absence of careful and regular echocardiographic follow-up, but they do not provide specific criteria to define BVD and/or the indication for reoperation. Based on changes in transprosthetic gradients and the severity of regurgitation on echocardiography, the term "hemodynamic deterioration of the valve" has been introduced. There are several proposed definitions for bioprosthetic valve degeneration according to echocardiographic criteria, including a progression of the transprosthetic aortic gradient, leading to a mean gradient of ≥30 mm Hg associated with a reduced effective orifice area to ≤1 cm2 or intraprosthetic aortic insufficiency grade ≥3. [2-4]

There are several limitations regarding the assessment of transcatheter valve durability and the incidence of BVD in the surgical literature. This is because the absence of valve reintervention is a common clinical outcome, which underestimates the true incidence of BVD, as reoperation may not be offered to all patients and some may die before echocardiographic detection of BVD. [3,4]

A patient reported in this article underwent SAVR via minimally invasive surgery. Minimally invasive surgery has proven to be an excellent option for treating atrioventricular valve diseases. Surgeons specifically trained in this approach have achieved excellent results compared to those obtained by sternotomy, but with some advantages over conventional techniques such as: better pain control, shorter hospital stay, faster recovery time, less need for blood transfusion, lower rates of perioperative infection, less need for imaging and laboratory tests, lower rate of readmission in the first year postoperatively, better aesthetic outcome, and lower overall cost. [8,9-11]

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