REVIEW

Transcatheter Bioprosthetic Aortic Valve Durability: Where do we Stand?

Konstantinos Triantafyllou, MD,* Ilektra Papadopoulou, MD, Dimitrios Oikonomou, MD, Dimitra Benia, MD, Dimitrios Stalikas, MD, Konstantinos Kappos, MD

First Department of Cardiology, Evagelismos Hospital, *Athens, Greece* / * Email: kontriad@gmail.com

Abstract

Transcatheter aortic valve implantation (TAVI) was initially reserved for inoperable or high surgical risk patients with severe symptomatic aortic valve stenosis, but after the recent publication of randomized studies comparing TAVI to surgical aortic valve replacement (AVR) among intermediate risk (PARTNER 2 and SURTAVI) and low risk patients (PARTNER 3 and Evolut Low Risk), the momentum for further expansion of TAVI at the expense of AVR seems irreversible. The main obstacle before the wider application of TAVI for intermediate and lower risk patients is the uncertainty regarding bioprosthetic valve durability and the potential for structural deterioration and dysfunction over time. A concise overview regarding bioprosthetic valve durability issues, the relevant current scientific data and their importance for TAVI patient selection and management is herein presented. ***Rhythmos 2020;15(1):6-10.***

Keywords: TAVI; AVR; bioprosthetic valve; durability; degradation

Abbreviations: AVR = aortic valve replacement; BVF = bioprosthetic valve failure; HALT = hypoattenuated leaflet thickening; MDCT = multidetector computed tomography; PET-CT = positron emission tomography-computed tomography; PPM = patient-prosthesis mismatch; RELM = reduced leaflet motion; SVD = structural valve deterioration; TAVI = transcatheter aortic valve implantation

Introduction

During the last 20 years, the treatment of severe aortic valve stenosis (AS) has evolved dramatically. Surgical aortic valve replacement (AVR) techniques have been optimized, as with the more frequent use of new generation bioprosthetic sutureless valves which decreases significantly the aortic cross-clamp time. But the most significant evolution has undisputedly been the expansion of transcatheter aortic valve implantation (TAVI) that has revolutionized the treatment of severe AS, which is the most common valvular heart disease. Irrespective of the interventional technique selected (TAVI or AVR), any given patient older than 65 years will most probably receive a bioprosthetic aortic valve.[1] Depending on his life expectancy, the treating physician should anticipate the immutable degradation of prosthetic biological tissues and be vigilant for the advent of bioprosthetic valve structural deterioration and possible dysfunction. The medium and long-term durability of the new bioprosthetic aortic valves, especially those implanted with TAVI, constitutes a burning question and possibly the final frontier before TAVI domination over AVR for the treatment of severe AS.

Significant recent studies heralding further expansion of TAVI

The most recent valvular heart disease management guidelines recommend TAVI for patients inoperable or at very high surgical risk, but also for patients at intermediate surgical risk based on data of two recently published large randomized trials, PARTNER 2 and SURTAVI.[1]

The PARTNER 2 study randomly assigned 2032 intermediate-risk patients with severe AS to undergo either TAVI (1001 patients, mean age 81.5 years) or AVR (1021 patients, mean age 81.7 years). [2] The primary end point was death from any cause or disabling stroke at 2 years. The valve used for TAVI was the balloon-expandable Sapien XT (Edwards Lifesciences). Before randomization, patients were entered into one of two cohorts on the basis of clinical and imaging findings; 76.3% of the patients were included in the transfemoral-access cohort and 23.7% in the transthoracic-access cohort. The rate of death from any cause or disabling stroke was similar in the TAVI group and the AVR group. At 2 years, the event rates were 19.3% in the TAVI group and 21.1% in the AVR group (hazard ratio-HR in the TAVI group, 0.89, p=0.25). In the transfemoral-access cohort, TAVI resulted in a lower rate of death or disabling stroke than AVR (HR 0.79, p=0.05), whereas in the transthoracic-access cohort outcomes were similar in the two groups. TAVI compared to AVR resulted in larger aortic-valve areas and also in lower rates of acute kidney injury, severe bleeding, and new-onset atrial fibrillation, while AVR compared to TAVI resulted in fewer major vascular complications and less paravalvular aortic regurgitation.[2]

The SURTAVI study evaluated the clinical outcomes of 1660 intermediate-risk patients with symptomatic severe AS in a randomized trial comparing TAVI (performed with the use of a self-expanding prosthesis: CoreValve 86%, Evolut R 14%, Medtronic) with AVR. The mean (±SD) age of the patients was 79.8±6.2 years, and all were at intermediate risk for surgery (Society of Thoracic Surgeons – STS Predicted Risk of Mortality, 4.5±1.6%). The primary end point was a composite of death from any cause or disabling stroke at 24 months. At 24 months, the estimated incidence of the primary end point was 12.6% in the TAVI group and 14% in the AVR

group (probability of noninferiority, p >0.999). TAVI had higher rates of residual aortic regurgitation and need for pacemaker implantation, but resulted in lower mean gradients and larger aortic-valve areas than AVR, which was associated with higher rates of acute kidney injury, atrial fibrillation, and transfusion requirements. Structural valve deterioration at 24 months did not occur in either group. Consequently TAVI constitutes a non-inferior alternative to AVR, yet with differences regarding the probability of associated events.[3]

There have also been recently published data regarding the comparison of TAVI to AVR among low risk patients with severe aortic valve stenosis coming from the PARTNER 3 and Evolut Low Risk studies.

The PARTNER 3 study randomly assigned 1000 patients with severe AS and low surgical risk (mean age 73 years, mean STS risk score 1.9%) to undergo either TAVI with transfemoral implantation of a balloon-expandable valve (Sapien 3, Edwards Lifesciences) or surgery. The primary end point was a composite of death, stroke or rehospitalization at 1 year and it was significantly lower in the TAVI group than in the surgery group (8.5% vs. 15.1%; p<0.001 for noninferiority; HR, 0.54; p = 0.001 for superiority). At 30 days, TAVI compared to AVR resulted in lower rates of stroke (p=0.02), death or stroke (p=0.01) and new-onset atrial fibrillation (p<0.001), while it also resulted in a shorter index hospitalization (p<0.001) and in a lower risk of a poor treatment outcome (p<0.001). There were no significant between-group differences in major vascular complications, new pacemaker insertions, or moderate or severe paravalvular regurgitation.[4]

In the Evolut Low Risk study, TAVI with a self-expanding supra-annular bioprosthesis (Evolut R, Medtronic) was compared with AVR among 1468 randomized patients who had severe AS and were at low surgical risk. The primary end point, a composite of death or disabling stroke at 24 months, had an incidence of 5.3% in the TAVI group and of 6.7% in the AVR group (probability of noninferiority p >0.999). At 30 days, patients who had undergone TAVI, as compared with AVR, had a lower incidence of disabling stroke (0.5% vs. 1.7%), bleeding complications (2.4% vs. 7.5%), acute kidney injury (0.9% vs. 2.8%), and atrial fibrillation (7.7% vs. 35.4%) and a higher incidence of moderate or severe aortic regurgitation (3.5% vs. 0.5%) and pacemaker implantation (17.4% vs. 6.1%). At 12 months, patients in the TAVI group had lower aortic-valve gradients than those in the surgery group (8.6 mm Hg vs. 11.2 mm Hg) and larger effective orifice areas (2.3 cm^2 vs. 2.0 cm^2).[5]

Thus, both PARTNER 3 and Evolut Low risk were positive studies that reached their primary objective of non-inferiority of TAVI over AVR; furthermore, PARTNER 3 has demonstrated superiority of TAVI over AVR. Therefore, TAVI has the potential to become the treatment of reference for symptomatic severe AS instead of AVR, with the final decision to be taken in each case by the Heart Team. Severe AS is a disease of the elderly with the average patient being more than 80-year-old and the current trend is to choose TAVI as treatment for elderly active patients, with active social life demanding to keep up with their activities. However, for intermediate or low risk patients the follow-up data are relatively short and it cannot be certain that TAVI would be non-inferior to AVR in younger populations, where the probability of structural valve deterioration (SVD) and bioprosthetic valve failure (BVF) over time is a major issue. SVD is more common in younger patients, while studies of both the initial and current TAVI devices included mainly elderly patients. Logically, it will take several years and possibly a decade, before younger, lower risk patients undergoing TAVI will enable reliable long-term follow-up data collection concerning the long-term durability of TAVI devices.[6]

Bioprosthetic valve structural deterioration: definitions and diagnosis

The echocardiographic evaluation of valve morphology (morphologic deterioration) and function (hemodynamic deterioration) is the key element to evaluate if SVD exists.[7,8] SVD usually presents as leaflet calcification resulting in stenosis, but also as leaflet flail or tear resulting in regurgitation. Early SVD is associated with several well known risk factors such as young patient age, renal failure, abnormal calcium metabolism, and prosthesis-patient mismatch of the implanted valve.[9] The exact echocardiographic criteria of SVD and the relevant hemodynamic quantitative parameters were not determined until recently, when the publication of 2 papers proposed standardized definitions. Two different expert groups provided such definitions: one European (including EAPCI, ESC and EACTS) and one international (group VIVID: Valve-in-Valve International Data).[10,11]

These two papers provide definitions that have many similarities and few differences, but their in-depth analysis is beyond the scope of this article. They both recommend to perform a reference echocardiography post-implantation and a systematic echocardiographic assessment annually. This follow-up plan allows the early detection of structural deterioration signs as well as any non-structural dysfunction. Any signs of morphologic deterioration of the bioprosthetic leaflets (structural or motion abnormality) even if they are not associated with clinically significant functional degradation of the bioprosthetic valve should lead to a diagnosis of structural

deterioration. Any hemodynamic deterioration is defined as functional degradation of the bioprosthetic valve compared to the reference echocardiography, such in case of increase of the mean gradient and/or appearance (or aggravation) of intra-bioprosthetic regurgitation. The hemodynamic deterioration is classified in grades of increasing severity compared to the reference echocardiography post-implantation: moderate or severe dysfunction according to the European societies and stages 2 and 3 for the international VIVID definition.[10,11] The designation of a stage that combines echocardiographic deterioration and clinical repercussions as bioprosthetic valve failure (BVF) facilitates the study of the clinical impact of any structural deterioration.[10]

The limits of transthoracic echocardiography to detect and analyze tissue lesions of bioprosthetic valve leaflets create the necessity of using other imaging modalities. The transesophageal echocardiography allows in certain cases to objectively assess the presence of leaflet tissue lesions and rule out other alternative diagnoses. Multidetector computed tomography (MDCT) without contrast agent allows the detection of bioprosthetic leaflet calcifications which is an independent predictor of future hemodynamic deterioration but also of clinical events, such as death and re-intervention.[12,13] Furthermore, MDCT with contrast agent allows the detection of subclinical bioprosthetic leaflet thrombosis in the forms of leaflet thickening (HALT: hypoattenuated leaflet thickening) and reduced leaflet mobility (RELM: reduced leaflet motion).[14] Such lesions are more frequently observed in cases of bioprosthetic valves implanted with TAVI instead of AVR, they are mostly without symptoms, do not cause an increase of the mean gradient and are reversible after some weeks of therapeutic oral anticoagulation.[15] However, since there is a continuum linking leaflet thrombosis and structural deterioration the persistence of this type of lesions despite curative anticoagulation for more than 3 months should be considered as SVD.[16] Finally nuclear imaging with positron emission tomography - computed tomography (PET-CT) using ^{18}F-fluoride as tissue activity marker can predict future development of SVD, but an increased uptake can also signify the presence of pannus or thrombus.[17] According to the recently published standardized definitions cardiac imaging thus permits to classify SVD in 4 stages (Table 1).[10,11]

In *conclusion*, the annual follow-up with echocardiography is the basic and only examination to analyze valvular hemodynamic conditions, while MDCT and PET-CT can have in occasional cases a complementary role in order to add detail to the morphologic evaluation and do the differential diagnosis of BVF. Clinical criteria should obviously be considered to assess the impact of BVF (such as the presence of dyspnea, rehospitalization for heart failure, reintervention for valve dysfunction and death).

Table 1. Classification of structural valve deterioration

Stage 0	Absence of morphologic abnormality and stability of hemodynamic parameters
Stage 1	Morphologic abnormalities without significant hemodynamic deterioration
Stage 2	Morphologic abnormalities with moderate hemodynamic deterioration (stenosis and/or regurgitation)
Stage 3	Morphologic abnormalities with severe hemodynamic deterioration (stenosis and/or regurgitation)

Bioprosthetic valve structural deterioration: pathophysiology and mechanisms

Data related to the pathophysiology of SVD are mainly derived from studies concerning AVR. The mechanical stress on the bioprosthetic valve leaflets is an important factor that determines the speed of SVD. Some bioprosthetic valve designs related to suboptimal hemodynamic results post-intervention show a tendency for faster degeneration.[13] Patient-prosthesis mismatch (PPM) is an important factor promoting early leaflet calcification, hemodynamic degeneration and the need for secondary reintervention due to severe structural dysfunction.[12,13,18,19] As the severity of PPM increases the risk of hemodynamic deterioration becomes more and more important.[13] Leaflet calcification is the main lesion observed in SVD and is the result of an active mineralization phenomenon as shown in histopathologic studies.[20] The absence of anti-calcification treatment of the valve and the presence of dysregulation of calcium and phosphorus metabolism participate in this process.[12] Renal insufficiency and an abnormal renal clearance similarly promote early SVD.[13] Metabolic abnormalities such as diabetes mellitus, dyslipidemia and metabolic syndrome are strongly correlated to a risk of early SVD. Histopathologic studies of explanted valves have confirmed the presence of leucocytes and macrophages and a lipid-mediated inflammation in degenerated leaflet lesions suggesting an active immune mechanism resembling that of atherosclerosis.[21]

Common mechanisms seem to pertain to bioprosthetic valves implanted percutaneously but several particularities related to their design and implantation technique may be specifically implicated in the SVD process post-TAVI. The percutaneous implantation technique depending on the valve type includes crimping, charging of the valve in the implantation system, implantation of the valve by balloon dilatation and possible optimization with post-

dilatation. All these steps can lead to alteration of the prosthetic leaflets mechanical properties and cause micro-lesions secondary to valve manipulation that predispose to subclinical leaflet thrombosis.[22] In certain native aortic valve anatomies with severe calcification the bioprosthetic valve can be deployed in a non-circular and irregular fashion leading to areas of turbulent flow or more important mechanical stress upon the leaflets and other areas where to the contrary a certain degree of stasis can predispose to thrombosis.[23,24] Significant oversizing of the bioprosthetic valve compared to the native annulus dimensions can lead to incomplete expansion with a disproportion between leaflets and orifice having as consequences increased mechanical stress and turbulent transvalvular flow.[24] All these implantation issues specific to TAVI can explain the increased incidence of subclinical thromboses compared to AVR.[15] The link found by histopathologic studies between thrombosis and structural deterioration suggests a theoretical increased risk for SVR after TAVI.[16,17] However this theoretical increased risk is not yet proven in studies.

Bioprosthetic valve durability

The durability of surgical bioprosthetic valves depends on the advent of structural deterioration, its severity and its clinical impact. The new generations of surgical bio-prosthetic valves demonstrate excellent durability with rates of re-intervention 2-10% at 10 years, 10-20% at 15 years and ~40% at 20 years.[25] However, the incidence of at least moderate (stage 2) hemodynamic deterioration detected by echocardiography seems to be more important, ~40% for the 10 first years after implantation.[13] The durability of percutaneously implanted bioprosthetic valves is excellent at short- and medium-term and comparable to surgical ones, with comparative studies of hemodynamic deterioration providing reassuring results.

It should be underlined that data regarding long term durability are limited by the short follow-up of patients treated with TAVI since initially the technique was applied to elderly patients with many comorbidities having survival rates at 5 years around 50%.

Recent studies (Table 2) interrogating the long-term durability of percutaneously implanted bioprosthetic valves report a moderate hemodynamic deterioration rate of 4.5-15.8% between 5 to 8 years post TAVI.[26-35]

Blackman et al reported an incidence of moderate and severe SVD of 8.7% and 0.4% respectively at 5-10 years post-intervention.[35] A recent analysis of NOTION study has found less moderate or severe SVD at 6 years after TAVI compared to AVR (4.8% vs 24% respectively), while there was no difference in BVF rates (7.5% vs 6.7%; p=0.89).[34]

Table 2. Studies of TAVI bioprosthetic valves durability

Author / year	No. of Pts	Median FU (y)	Valve types	Moderate or severe structural valve deterioration (SVD)
Søndergaard et al.[34] / 2019	139	6	CoreValve	4.8%
Durand et al.[29] / 2019	1043	3.9	Edwards: 70 Sapien: 475 Sapien XT: 512 CoreValve:199 Jena: 7	11.2% (at 7 years)
Kumar et al.[33] / 2019	276	3.8	Edwards: 7 Sapien: 244 Sapien XT: 25	8.3% (at 5 years)
Blackman et al. [35] / 2019	241	5.8	Sapien: 45 Sapien XT: 35 CoreValve: 149 Portico : 214	9.1%
Holy et al.[32]/ 2018	152	5	CoreValve	7.9% (actuarial) & 4.5% (actual) at 8 yrs
Barbanti et al.[26] / 2018	288	6,7	CoreValve: 238 Sapien XT: 48	10.38% at 8 years
Deutsch et al.[27] / 2018	300	7.1	CoreValve: 214 Sapien: 86	14.9% at 7 years
Gleason et al.[31] / 2018	391	4.2	CoreValve	10% at 5 years
Didier et al.[28] / 2018	4201	-	Sapien:2774 CoreValve: 1413	15.8% at 5 years

A multicenter French study that included 1403 TAVI patients has used the newest European definitions for bioprosthetic valve deterioration and failure and found a BVF incidence of 1.9% at 7 years and of moderate or severe SVD at 7% and 4.2% respectively.[10,28]

These numbers are reassuring but they should be confirmed by large randomized studies of TAVI with long follow-up among intermediate to low risk patients. On final analysis the choice of bioprosthetic valve should be based on the patient's life expectancy and the proven durability of the bioprosthetic valve, which should exceed the patient's life expectancy at the time of implantation.

Conclusion

Recent randomized studies support the use of TAVI for the treatment of symptomatic severe AS among intermediate and low risk patients, since it has been shown to be at least non inferior compared to AVR. Bioprosthetic valve deterioration and failure over time has been an important concern for surgical valves and is a major issue before TAVI widely expands to treat younger and low risk patients with prolonged life expectancy. Recent consensus papers proposed standardized definitions for bioprosthetic valve deterioration and dysfunction, which will positively affect the follow-up of TAVI and AVR patients with echocardiography (and when necessary MDCT or PET-CT), help better understand pathophysiologic mechanisms and most importantly help determine the true incidence of SVD and BVF. Several initial studies provide reassurance

regarding the durability of bioprosthetic valves implanted with TAVI, but larger studies and several years will be still needed in order to have definitive answers.

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