Left Ventricular Diastolic Dysfunction and Transcatheter Aortic Valve Replacement Outcomes: A Review

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ABSTRACT

Aortic stenosis (AS) is the most common valvular disease that can lead to increased afterload, left ventricular (LV) remodeling, and myocardial fibrosis. We reviewed the literature addressing the impact of transcatheter aortic valve replacement (TAVR) on LV remodeling and patients' outcomes by elimination of AS-related high afterload. TAVR reduces afterload and improves LV remodeling recovery. However, myocardial fibrosis may not completely reverse after the TAVR. The LV diastolic dysfunction (LVDD) induced by AS is an independent predictor of post-TAVR mortality, and mortality increases with severity of LVDD. The impact of diastolic dysfunction on patient outcomes emerges at 30 days but continues to persist during mid-term follow-up. Based on severity of the baseline LVDD, some patients may tolerate post-TAVR aortic regurgitation (AR), but even minimal post-TAVR AR in patients with severe baseline LVDD can have an additive negative impact on survival. It is crucial to consider TAVR prior to development of advanced LVDD. Appropriate device selection and deployment technique are important in improvement of TAVR outcomes via elimination of AR.

Keywords: Aortic regurgitation; Aortic stenosis; Heart valve prosthesis; Heart valve replacement; Left ventricular afterload; Left ventricular remodeling; Myocardial fibrosis

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INTRODUCTION

Aortic valve stenosis (AS) is the most common valvular disease in developed countries. AS is a progressive disease and once it becomes symptomatic, the mortality rate can be as high as 68% at 2 years in patients who receive medical therapy or balloon aortic valvuloplasty with no valve replacement [1]. Patients with AS usually have a long asymptomatic phase and then develop a short symptomatic phase. It was reported that the outcome of AS patients is significantly associated with timing of afterload elimination [2]. Aortic valve replacement is not usually considered for patients with asymptomatic AS, while left ventricle (LV) remodeling and myocardial fibrosis secondary to AS can begin in the asymptomatic phase. The LV

remodeling leads to diastolic dysfunction (LVDD), which affects the outcome of patients with AS who undergo aortic valve replacement [3]. Transcatheter aortic valve replacement (TAVR) is accepted as an appropriate treatment approach for inoperable, high-risk, and intermediate risk patients who are not eligible for surgical aortic valve replacement [4, 5]. TAVR was shown to improve cardiac function and patients' outcomes, but not all effects of prolonged AS on LV [6]. In this article, we aimed to review the clinical aspects of AS-related LVDD and its impact on patients' outcomes. We will also review the role of TAVR in the recovery of LVDD and procedural factors that can influence patients' outcomes.

METHODS

The current study is based on literature review, and no direct human or animal intervention was performed for this report. We searched PubMed, Google Scholar, and Google to find appropriate studies. Search terms were 'transcatheter aortic valve replacement' or 'TAVR' in combination with 'left ventricular diastolic dysfunction' or 'LVDD.' There was no time or geographic limit in the search strategy and articles that were published by the end of December 2018 were eligible. All available studies were reviewed, and applicable results were used for the current review article. This article is based on previously conducted studies and does not contain any studies with human participants or animals performed by any of the authors.

AS AND DIASTOLIC DYSFUNCTION

Mechanical obstruction of LV secondary to AS increases LV afterload pressure, leading to compensatory cardiomyocyte hypertrophy and collagen network abnormality, which result in myocardial fibrosis and eventually LVDD [7–9]. As the duration of high afterload pressure prolongs, severity of LVDD progresses and myocardial fibrosis becomes more prominent, which increases the risk of irreversibility of unfavorable LV remodeling [10]. It was shown that LV remodeling-induced myocardial fibrosis is the main component of transition from compensatory hypertrophy to heart failure (HF) in AS patients [8, 9]. In addition to lower ejection fraction (EF), higher pulmonary artery pressure and more mitral or tricuspid valve regurgitation were also found among AS patients with LVDD [11, 12]. The myocardial fibrosis itself was found to be an independent predictor of mortality in AS patients [8]. It was reported that up to 67% of patients with severe AS who undergo TAVR have some degree of LVDD [11]. Although TAVR was suggested to be an effective approach for afterload and wall stress reduction [13], some degree of LVDD may persist after aortic valve replacement [3].

LVDD IMPROVEMENT AFTER TAVR

Elimination of AS-induced afterload by TAVR can improve cardiac function in an acute phase and also reverse LV remodeling in a slower process [13]. In general, myocardium hypertrophy regresses much faster than the fibrotic tissue, and fibrosis reduction may happen during the delayed phase, if at all [14]. In a study using magnetic resonance imaging, it was reported that myocardial fibrosis does not recover until 9 months after the afterload elimination [15]. Consistently, significant LV mass reduction was seen at 6 and 12 months after TAVR [16, 17]. Patients with higher LV mass regression were found to have a 50% lower readmission rate within the first year after TAVR [18]. However, the LV mass reduction does not necessarily lead to complete LV diastolic function improvement because LV mass decrease is slow and continues in a nonlinear fashion. The fibrotic component of the LV mass may take several years to regress after the TAVR and can even become permanent [14, 19, 20].

There are controversial reports about changes in diastolic function parameters (lateral e' velocity, E/lateral e', and left atrium volume index (LAVI), septal e', and E/A ratio, and LV

mass) after TAVR [21, 22]. Pre-procedural E/e' was found to be a good measure of LV end diastolic pressure (LVEDP) and an excellent predictor of poor outcome and cardiac function in patients who underwent surgical aortic valve replacement [21]. This ratio might not be generalizable to TAVR patients due to a greater age-related mitral annulus calcification. Asami et al. have shown that despite improvement of individual diastolic parameters within 6–18 months after TAVR, the overall LVDD grade does not change in at least 50% of patients [11]. Although in a study by Blair et al. the number of patients with grade III LVDD at 30 days after TAVR was less than that at baseline, some (lateral e' velocity, $E/$lateral e', and LAVI) but not all (LV mass, septal e', and E/A ratio) diastolic dysfunction parameters improved after TAVR [12]. The authors suggested that in patients with prolonged severe AS, LVDD can improve after TAVR but does not normalize due to sustained LV stiffness from myocardial fibrosis. Even after TAVR, the myocardial fibrosis can increase the risk of arrhythmia and sudden cardiac death [23]. This indicates the importance of timing for elimination of afterload by TAVR in high-risk and inoperable patients. Early TAVR, especially in patients with baseline LVDD, might prevent additional LV fibrosis and increase the odds of potential LV recovery.

TAVR can decrease left atrium volume and improve transmitral filling and mitral annular tissue Doppler velocity in early diastolic phase [12]. Severe mitral-valve regurgitation was reported in up to 49% of AS patients before TAVR and up to 29% of patients after TAVR [24]. A prospective study showed that afterload elimination by aortic valve replacement improves mitral valve regurgitation, especially in those with functional mitral regurgitation rather than myxomatous degeneration. The lower EF and larger LV mass were found to be associated with post-procedure reduction in degree of mitral regurgitation [25, 26]. Despite improvement in diastolic parameters, the degree of mitral regurgitation did not improve after TAVR in the study by Blair et al. possibly because of the advanced age of the study patients [12].

LVDD AND POST-TAVR OUTCOME

Hospital readmission within 1 year after TAVR was found to be significantly associated with mortality. The most common reason for hospitalization after TAVR was HF [27, 28]. Baseline LVDD was found to play an important role in sustaining HF after TAVR [10]. Hospitalization duration increases with worsening of LVDD grade [11]. A higher rate of mortality was seen at 30 days in patients with LVDD, but the difference in cardiovascular mortality rate continues to be significant at mid-term follow-up, regardless of patients' LVEF [11]. Blair et al. found that patients with grade 1a LVDD are not at increased risk of mortality after TAVR, and poor outcome emerges when LVDD grade progresses to grade 2 [12]. However, the grade 1a only existed when diastolic dysfunction was classified based on the Kuwaki et al. grading system (Table 1) [29, 30]. Baseline LVDD grade 3 was reported to be the strongest predictor of all-cause mortality at 1 year [11]. Chin et al. found that a higher mortality rate in patients with worse LVDD grade is due to a higher component of myocardial fibrosis in these patients [8]. Importantly, it was reported that 1-year mortality increases by 16.3% for each LVDD grade worsening [12]. Kampaktsis and colleagues also found a higher mortality rate in patients with severe LVDD (29%) versus those with moderate or mild LVDD (19%), but the difference in mortality rates was not statistically significant in their study [31]. Although LVDD grade can change after TAVR, no significant association was found between post-TAVR LVDD and mortality [12].

Muratori et al. did not find any association between baseline LVDD and 1-year mortality, despite improvement in New York Heart Association (NYHA) class and LVDD following TAVR [32]. Initial improvement in NYHA class may not persist, as the study found a higher proportion of patients with NYHA class III and IV at 3 years compared with 1 year after TAVR. The worsening of NYHA class can originate from patients' old age and other baseline comorbidities [33, 34].

Table 1 Diastolic dysfunction classification based on Kuwaki et al. and American Society of Echocardiography (ASE) and European Association of Cardiovascular Imaging (EACVI) systems

Grading system	Grade 0	Grade 1	Grade 1a	Grade 2	Grade 3
Kuwaki et al.					
E/A	$0.75 <$ to < 1.5	≤ 0.75	≤ 0.75	$0.75 <$ to < 1.5	≥ 1.5
DT	> 140 ms	> 140 ms	> 140 ms	> 140 ms	≤ 140 ms
E/e'	< 10	< 10	≥ 10	≥ 10	≥ 10
ASE/EACVI			–		
Septal e'	≥ 8	< 8		< 8	< 8
Lateral e'	≥ 10	< 10		< 10	< 10
Left atrium volume	< 34 ml/m^2	≥ 34 ml/m^2		≥ 34 ml/m^2	≥ 34 ml/m^2
E/A	–	< 0.8		$0.8–1.5$	≥ 2
DT	–	> 200 ms		$160–200$ ms	< 160 ms
Av E/e'	–	≤ 8		$9–12$	≥ 13
Ar-A	–	< 0 ms		≥ 30 ms	≥ 30 ms
Val $\Delta E/A$	–	< 0.5		≥ 0.5	≥ 0.5

E early mitral inflow velocity, *A* duration of the pulmonary flow reversal, *DT* E wave velocity deceleration time, *e'* early diastolic mitral annular velocity, *Av* average, *Ar* pulmonary venous atrial flow reversal, *Val* Valsalva maneuver

IMPACT OF PROSTHESIS-PATIENT MISMATCH AND AORTIC REGURGITATION ON LVDD AND PATIENTS' OUTCOMES

Prevalence of post-TAVR paravalvular regurgitation (PVR) was estimated as high as 100%, with up to 39% moderate to severe PVR among patients who underwent TAVR using an early generation valve [35, 36]. Fortunately, the rate of PVR ≥ moderate has decreased significantly, even to 0%, with newer- generation valves [37]. PVR increases LVEDP, leading to greater hemodynamic decompensation [38]. The LV of patients with diastolic dysfunction does not have the ability to increase dimensions and compliance for acute post-TAVR PVR, leading to a very high elevation in LVEDP [39]. Hence, post-TAVR PVR can exacerbate the baseline HF, adversely affect LV remodeling, and have an additional negative impact on mortality after TAVR (Fig. 1). Controversial studies were reported about the impact of different degrees of PVR on outcomes after TAVR [31, 40]. Sato et al. found increased LVEDP among patients who died within 1 year after TAVR, and presence of post-TAVR aortic regurgitation was the only independent predictor of mortality at 1 year [20]. Halkin et al. did not find any significant association between mild PVR and post-TAVR mortality, but moderate to severe PVR was shown to be an independent predictor of all-cause mortality at mid-term follow-up (30 months) [22]. They found that deceleration time (DT) of early filling velocity < 160 ms (suggestive of severely impaired LV diastolic filling) is an independent predictor of mortality in patients with mild and moderate to severe PVR. It was suggested that baseline LVDD plays an important role in exacerbation of post-TAVR PVR-related volume overload, and makes PVR an independent predictor of mortality [4]. The presence of PVR ≥ mild after TAVR is associated with increased mortality, with up to four times increased risk of mortality at 2 years among
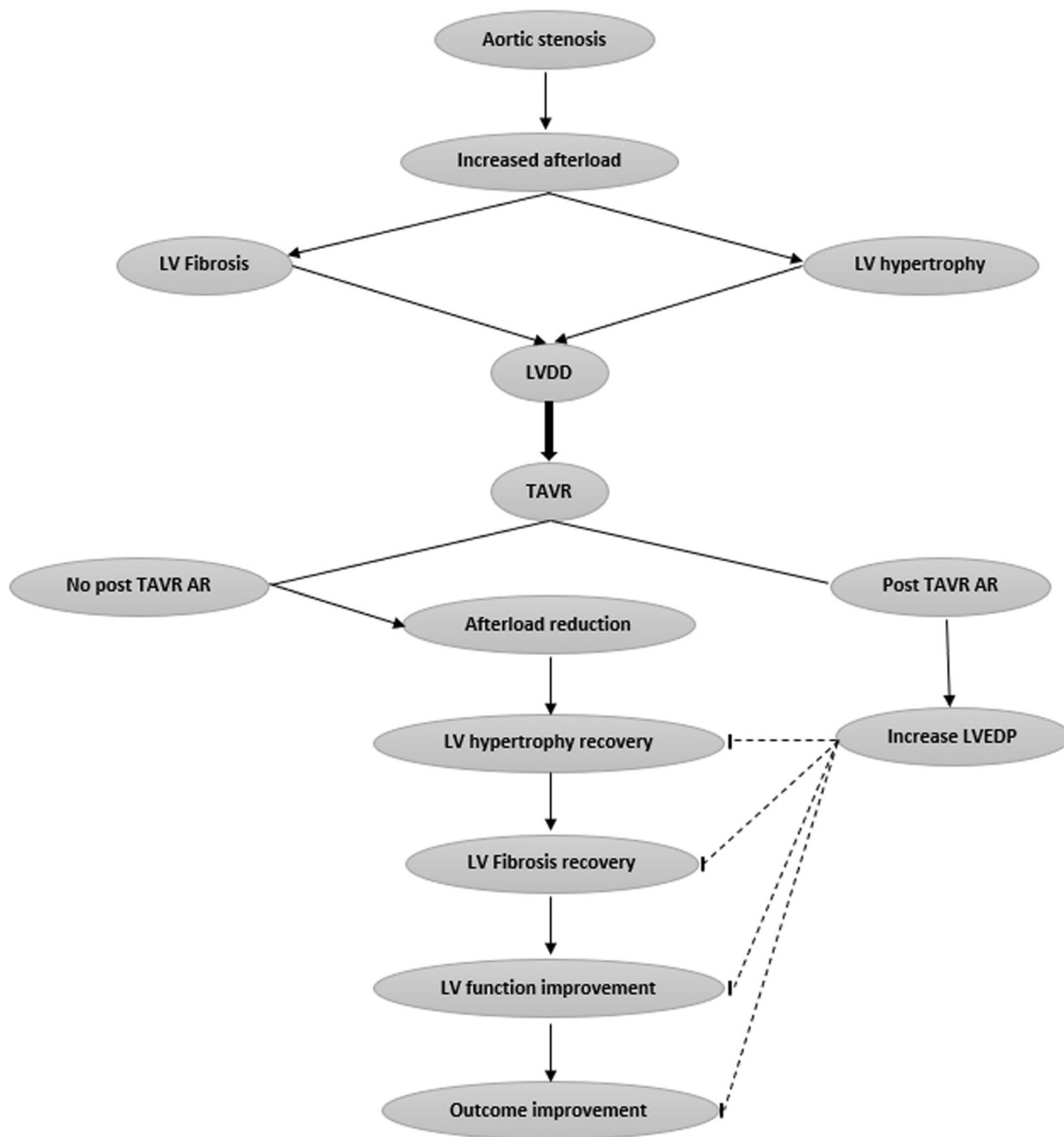
Fig. 1 Impact of transcatheter aortic valve replacement (TAVR) and aortic regurgitation (AR) on left ventricular diastolic dysfunction (LVDD) and patients' outcomes. *LVEDP* left ventricular end diastolic pressure. The arrows show stimulatory effect and the flat heads show inhibitory effect

those with severe baseline LVDD. The degree of LVDD alone (without post-TAVR PVR) was not found to be significantly associated with mortality [31]. The difference in impact of various degrees of PVR on TAVR outcome originates from baseline LVDD. In patients with severe LVDD, even trace PVR can increase LVEDP and deteriorate heart function, increasing mortality, but those with more compliant LV may better tolerate higher degrees of PVL [40]. Post-TAVR

PVR was found to be related to device size and form, patient native valve and LV outflow tract anatomy, and technical issues [22]. Therefore, it is important to use the most appropriate TAVR device with the lowest reported degree of PVL. It is also crucial to assess baseline diastolic function before TAVR and have a meticulous deployment technique to prevent any PVL. Post-deployment ballooning may be beneficial in elimination of observed PVL in selected cases.

Post-TAVR prosthesis–patient mismatch (PPM) incidence was reported up to 42%, with a severe PPM rate of 9% [6, 41]. A negative impact of PPM on LV remodeling and function was reported. Although a higher rate of post-TAVR PVR was found in patients without PPM than in those with PPM (41 versus 17%, $p = 0.01$), higher diastolic function improvement and more LV mass regression was found in the no-PPM group versus the PPM group. However, no significant difference was found in midterm survival rates between PPM and no-PPM groups [41]. In one report, PPM occurred most commonly in patients who underwent TAVR with smaller-size prosthetics (Sapein and CoreValve size < 29 mm) [41]. On the other hand, it was suggested that patients with a larger aortic annulus can have suboptimal valve deployment due to less prosthesis–annulus congruence, leading to post-TARV PVR [42]. The presence of PVR after TAVR restrains any benefits of PPM absence in improvement of LVDD [43]. As TAVR use is expanding to low-risk and young patients, elimination of both PPM and AR by appropriate valve selection and deployment technique is ideal. However, since post-TAVR PVR is an independent predictor of mortality in patients with baseline LVDD, and PPM is not, prevention of AR might outweigh prevention of PPM.

CONCLUSIONS

LVDD plays an important role in patient outcomes with TAVR. Progression of LVDD to an advanced stage in patients with AS increases fibrotic tissue in the LV and decreases chance of LV recovery after the TAVR. Further, patients with severe LVDD may not tolerate minimal PVR and deteriorate after TAVR, but those with close to normal LV diastolic function can tolerate significant amounts of PVR. Therefore, evaluation of LVDD severity along with AS assessment, and consideration of TAVR prior to significant LVDD development with an appropriate valve and deployment technique, can potentially eliminate post-TAVR PVR and improve patient outcomes.

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