

Original Research Article

THE EFFECT OF TAVI ON LEFT ATRIAL FUNCTION AND HEMODYNAMICS

ABSTRACT

Introduction:

Aortic stenosis (AS) is a highly prevalent and clinically relevant cause of valvular heart disease worldwide, and, if left untreated, leads to substantial morbidity and mortality. This study provides further evidence in support of TAVI as a transformative therapy for severe AS and highlights the importance of comprehensive assessment of the effects of TAVI on both left and right heart function.

Study design:

Prospective observational monocentric study

Place and duration of study:

Apollo Hospitals, Chennai on 30 patients with severe aortic stenosis who have TAVI from April 2022 to October 2023

Methodology:

This prospective study included 30 patients with symptomatic severe aortic stenosis (AS) who underwent baseline transthoracic echocardiography (TTE) and transfemoral transcatheter aortic valve implantation (TAVI). Patients were stratified into two groups based on left ventricular ejection fraction (LVEF $\geq 50\%$ and LVEF $< 50\%$) for comparative analysis. Comprehensive echocardiographic and Doppler assessments followed standardized guidelines, including evaluations of left ventricular mass, stroke volume, and diastolic function. Left atrial function was

assessed using speckle tracking echocardiography (STE) to evaluate reservoir, conduit, and contractile functions. Inter- and intraobserver variability analyses ensured the reliability of strain measurements. Statistical analysis was performed using SPSS (IBM, 28.0). Summary statistics were presented with Mean \pm SD and frequency (percentage) for the continuous and categorical factors respectively. Chi square/Fisher's exact test was used to determine the association between two independent categorical factors. *P*-value <0.05 was considered statistically significant. Ethical clearance was obtained from the Institutional Review Board for the conduct of the study. (IEC: AMH - DNB-081/11-22).

Results:

Our study demonstrated significant improvements in left ventricular (LV) systolic and diastolic function three months after transcatheter aortic valve implantation (TAVI), with notable increases in global longitudinal strain (GLS) and reductions in LV mass and filling pressures. Left atrial (LA) function improved, with significant enhancements in reservoir and conduit functions and reductions in LA volumes. Hemodynamic parameters, including aortic valve area and mean gradient, showed substantial recovery. LVEF exhibited modest improvement compared to strain parameters, emphasizing the importance of advanced echocardiographic techniques. These findings underscore the role of TAVI in promoting reverse remodelling and improving myocardial and atrial mechanics in high-risk severe AS patients.

Conclusion:

TAVI is associated with significant improvements in systolic and diastolic LV function, LA remodelling, and overall hemodynamics in high-risk patients with severe AS.

Keywords

Left Atrial Strain, Aortic Stenosis, TAVI, Paradoxical Low Flow

INTRODUCTION

Aortic stenosis (AS) is a highly prevalent and clinically relevant cause of valvular heart disease worldwide, and, if left untreated, leads to substantial morbidity and mortality.¹ Described as a chronological reduction of the aortic valve, severe AS is defined by an effective orifice area (EOA) of less than 1.0 cm² and a mean transvalvular gradient of at least 40 mmHg.² It has become a growing burden on healthcare systems due to its high prevalence, especially in older generations, as well as its major engulfment of cardiac pumping ability and patient quality of life.¹ Although the pathophysiology of AS is focused on the left ventricular (LV) outflow obstruction, it has widespread effects on the structure and function of the heart.^{1,2} The elevated pressure gradient across the stenotic valve leads to chronic pressure overload and induces compensatory hypertrophic remodelling of the left ventricle. Over time, these changes extend to the left atrium (LA) as the chamber remodels to compensate for elevated filling pressures and reduced compliance of the left ventricle. Altered LA function is a prevalent consequence of severe AS and has been linked to worst prognosis, including heart failure and atrial arrhythmias.

Nonetheless, some heterogeneity exists and patients with severe AS are comprised of relatively distinct subgroups which demonstrate unique hemodynamic and functional characteristics. One example of such a group is those with reduced left

ventricular ejection fraction (LVEF) indicating systolic dysfunction.³ A distinct cohort falls under the category of paradoxical low-flow (PLF) AS, characterized by normal LVEF but reduced stroke volume and relatively moderate pressure gradients.⁴ This condition has emerged as a marker of late disease with a prognosis more grim than that of standard high-gradient AS with normal flow. These subgroups remain challenging to manage, despite advances in diagnostic and therapeutic approaches, highlighting the need for more exploration of their unique pathophysiological profiles and treatment responses.

Transcatheter aortic valve implantation (TAVI) has become an innovative treatment option for severe AS, enabling less invasive valve replacement therapy that has been demonstrated to be beneficial in high-risk or inoperable patients.⁵ TAVI has proven significant mortality and quality of life benefits in patients with severe AS. TAVI significantly enhances survival compared to medical management and has been shown in select populations to be non-inferior to surgical aortic valve replacement. Although the hemodynamic benefits seen with TAVI (eg, decreased transvalvular gradients and increased EOA) are well known, its effects on myocardial mechanics and atrial function, particularly at longer-term follow-up, are poorly characterized.⁶

Left atrial function assessment is one of the most important but frequently neglected part of cardiac evaluation in AS patients. The LA has three main functions; as a reservoir during ventricular systole, as a conduit for passive LV filling in early diastole, and as an active pump during atrial systole. Chronic pressure overload associated with AS can affect all three pillars of LA function, resulting in elevated

filling pressures, decreased cardiac output and increased propensity for arrhythmias, including atrial fibrillation.⁷ Although benefits to LA performance have been observed after surgical valve replacement in certain cohorts, the degree and timing of changes in LA function after TAVI, especially in high-risk populations, has not been established. In this regard, speckle tracking echocardiography (STE) represents an important advancement in the evaluation of myocardial mechanics and provides a foundation for robust measurements of strain and deformation. STE offers insight into LA function and can detect small changes in the performance of the reservoir, conduit, and contractile components of the LA that may not be apparent with traditional echocardiographic parameters. Analysis of STE is valuable in observing diminished longitudinal strain further revealing the myocardial impairment that is the most significant in this group of patients with aortic stenosis.^{7,8}

The present study sought to evaluate the impact of TAVI on left atrial function and hemodynamics after 3 months of follow-up using advanced echocardiographic techniques to assess strain-derived parameters. This study aims to cover such a gap by (i) looking at a wide patient population with severe AS, and at the same time, (ii) by including patients with normal flow - and preserved left ventricular ejection fraction (LVEF), (iii) patients with post-TAVI low flow and (iv) low LVEF, thus providing clinical and pathophysiological insights on myocardial and hemodynamic adaptations after TAVI. In particular, it investigates whether TAVI is associated with recovery of LA function across its various functional components, and whether this such recovery is heterogeneous, depending on baseline hemodynamic and functional characteristics. Appreciating the influence of TAVI on LA performance is not simply an academic pursuit but pertains to important clinical implications. An enhanced

understanding of LA mechanics after valve intervention may help in refining patient selection, is a potential marker for post-procedural management, and possibly prognosticate in AS patients on a long-term basis. Consequently, this study provides further evidence in support of TAVI as a transformative therapy for more severe AS and highlights the importance of comprehensive assessment of the effects of TAVI on both left and right heart function.

Material and Methods

This prospective study included 30 patients with symptomatic severe aortic stenosis (AS) who underwent baseline transthoracic echocardiography (TTE) and subsequent transfemoral transcatheter aortic valve implantation (TAVI) at our institution. Patients were eligible if they fulfilled diagnostic criteria for severe AS, which was defined as an aortic valve area (AVA) ≤ 1.0 cm², and were deemed high-risk surgical candidates as per a European System for Cardiac Operative Risk Evaluation (EuroSCORE) score $>20\%$. Informed written consent was acquired from all participants, and the project was approved by Institutional Ethics Committee.

The study population was stratified by left ventricular ejection fraction (LVEF). Group 1 patients had preserved LVEF ($\geq 50\%$) and Group 2 had reduced LVEF ($<50\%$) The current classifications facilitated tailored comparisons of the impacts of TAVI on LA function and hemodynamics between different groups of baseline cardiac function.

Routine echocardiographic and Doppler assessments were performed at baseline and during follow-up, following a standardized protocol in accordance with the American Society of Echocardiography (ASE) guideline. These included the Ejection fraction(EF), stroke volume (SV) and stroke volume index (SVI), obtained from the

pulsed-wave Doppler at the left ventricular outflow tract (LVOT) and indexed to BSA; and severity of aortic regurgitation (AR) and mitral regurgitation (MR), scored according to guidelines from the European Association of Cardiovascular Imaging. In this analysis, LV diastolic function was evaluated by pulsed-wave and tissue Doppler imaging parameters.

Left atrial function was assessed by speckle tracking echocardiography (STE) according to its three main components: reservoir function, conduit function, and contractile function. Reservoir function was defined as the maximal positive deformation during ventricular systole, whereas conduit function was determined as the difference between reservoir strain and strain during the early diastole. Contractile function was calculated as the difference between early diastolic strain and atrial contraction strain. The images for the strain analysis were obtained in apical two-, three-, and four-chamber views at 60–80 frames per second; offline analysis was conducted using a single semi-automated algorithm to maintain standardization.

To validate the fidelity of strain measurements, both inter- and intraobserver variability were assessed. Strain analysis was performed by 2 independent blinded echocardiographers (using the methodology described above), allowing for interobserver variation data. Intraobserver variability was assessed by repeated measurements on different days by an experienced echocardiographer, expressed as intraclass correlation coefficients.

Statistical Data obtained was entered into excel sheet (MS office 2021). All continuous variables were assessed for normality using Shapiro–Wilk's test and

Continuous variables following Gaussian distribution were expressed as mean \pm standard deviation (SD). Otherwise, they were expressed as median (inter quartile range). Comparison of continuous variables was done using independent sample t test and Comparison of categorical variables was taken care of by Chi – square test or Fisher’s Exact test based on the number of observations. Univariate logistic regression analysis was used to determine the influence of the independent factors on the outcome parameter. All p-values less than 0.05 were considered as statistically significant. Data analysis was carried out by SPSS version 25.0

Results

The baseline characteristics of 30 patients who were divided into two groups based on left ventricular ejection fraction (LVEF \geq 50% and LVEF <50%) (Table 1, Figure 1). The mean age of patients in the reduced LVEF group (LVEF \leq 50%) was slightly higher at 75.73 ± 7.33 years compared to 73.53 ± 8.74 years in the preserved LVEF group (LVEF >50%), though this difference was not statistically significant ($p = 0.48$). Gender distribution revealed that all patients in the reduced LVEF group were male, whereas the preserved LVEF group included 26.3% females and 73.7% males, a difference approaching but not reaching statistical significance ($p = 0.06$). The mean EUROSCORE of the study population was 14%.

Coronary artery disease (CAD) was significantly more prevalent in the reduced LVEF group, with 100% of patients affected compared to 47.4% in the preserved LVEF group ($p = 0.003$). Patients with the recent history of ACS were excluded from the study and the one with known CAD and reduced EF were continued on GDMT. Diabetes mellitus was also more common in the reduced LVEF group (63.6%)

compared to the preserved LVEF group (21.1%), showing a statistically significant difference ($p = 0.02$). In contrast, arterial hypertension, chronic obstructive pulmonary disease (COPD), and smoking were similarly distributed between the groups, with no significant differences observed.

Pulmonary hypertension severity differed significantly between the groups ($p = 0.009$). While 36.4% of patients in the reduced LVEF group had moderate pulmonary hypertension and 18.2% had severe pulmonary hypertension, none of the patients in the preserved LVEF group had severe pulmonary hypertension, and only one had moderate pulmonary hypertension. Chronic kidney disease (CKD) was more common in the preserved LVEF group (78.9%) compared to the reduced LVEF group (54.5%), but this difference was not statistically significant ($p = 0.16$). Similarly, dyslipidaemia was more frequent in the reduced LVEF group (81.8%) compared to the preserved LVEF group (52.6%), though this difference did not reach statistical significance ($p = 0.06$).

In terms of concomitant medication use, there were significant differences between the groups. The use of beta-blockers (90.9% vs. 31.6%, $p = 0.002$), diuretics (100% vs. 31.6%, $p < 0.001$), mineralocorticoid receptor antagonists (MRAs) (100% vs. 0%, $p < 0.001$), and antiplatelets/oral anticoagulants (90.9% vs. 47.4%, $p = 0.04$) was significantly higher in the reduced LVEF group compared to the preserved LVEF group. However, ACE inhibitors or ARBs were similarly used in both groups ($p = 0.75$).

Symptomatic presentation, assessed through New York Heart Association (NYHA) classification, revealed that most patients in the reduced LVEF group were in NYHA class III-IV (90.9%) compared to 63.2% in the preserved LVEF group, although this difference did not reach statistical significance ($p = 0.09$). Dyspnoea was prevalent in both groups, affecting 90.9% in the reduced LVEF group and 84.2% in the preserved LVEF group ($p = 0.61$). Syncope and palpitation were also comparable between the groups, with no statistically significant differences observed.

Electrocardiogram (ECG) findings were diverse but not significantly different between the groups ($p = 0.12$). The most common abnormalities in the reduced LVEF group included LV hypertrophy (LVH) with strain (54.6%) and LVH combined with other conduction abnormalities. In the preserved LVEF group, the most frequent findings included LVH with strain (52.6%) and left bundle branch block (21.1%). Similarly, chest X-ray findings showed a significant difference between the groups ($p = 0.001$), with structural changes such as LV and LA enlargement and pulmonary venous hypertension more prevalent in the reduced LVEF group, while the majority (94.7%) of the preserved LVEF group had normal X-ray findings.

Table 1: Distribution of baseline characteristics among the study participants (N=30)

| Sino | Variable | EF \leq 50% (n=11) | EF >50% (n=19) | P value |
|------|---|-------------------------|-------------------------|---------|
| 1 | Age | 75.73 \pm 7.33 | 73.53 \pm 8.74 | 0.48 |
| 2 | Gender Female Male | 0 (0) 11 (100%) | 5 (26.3%) 14 (73.7%) | 0.06 |
| 3 | Coronary artery disease Yes No | 11 (100%) 0 (0) | 9 (47.4%) 10 (52.6%) | 0.003 |

| | | | | |
|----|---|--|--|---|
| 4 | Arterial Hypertension Yes No | 9 (81.8) 2 (18.2) | 14 (73.7) 5 (26.3) | 0.61 |
| 5 | Diabetes Mellitus Yes No | 7 (63.6) 4 (36.4) | 4 (21.1) 15 (78.9) | 0.02 |
| 6 | COPD Yes No | 2 (18.2) 9 (81.8) | 5 (26.3) 14 (73.7) | 0.61 |
| 7 | Pulmonary hypertension Mild Moderate Severe | 4 (36.4) 4 (36.4) 2 (18.2) | 7 (36.8) 1 (5.3) 0 (0) | 0.009 |
| 8 | CKD Yes No | 6 (54.5) 5 (45.5) | 15 (78.9) 4 (21.1) | 0.16 |
| 9 | Dyslipidemia Yes No | 9 (81.8) 2 (18.2) | 10 (52.6) 9 (47.4) | 0.06 |
| 10 | Smoking Yes No | 4 (36.4) 7 (63.6) | 4 (21.1) 15 (78.9) | 0.36 |
| 11 | Concomitant medications ACE/ARB Beta blocker Diuretics MRA Antiplatelet/OAC | 7 (63.6) 10 (90.9) 11 (100) 11 (100) 10 (90.9) | 11 (57.9) 6 (31.6) 6 (31.6) 0 (0) 9 (47.4) | 0.75 0.002 <0.001 <0.001 0.04 |
| 12 | NYHA I-II III-IV | 1 (9.1) 10 (90.9) | 7 (36.8) 12 (63.2) | 0.09 |
| 13 | Angina Yes No | 3 (27.3) 8 (72.7) | 6 (31.6) 13 (68.4) | 0.80 |
| 14 | Dyspnoea Yes No | 10 (90.9) 1 (9.1) | 16 (84.2) 3 (15.8) | 0.61 |
| 15 | Syncope Yes No | 3 (27.3) 8 (72.7) | 2 (10.5) 17 (89.5) | 0.23 |
| 16 | Palpitation Yes No | 1 (9.1) 10 (90.9) | 4 (21.1) 15 (78.9) | 0.39 |
| 17 | ECG LVH with LV strain AF AF with LVH LAHB + Incomplete RBBB LBBB | 6 (54.6) 0 (0) 2 (18.2) 0 (0) 0 (0) | 10 (52.6) 2 (10.5) 0 (0) 1 (5.3) 4 (21.1) | 0.12 |

| | | | | |
|----|--|----------|-----------|-------|
| | LBBB with LVH | 1 (9.1) | 0 (0) | |
| | LVH + LAHB + Incomplete RBBB | 1 (9.1) | 0 (0) | |
| | Normal | 1 (9.1) | 2 (10.5) | |
| 18 | X-Ray | | | |
| | Grade 1 PVH | 3 (27.3) | 0 (0) | 0.001 |
| | Grade 1 PVH with LV enlargement | 2 (18.2) | 0 (0) | |
| | Grade 1 PVH with LA and LV enlargement | 1 (9.1) | 0 (0) | |
| | Grade 2 PVH with LA and LV enlargement | 1 (9.1) | 0 (0) | |
| | Grade 2 PVH with LV enlargement | 2 (18.2) | 1 (5.3) | |
| | LV enlargement | 1 (9.1) | 0 (0) | |
| | Normal | 1 (9.1) | 18 (94.7) | |

Figure 1: Distribution of LVEF among the study participants (N=30)

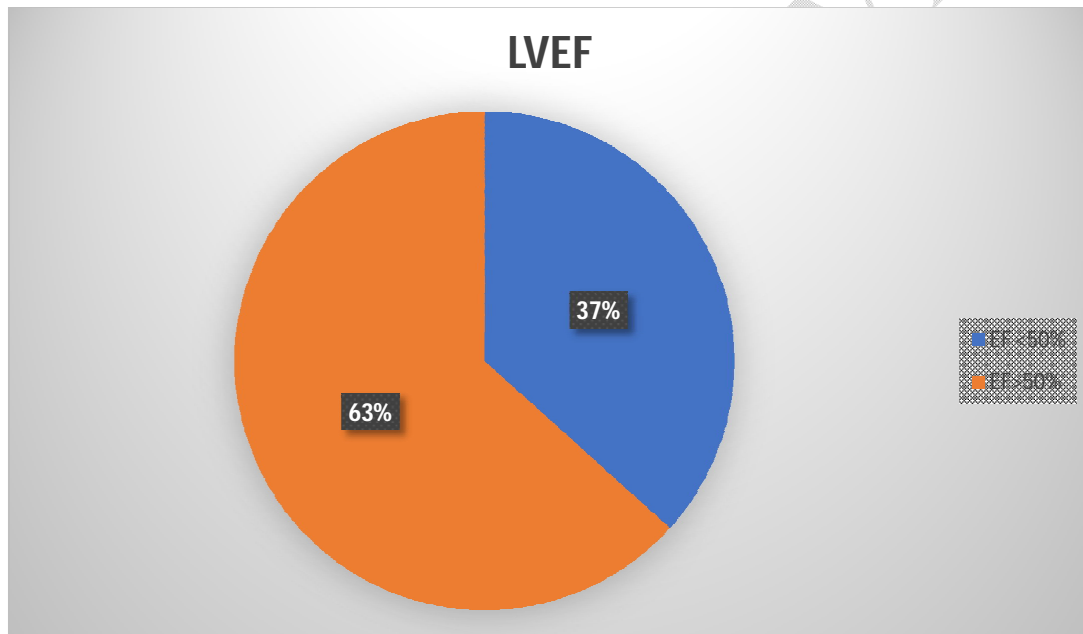


Table 2: Association of descriptive variables between baseline and post TAVI among the study participants (N=30)

| Slno | Descriptive Statistics | Pre TAVI Mean± SD | Post TAVI (3 months) Mean± SD | P value |
|------|---|-------------------|-------------------------------|---------|
| 1 | Velocity across AV (m/sec) | 4.22±0.66 | 2.07±0.56 | <0.001 |
| 2 | Aortic Valve area (cm ²) | 0.61±0.19 | 1.67±0.20 | <0.001 |
| 3 | Aortic Valve area index (cm ² / m ²) | 0.33±0.11 | 0.94±0.12 | <0.001 |
| 4 | Aortic mean gradient (mm Hg) | 50.9±12.53 | 6.3±3.43 | <0.001 |

| | | | | |
|----|--|-------------|-------------|--------|
| 5 | LV end-diastolic diameter (cm) | 5.05±0.83 | 4.90±0.83 | <0.001 |
| 6 | LV end-systolic diameter (cm) | 3.45±0.91 | 3.13±0.78 | <0.001 |
| 7 | LV diastolic volume (ml) | 95.4±33.91 | 87.63±30.84 | <0.001 |
| 8 | LV systolic volume (mL) | 46.73±31.6 | 40.57±24.14 | <0.001 |
| 9 | LVOT Diameter (cm) | 1.97±0.15 | 2.053±0.12 | 0.001 |
| 10 | LVOT VTI | 19.6±4.05 | 20.4±3.52 | 0.131 |
| 11 | Stroke Volume (mL/beat) | 30±58.93 | 64.3±13.49 | 0.015 |
| 12 | LVEF (%) | 54.2±12.18 | 56.6±12.54 | 0.003 |
| 13 | Lateral \dot{E} (m/sec) | 0.05±0.01 | 0.06±0.01 | <0.001 |
| 14 | E/ \dot{E} ratio | 20.423±8.80 | 14.63±5.81 | <0.001 |
| 15 | Septal \dot{E} (m/sec) | 0.04±0.01 | 0.06±0.01 | <0.001 |
| 16 | RVSP (mm Hg) | 41.27±18.84 | 33.67±13.44 | <0.001 |
| 17 | IVS (ed) cm | 1.35±0.13 | 1.14±0.14 | <0.001 |
| 18 | Trv max (m/s) | 2.92±0.68 | 2.62±0.53 | <0.001 |
| 19 | Ao (ed) cm | 3.35±0.62 | 3.35±0.65 | 0.932 |
| 20 | LVPW (ed) cm | 1.14±0.22 | 1.01±0.17 | 0.001 |
| 21 | LV Global Longitudinal Strain % | -13.20±6.33 | -17.27±4.28 | <0.001 |
| 22 | LV Global longitudinal Strain Rate(S-1) | 0.63±0.17 | 0.80±0.22 | <0.001 |
| 23 | Grade of Diastolic Dysfunction | 2.33±0.75 | 1.6±0.56 | <0.001 |
| 24 | PALS | 30±11.46 | 18.83±6.91 | <0.001 |
| 25 | LA Volume (ml) | 38.67±9.34 | 31.33±7.61 | <0.001 |
| 26 | LA diameter (cm) | 4.12±0.63 | 3.76±0.53 | <0.001 |
| 27 | LA Volume Index Diastole ml/m ² | 22.75±5.49 | 18.43±4.47 | <0.001 |

The comparison of echocardiographic parameters before and three months after transcatheter aortic valve implantation (TAVI) reveals significant improvements in multiple aspects of cardiac function and structure among the study participants (Table 2). Aortic valve parameters showed remarkable improvement post-TAVI. The velocity across the aortic valve decreased significantly from 4.22 ± 0.66 m/s to 2.07 ± 0.56 m/s ($p < 0.001$). The aortic valve area increased substantially from 0.61 ± 0.19 cm² to 1.67 ± 0.20 cm² ($p < 0.001$), along with the aortic valve area index, which improved from 0.33 ± 0.11 cm²/m² to 0.94 ± 0.12 cm²/m² ($p < 0.001$). The aortic mean gradient also decreased significantly from 50.9 ± 12.53 mmHg to 6.3 ± 3.43 mmHg ($p < 0.001$), indicating an effective reduction in outflow obstruction. Left ventricular (LV) dimensions and volumes exhibited significant reductions. The LV

end-diastolic diameter decreased from 5.05 ± 0.83 cm to 4.90 ± 0.83 cm ($p < 0.001$), and the LV end-systolic diameter reduced from 3.45 ± 0.91 cm to 3.13 ± 0.78 cm ($p < 0.001$). Similarly, LV diastolic volume and systolic volume decreased significantly from 95.4 ± 33.91 mL to 87.63 ± 30.84 mL and from 46.73 ± 31.6 mL to 40.57 ± 24.14 mL, respectively (both $p < 0.001$). These reductions indicate improved ventricular unloading post-TAVI. Stroke volume increased significantly from 30 ± 58.93 mL/beat to 64.3 ± 13.49 mL/beat ($p = 0.015$), reflecting improved cardiac output. The LV ejection fraction (LVEF) showed a modest but significant improvement from $54.2 \pm 12.18\%$ to $56.6 \pm 12.54\%$ ($p = 0.003$).

Tissue Doppler and diastolic function indices also improved. The lateral \dot{E} velocity increased from 0.05 ± 0.01 m/s to 0.06 ± 0.01 m/s ($p < 0.001$), while the E/\dot{E} ratio decreased significantly from 20.423 ± 8.80 to 14.63 ± 5.81 ($p < 0.001$), indicating better diastolic function. The septal \dot{E} velocity also increased significantly from 0.04 ± 0.01 m/s to 0.06 ± 0.01 m/s ($p < 0.001$). There was a significant reduction in the right ventricular systolic pressure (RVSP) from 41.27 ± 18.84 mmHg to 33.67 ± 13.44 mmHg ($p < 0.001$), reflecting reduced pulmonary pressure post-intervention. Structural changes were observed in LV and left atrium (LA) parameters. The interventricular septal thickness decreased from 1.35 ± 0.13 cm to 1.14 ± 0.14 cm ($p < 0.001$), and the left ventricular posterior wall thickness reduced from 1.14 ± 0.22 cm to 1.01 ± 0.17 cm ($p = 0.001$). LA dimensions also showed improvement, with the LA diameter decreasing from 4.12 ± 0.63 cm to 3.76 ± 0.53 cm ($p < 0.001$), and LA volume decreasing from 38.67 ± 9.34 mL to 31.33 ± 7.61 mL ($p < 0.001$). The LA volume index similarly reduced from 22.75 ± 5.49 mL/m² to 18.43 ± 4.47 mL/m² ($p < 0.001$), suggesting favourable atrial remodelling post-TAVI.

Functional improvement in myocardial mechanics was evident. The LV global longitudinal strain improved significantly from $-13.20 \pm 6.33\%$ to $-17.27 \pm 4.28\%$ ($p < 0.001$), along with an increase in the LV global longitudinal strain rate from $0.63 \pm 0.17 \text{ S}^{-1}$ to $0.80 \pm 0.22 \text{ S}^{-1}$ ($p < 0.001$). The grade of diastolic dysfunction improved significantly, with the mean grade reducing from 2.33 ± 0.75 to 1.6 ± 0.56 ($p < 0.001$). Pulmonary arterial systolic pressures and diastolic filling indices also improved significantly, reflecting better overall cardiac performance.

Table 3: Association of descriptive variables between baseline and post TAVI with respect to LVEF among the study participants (N=30)

| Slno | Descriptive Statistics | EF \leq 50% (n=11) | | EF >50% (n=19) | |
|------|--|---------------------------|--|---------------------------|--|
| | | Pre TAVI Mean \pm SD | Post TAVI (3 months) Mean \pm SD | Pre TAVI Mean \pm SD | Post TAVI (3 months) Mean \pm SD |
| 1 | Velocity across AV (m/sec) | 3.84 \pm 0.67 | 1.78 \pm 0.57 | 4.45 \pm 0.57 | 2.24 \pm 0.5 |
| 2 | Aortic Valve area (cm ²) | 0.61 \pm 0.14 | 1.71 \pm 0.18 | 0.62 \pm 0.22 | 1.66 \pm 0.22 |
| 3 | Aortic Valve area index (cm ² / m ²) | 0.33 \pm 0.08 | 0.96 \pm 0.12 | 0.34 \pm 0.13 | 0.93 \pm 0.13 |
| 4 | Aortic mean gradient (mm Hg) | 41.73 \pm 10.4 | 4.36 \pm 1.63 | 56.21 \pm 10.58 | 7.42 \pm 3.73 |
| 5 | LV end-diastolic diameter (cm) | 5.66 \pm 0.97 | 5.44 \pm 1.05 | 4.71 \pm 0.5 | 4.6 \pm 0.49 |
| 6 | LV end-systolic diameter (cm) | 4.16 \pm 1.09 | 3.63 \pm 1.04 | 3.05 \pm 0.46 | 2.85 \pm 0.39 |
| 7 | LV diastolic volume (ml) | 117.55 \pm 44.71 | 106.64 \pm 40.56 | 82.58 \pm 16.42 | 76.63 \pm 16.44 |
| 8 | LV systolic volume (mL) | 75.73 \pm 36.55 | 63.64 \pm 26.01 | 29.95 \pm 7.46 | 27.21 \pm 7.05 |
| 9 | LVOT Diameter (cm) | 1.99 \pm 0.13 | 2.08 \pm 0.13 | 1.96 \pm 0.16 | 2.04 \pm 0.12 |
| 10 | LVOT VTI | 16.91 \pm 3.94 | 17.55 \pm 3.17 | 21.16 \pm 3.3 | 22.05 \pm 2.55 |
| 11 | Stroke Volume (mL/beat) | 51.82 \pm 10.19 | 57.0 \pm 15.03 | 63.05 \pm 15.87 | 68.53 \pm 10.8 |
| 12 | LVEF (%) | 40.09 \pm 8.56 | 43.55 \pm 11.0 | 62.37 \pm 2.54 | 64.16 \pm 4.66 |
| 13 | Lateral É (m/sec) | 0.05 \pm 0.02 | 0.06 \pm 0.02 | 0.06 \pm 0.02 | 0.07 \pm 0.01 |

| | | | | | |
|----|--|---------------|---------------|---------------|---------------|
| 14 | E/Ē ratio | 25.36 ± 10.61 | 18.27 ± 7.21 | 17.56 ± 6.22 | 12.53 ± 3.56 |
| 15 | Septal Ē (m/sec) | 0.04 ± 0.01 | 0.06 ± 0.01 | 0.05 ± 0.01 | 0.07 ± 0.01 |
| 16 | RVSP (mm Hg) | 54.82 ± 22.78 | 41.73 ± 16.08 | 33.42 ± 10.32 | 29.0 ± 9.21 |
| 17 | IVS (ed) cm | 1.36 ± 0.17 | 1.12 ± 0.18 | 1.34 ± 0.11 | 1.15 ± 0.12 |
| 18 | Trv max (m/s) | 3.45 ± 0.72 | 2.96 ± 0.59 | 2.62 ± 0.44 | 2.43 ± 0.4 |
| 19 | Ao (ed) cm | 3.64 ± 0.88 | 3.59 ± 0.98 | 3.19 ± 0.35 | 3.21 ± 0.3 |
| 20 | LVPW (ed) cm | 1.11 ± 0.19 | 1.0 ± 0.15 | 1.16 ± 0.24 | 1.02 ± 0.19 |
| 21 | LV Global Longitudinal Strain % | -11.26 ± 1.69 | -13.21 ± 3.73 | -14.33 ± 7.71 | -19.63 ± 2.43 |
| 22 | LV Global longitudinal Strain Rate(S-1) | 0.46 ± 0.12 | 0.59 ± 0.21 | 0.74 ± 0.11 | 0.94 ± 0.1 |
| 23 | Grade of Diastolic Dysfunction | 2.73 ± 0.47 | 2.0 ± 0.45 | 2.11 ± 0.81 | 1.37 ± 0.5 |
| 24 | PALS | 10.11 ± 6.06 | 14.0 ± 4.17 | 12.25 ± 8.75 | 21.63 ± 6.69 |
| 25 | LA Volume (ml) | 46.55 ± 7.89 | 36.27 ± 7.93 | 34.11 ± 6.81 | 28.47 ± 5.91 |
| 26 | LA diameter (cm) | 4.63 ± 0.59 | 4.13 ± 0.54 | 3.83 ± 0.45 | 3.55 ± 0.41 |
| 27 | LA Volume Index Diastole ml/m2 | 27.38 ± 4.64 | 21.34 ± 4.66 | 20.07 ± 4.01 | 16.75 ± 3.48 |

Patients with LVEF ≤50% represent a population with reduced left ventricular systolic function, indicating potential myocardial dysfunction (Table 3). These patients showed significant improvements post-TAVI, with LVEF increasing from 40.09 ± 8.56% to 43.55 ± 11.0%. This suggests that TAVI contributes to better cardiac function even in patients with compromised ejection fraction, albeit modestly. The stroke volume (51.82 ± 10.19 mL/beat to 57.0 ± 15.03 mL/beat) and LV global longitudinal strain (-11.26 ± 1.69% to -13.21 ± 3.73%) also improved, reflecting enhanced myocardial mechanics. In contrast, patients with LVEF >50%, indicating preserved systolic function, demonstrated smaller but notable improvements post-TAVI (Table 3). LVEF increased slightly from 62.37 ± 2.54% to 64.16 ± 4.66%, and stroke volume improved from 63.05 ± 15.87 mL/beat to 68.53 ± 10.8 mL/beat. Importantly, the LV global longitudinal strain improved significantly from -14.33 ± 7.71% to -19.63 ± 2.43%, suggesting better myocardial strain dynamics in patients

with preserved function compared to those with reduced ejection fraction. These findings highlight that while TAVI benefits both groups, the degree of improvement in myocardial strain and diastolic function parameters (e.g., PALS, E/É ratio) may differ based on baseline systolic function.

DISCUSSION

Systolic Function

Our study highlights significant improvements in left ventricular (LV) systolic function following transcatheter aortic valve implantation (TAVI), particularly in high-risk patients with severe aortic stenosis (AS). Pre-TAVI assessments revealed that despite near-normal left ventricular ejection fraction (LVEF) in some patients, reduced global longitudinal strain (GLS) and strain rate indicated subtle but significant systolic dysfunction. Following TAVI, marked improvement in GLS and strain rate was observed at three months, whereas LVEF showed only a modest increase. These findings emphasize the limitations of relying solely on LVEF for assessing global systolic function in AS patients and highlight the utility of speckle tracking echocardiography (STE) in detecting and monitoring subtle myocardial changes.⁹

Interestingly, we found that improvements in longitudinal function were more pronounced in basal and medial segments of the left ventricle initially. Over time, recovery extended to the apical segments, reflecting a progressive and uniform reversal of myocardial dysfunction. This pattern aligns with prior findings suggesting that recovery post-TAVI occurs from the base to the apex over an extended period.¹⁰ Furthermore, our data showed a significant reduction in LV mass and

dimensions, indicative of reverse remodelling, which corroborates the observed enhancements in systolic function.¹¹

Diastolic Function

Diastolic function, often impaired in patients with longstanding LV pressure overload due to AS, also demonstrated significant improvement post-TAVI. In our study, lateral and septal E' velocities increased significantly, reflecting improved LV relaxation. Additionally, the E/E' ratio, a marker of LV filling pressure, decreased substantially, suggesting reduced filling pressures and improved diastolic mechanics. At baseline, all patients exhibited some degree of diastolic dysfunction. Post-TAVI, nearly half of the patients showed improvement in diastolic function grading, and normalization of diastolic function was observed in a subset of patients. These findings reinforce the potential of TAVI to ameliorate diastolic dysfunction, particularly in high-risk populations with longstanding pressure overload.¹²

Left Atrial Function

The left atrium (LA), which plays a critical role in cardiac hemodynamics, demonstrated significant remodelling and functional improvement following TAVI. Strain analysis revealed enhanced reservoir and conduit functions three months post-TAVI, consistent with improved LV filling pressures and relaxation. Reservoir function, primarily influenced by LV compliance, and conduit function, associated with LV relaxation, showed notable gains, aligning with the observed recovery of diastolic function. However, active LA contraction did not exhibit a similar improvement, indicating persistent limitations in atrial contractile function.

Interestingly, LA volumes, including indexed volumes, reduced significantly after TAVI, suggesting favourable structural remodelling. These results contrast with earlier reports on surgical aortic valve replacement in low-risk patients, where LA volumes remained unchanged. This difference may be attributed to the advanced stage of atrial remodelling in our high-risk population, which may limit the capacity for complete functional recovery. Nevertheless, the observed improvements in LA reservoir and conduit functions highlight the interplay between LV and LA mechanics in response to TAVI.¹³

Hemodynamic Performance

The hemodynamic outcomes following TAVI were outstanding, with significant reductions in aortic valve velocity and mean gradient and substantial increases in aortic valve area and stroke volume. These changes underscore the effectiveness of TAVI in alleviating pressure overload and restoring efficient hemodynamics. Importantly, improvements in LV outflow tract velocity-time integral (VTI) and global strain metrics highlight enhanced forward flow and myocardial deformation, contributing to better cardiac performance overall.¹⁴

Our study provides valuable insights into the mid-term effects of TAVI on LV and LA function, emphasizing its role in promoting reverse remodelling and improving cardiac mechanics. These findings are particularly significant for high-risk patients, many of whom are not candidates for surgical intervention due to comorbidities. The pronounced recovery in GLS and strain rate underscores the importance of advanced echocardiographic techniques in assessing outcomes and tailoring patient management.

The left atrial (LA) function is closely tied to ventricular performance. In patients with LVEF $\leq 50\%$, impaired ventricular systolic function contributes to higher left atrial pressure and reduced compliance, as evidenced by larger LA volume indices (27.38 ± 4.64 mL/m² pre-TAVI). Post-TAVI, LA volume and diameter significantly decreased, reflecting reduced left atrial strain and improved hemodynamics. In patients with LVEF $>50\%$, TAVI further enhanced left atrial function, as demonstrated by a reduction in LA volume index (from 20.07 ± 4.01 mL/m² to 16.75 ± 3.48 mL/m²) and improved PALS (from 12.25 ± 8.75 to 21.63 ± 6.69). This suggests that even in preserved LVEF, TAVI relieves the pressure overload on the left atrium, improving its reservoir and conduit functions. These improvements in LA function are critical, as they reduce the risk of atrial fibrillation and enhance overall cardiovascular outcomes, particularly in patients with reduced baseline function.

Conclusion

TAVI is associated with significant improvements in systolic and diastolic LV function, LA remodelling, and overall hemodynamics in high-risk patients with severe AS. The observed recovery of myocardial and atrial mechanics reinforces the potential of TAVI as a transformative intervention, particularly in patients with advanced disease and limited surgical options. Future studies with longer follow-up may provide further insights into the sustained benefits and prognostic implications of these findings.

Disclaimer (Artificial intelligence)

Option 1:

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.) and text-to-image generators have been used during the writing or editing of this manuscript.

UNDER PEER REVIEW

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