Impact of Transcatheter Aortic Valve Implantation on Left Ventricular Function and Mass in Patients with Severe Aortic Stenosis

Original Article

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ABSTRACT

Background: Degenerative aortic stenosis (AS) is most common heart valve disease requiring intervention, particularly in aging populations. Left ventricular (LV) remodeling and dysfunction are significant complications associated with symptomatic AS, necessitating effective treatment options like transcatheter aortic valve implantation (TAVI).

Aim of the Work: To assess impact of TAVI on LV functional recovery and mass regression in patients with severe aortic stenosis.

Patients and Methods: This observational cross-sectional study included 50 patients with severe symptomatic AS who underwent TAVI between. Baseline and 6-month post-procedural echocardiographic parameters, including LV mass, LV mass index, LVEF, left atrial (LA) size, aortic valve area (AVA), and peak aortic velocity, were recorded. Statistical analysis was performed using paired t-tests, and correlation analysis was conducted between preoperative LV mass index and postoperative parameters.

Results: Baseline characteristics included a mean body surface area of 1.86 ± 0.17 m², with 74% of patients having hypertension and 98% experiencing dyspnea. Post-TAVI, significant improvements were observed in LV mass (from 300.12 ± 85.24 gm to 193.36 ± 44.71 gm, $p < 0.0001$) and left atrial size (from 45.64 ± 4.84 mm to 42.56 ± 3.73 mm, $p < 0.0001$). Peak aortic velocity decreased from 4.35 ± 0.59 cm/s to 1.64 ± 0.45 cm/s ($p < 0.0001$), while aortic valve area significantly increased ($p < 0.0001$). LVEF improved post-TAVI but without statistical significance ($p = 0.292$).

Conclusions: It was found that TAVI significantly improved LV function and induced mass regression in patients with severe aortic stenosis.

Key Words: Aortic stenosis; echocardiography, left ventricular function; mass regression, transcatheter aortic valve implantation.

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INTRODUCTION

Degenerative aortic stenosis (AS) is most prevalent heart valve disease requiring intervention, especially in developed countries due to aging populations. For patients presenting with symptomatic AS—characterized by angina, dyspnea, syncope, or a reduction in LVEF to below 50%—treatment typically necessitates either TAVI or SAVR. In absence of these therapeutic interventions, prognosis for patients with severe AS worsens substantially, with a markedly elevated risk of cardiovascular events and mortality[1].

LV remodeling, marked by either hypertrophy or concentric restructuring, initially serves as a compensatory mechanism aimed at sustaining LV systolic performance and maintaining normal wall stress in context of AS. However, as disease advances, increased afterload and heightened LV wall tension progressively impair myocardial contractility. This maladaptive remodeling cascade triggers a range of pathological outcomes, including both diastolic and systolic dysfunction, subendocardial ischemia, myocardial fibrosis, elevated left ventricular end-diastolic pressure, development of pulmonary hypertension, and, ultimately, symptomatic deterioration and increased mortality risk[2].

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Reductions in LVEF in patients with AS may be consequence of two primary mechanisms: afterload-contractility mismatch, where intrinsic LV contractile function is preserved but increased afterload lowers stroke volume and LVEF, or irreversible myocardial damage due to fibrosis or coexisting CAD. While valve replacement can mitigate reduction in LVEF caused by afterload mismatch, extent of functional recovery remains constrained when irreversible myocardial damage has occurred[3].

TAVI is now a well-established treatment option for symptomatic severe AS patients who are not candidates for SAVR. It offers various clinical benefits, including reduced pressure gradients, improved LV function, normalized stroke volume, and LV mass regression, all contributing to symptom relief and improved survival. However, baseline cardiovascular comorbidities may impact extent of LV functional recovery and clinical outcomes, and evidence on long-term LV recovery after TAVI remains limited[4].

AIM OF THE WORK

Accordingly, this study aims to assess impact of TAVI on restoration of LV function and regression of myocardial mass in individuals with advanced AS.

PATIENTS AND METHODS

This observational cross-sectional study included 50 patients with severe symptomatic AS who underwent TAVI, Comprehensive echocardiographic assessment was performed before and six months post TAVI at the Cardiology Department of Nasr City Hospital and Mansoura University Hospital between December 2021 and May 2023. Study targeted patients with clinically significant aortic stenosis, as defined by established guidelines, who were considered suitable candidates for TAVI[5].

ETHICAL CONSIDERATION

The study received approval from the Medical Research Ethics Committee, Institutional Review Board (IRB), Mansoura Faculty of Medicine, Mansoura University (Ethical Approval Code Number: MS.22.01.1810, Date: 19/02/2022). Informed consent was obtained from all participants prior to enrolment, ensuring ethical compliance and respect for patient autonomy throughout research.

Inclusion criteria were, patients of both sexes with an age > 18 years, with symptomatic and clinically significant aortic stenosis according to ESC guidelines for AS management[5]. Exclusion criteria ruled out patients with high comorbidities (Charlson index ≥ 5), severely reduced cognitive function, a limited life expectancy of less than 1–2 years, high frailty, active infections, or presence of thrombi in left ventricle or aorta.

The analysis included, patient demographics and history (Age, sex, anthropometric measures, risks associated with cardiovascular disease, such as dyslipidemia, smoking status, DM, HTN, chronic kidney disease, history of stroke, or TIA, drug history, and previous PCI or CABG). symptoms presented by patients, including angina, syncope, and dyspnea, were evaluated based on established classification systems such as Canadian Cardiovascular Society (CCS) classification for angina[6] and New York Heart Association (NYHA) classification for dyspnea[7].

Detailed clinical examination was conducted for each patient, which included a full cardiac and neurological assessment to check for any associated conditions, particularly among individuals with a prior history of stroke. Investigations performed as part of pre-procedural assessment included ECG parameters such as heart rhythm (Sinus or AF), and conduction system for QRS duration, PR interval, and left or right bundle branch block. Additionally, a comprehensive transthoracic echocardiogram (TTE) was performed assessing parameters including LV septal thickness, LVEF, posterior wall thickness, LV mass, aortic root diameter, LV mass index, LA diameter, LVEDD, LVESD, E/A ratio, and E/e' ratio.

Patients underwent TAVI after a careful evaluation by heart team. Clinical follow-up, ECG, and transthoracic echocardiography were performed post-procedure with follow up to same variables, at 6months.

Statistical analysis

Data acquisition, revision, coding, and input were performed utilizing Statistical Package for Social Sciences (IBM SPSS), version 23. Quantitative variables following a parametric distribution were reported as mean, standard deviation, and range, whereas non-parametric variables were expressed as median accompanied by IQR. Numbers and percentages were employed to represent qualitative variables. For qualitative data, Chi-square test was employed to compare groups. For quantitative data with parametric distribution, independent t-test was employed, while Mann-Whitney test was employed for non-parametric data. Univariate and multivariate logistic regression analyses were performed to identify key determinants linked to conduction disturbances, with results expressed as odds ratios (OR) alongside their corresponding 95%

confidence intervals (CI). Statistical significance was established at a *p-value* threshold of less than 0.05.

RESULTS

Baseline characteristics of 50 cases included in study showed body surface area (BSA) ranged from 1.51 to 2.26 m², with a mean value of 1.86 ± 0.17 m². For risk factors, 74% of patients had hypertension, 56% had DM, 86% had dyslipidemia, 22% were smokers, 28% had CKD, and 14% had a history of stroke. Seven patients were receiving medical treatment for IHD, 4 underwent PCI, and 2 had a history of CABG. In terms of symptoms, 44% of patients reported chest pain, 98% had dyspnea, and 40% experienced syncope. Regarding medications, 40% of patients were on antiplatelet therapy, 76% were taking antihypertensive medications, 6% were on oral anticoagulants, and 76% were using statins (Table 1).

Table 1: Patients' demographic data and their baseline characteristics (n = 50).

Sample characteristics	No.	%
BSA		
Min.– Max.	1.51 – 2.26	
Mean ±SD.	1.86 ± 0.17	
Risk factors		
Hypertension	38	74
Diabetes Mellitus	28	56
Dyslipidemia	43	86
Smoking	11	22
Chronic kidney disease	14	28
Stroke	7	14
IHD		
Medical treatment	7	18
PCI	4	12
CABG	2	5.7
Symptoms		
Chest pain	22	44
Dyspnea	49	98
Syncope	20	40
Medications		
Anti-platelet	20	40
Anti-hypertensive	38	76
Oral anti-coagulant	3	6
Statin	38	76

BSA: body surface area; PCI: Percutaneous Intervention; CABG: Coronary Artery Bypass Graft; IHD: Ischemic Heart Disease.

The statistical analysis revealed significant improvements in several parameters following TAVI procedure at 6-month follow-up. LA size showed a statistically significant reduction from 45.64 ± 4.84 mm to 42.56 ± 3.73 mm (p<0.0001). Similarly, LV mass and LV mass index demonstrated substantial decreases from 300.12 ± 85.24 gm to 193.36 ± 44.71 gm and from 162.72 ± 44.34 to 104.43 ± 22.38, respectively (both p<0.0001). LVEDd and LVEDs also significantly reduced (p=0.001 and p=0.019, respectively). Further, IVsd and PWd both showed significant reductions (p<0.0001 for both), as did improvements in LVOT diameter, area, and VTI (all p<0.0001). E/e' ratio also decreased significantly (p<0.0001). Peak aortic velocity (Vmax) and AVA showed remarkable improvements, with Vmax decreasing from 4.35 ± 0.59 cm/s to 1.64 ± 0.45 cm/s (p<0.0001) and AVA increasing significantly (p<0.0001). Non-significant changes were observed in aortic diameter (p=0.32), LVEF (p=0.292), and E/A ratio (p=0.31) (Table 2).

Table 2: Statistical data for various parameters before and at 6-months postoperatively.

Parameters	Pre-operative, mean ± standard deviation	6-month post-operative, mean ± standard deviation	*p-value*
AO (mm)	29.60 ± 3.23	29.20 ± 3.21	0.32
LA (mm)	45.64 ± 4.84	42.56 ± 3.73	P< 0.0001*
LVEF (%)	62.92 ± 9.80	64.22 ± 5.75	0.292
LV mass (gm)	300.12±85.24	193.36 ± 44.71	P< 0.0001*
LV mass index	162.72± 44.34	104.43± 22.38	P< 0.0001*
LVEDd (mm)	51.66 ± 9.09	48.70 ± 6.03	P= 0.001*
LVEDs(mm)	33.16 ± 7.22	31.52 ± 4.92	P= 0.019*
IVsd (mm)	13.84 ± 1.61	10.78 ± 1.04	P< 0.0001*
PWd (mm)	13.44 ± 1.55	10.54 ± 1.11	P< 0.0001*
LVOTd	20.99 ± 1.60	21.96± 1.23	P< 0.0001*
LVOT area	3.53 ± 0.54	3.82± 0.47	P< 0.0001*
LVOT VTI	19.90± 2.87	26.30± 4.51	P< 0.0001*
E/A ratio	1.13 ± 0.58	1.06± 0.31	P= 0.31
E/e' ratio	10.10± 2.84	7.52± 0.99	P< 0.0001*
Vmax	4.35 ± 0.59	1.64 ± 0.45	P< 0.0001*
VTI (cm)	107.09±23.35	30.20 ± 10.06	P< 0.0001*
AVA	0.66± 0.16	29.82±2.14	P< 0.0001*

LA: left atrium; LV: left ventricle; LVEF: left ventricular ejection fraction; LVEDd: left ventricular end-diastolic diameter; LVEDs: left ventricular end-systolic diameter; IVsd: interventricular septal thickness at end-diastole; EA ratio: early-to-late mitral inflow velocity ratio; VTI: velocity time integral; AVA: aortic valve area. Pearson correlation coefficient was used. * Statistically significant as *P-value* less than 0.05.

The Pearson correlation analysis revealed several significant associations between preoperative LV mass index and various postoperative parameters. A strong positive correlation was observed between preoperative LV mass index and LV mass at 6 months postoperative ($r = 0.682$, $p < 0.0001$), as well as LV mass index at 6 months postoperative ($r = 0.700$, $p < 0.0001$). Similarly, significant positive correlations were found with LVEDd ($r = 0.582$, $p = 0.001$) and LVEDs ($r = 0.547$, $p < 0.0001$). Moderate correlations were also noted with LA size at 6 months postoperative ($r = 0.335$, $p = 0.017$), IVsd ($r = 0.375$, $p = 0.007$), and PWd ($r = 0.297$, $p = 0.036$). However, no significant correlations were found between preoperative LV mass index and other parameters such as AO, LVEF, LVOTd, LVOT area, VTI, or AVA at 6 months postoperative (Table 3).

Table 3: Pearson correlation between preoperative LV mass index and other variables.

Parameters	Pre-operative LV mass index	
	Pearson Correlation	Sig. (2-tailed)
AO at 6 months postoperative	0.012	0.936
LA at 6 months postoperative	0.335	0.017*
LVEF at 6 months postoperative	-0.260	0.068
LV mass at 6 months postoperative	0.682	<0.0001*
LV mass index at 6 months postoperative	0.700	<0.0001*
LVEDd at 6 months postoperative	0.582	0.001*
LVEDs at 6 months postoperative	0.547	<0.0001*
IVsd at 6 months postoperative	0.375	0.007*
PWd at 6 months postoperative	0.297	0.036*
LVOTd at 6 months postoperative	0.143	0.208
LVOT area at 6 months postoperative	0.294	0.086
LVOT VTI at 6 months postoperative	0.194	0.264
E/A ratio at 6 months postoperative	-0.037	0.833
E/e' ratio at 6 months postoperative	0.129	0.461
Vmax at 6 months postoperative	0.160	0.358
VTI at 6 months postoperative	0.074	0.673
AVA at 6 months postoperative	0.083	0.637

LA: left atrium; LV: left ventricle; LVEF: left ventricular ejection fraction; LVEDd: left ventricular end-diastolic diameter; LVEDs: left ventricular end-systolic diameter; IVsd: interventricular septal thickness at end-diastole; EA ratio: early-to-late mitral inflow velocity ratio; VTI: velocity time integral; AVA: aortic valve area. Pearson correlation coefficient was used. * Statistically significant as *P-value* less than 0.05.

DISCUSSION

AS is an increasingly prevalent cardiovascular condition, largely driven by longer life expectancies and shifting demographics in Western populations[8]. Aortic valve calcification, a key feature of AS, shares risk factors with systemic atherosclerosis, yet no medical interventions have proven effective in halting its progression[9]. TAVI and SAVR are currently only validated treatment options. Initially reserved for elderly, frail patients deemed high-risk for traditional surgery, TAVI has since been adapted for use in intermediate and low-risk populations, broadening its potential impact on AS management[10]. This investigation explored effects of TAVI on recovery of LV function and regression of myocardial mass in a cohort of 50 patients diagnosed with AS.

This investigation revealed a marked reduction in LV mass and LV mass index throughout a six-month follow-up period. LV mass index offers a more accurate assessment of myocardial mass relative to an individual's physical characteristics, as it adjusts for variations in body surface area. Sudden reduction of LV afterload following TAVI diminishes pathological trigger driving hypertrophy of LV myocytes, thereby initiating a gradual decline in myocardial mass over time[11]. Similarly, ***O'Leary et al***.[12] study has demonstrated a significant decrease in LV mass and mass index after surgical Aortic Valve Replacement, as evidenced even in echocardiograms performed prior to discharge. These findings indicate that most substantial regression in LV mass occurs within early postoperative months. However, evidence from other studies suggests that LV mass reduction persists beyond six-month mark following AVR[13].

Consistently, in a study conducted by ***Gotzmann et al.***[14], a significant decrease in LV mass index was observed 6 months after TAVI. They also indicated a notable reduction in LV mass index, suggesting a regression of LV hypertrophy following TAVI. Moreover, another study by ***Tzikas et al***.[15] also examined effects of TAVI on LV mass. Their findings revealed a decline in LV mass index, which dropped from an initial value of 126 ± 42 g/m² to 110 ± 30 g/m² over course of one year. This reduction in LV mass index was statistically significant ($p < 0.001$), indicating a significant decrease in LV mass over course of one year following TAVI.

The current study findings revealed a significant positive correlation between preoperative LV mass index and thickness of both septal wall and posterior wall 6 months postoperatively suggesting that higher LV mass is associated with increased thickness of these specific LV walls following TAVI. Similar to our results, a study by ***Vizzardi et al***.[16] showed that preoperative LV mass index

has positive correlation with thickness of both septal LV wall and posterior LV wall 6 months after TAVI. AS often leads to LV hypertrophy as a compensatory mechanism to overcome pressure overload caused by aortic valve stenosis. In this context, it is expected that patients with higher preoperative LV mass would exhibit greater septal wall thickness after TAVI. This is further corroborated by positive correlation identified between LV mass and posterior wall thickness, reflecting myocardial response to elevated LV workload[17].

According to present study, it was showed that LVEF improved following TAVI. However, not statistically significant difference was found. Our findings are consistent with several studies that have examined impact of TAVI on LVEF following procedure. ***Grabskaya et al***. study included 40 patients diagnosed with severe AS assessing LVEF after a six-month follow-up period. there was no significant change in LVEF immediately after TAVI. However, strain analysis values showed a significant increase after six-month follow-up period[18]. Additionally, ***Lwin et al***.[19] involved 40 patients diagnosed with significant AS scheduled for TAVI. They reported a noteworthy 4% improvement in LVEF after TAVI procedure, indicating a positive effect LV function.

Furthermore, ***D'Andrea et al***.[20] conducted an analysis on 55 patients with severe symptomatic AS to assess impact of TAVI on LV and LA longitudinal function using STE. results demonstrated a significant improvement in LVEF six months after TAVI procedure (with a p-value of less than 0.0001). Consistently, ***Kamperidis et al***.[21] demonstrated functional recovery of LV, as evidenced by improvements in GLS, occurring within first 6 months after TAVI and remaining stable thereafter. Another study by ***Stangl et al***.[22] has shown that improvement in LVEF is more likely in women, possibly due to a lower burden of myocardial fibrosis compared to men. However, individuals with irreversible myocardial damage, including infarcted or scarred regions and fibrotic changes, are unlikely to achieve meaningful restoration of LV function or significant regression of LV hypertrophy. These individuals often carry a significantly higher procedural risk[23].

It is worth noting that careful patient selection is crucial when considering TAVI as a treatment option due to potential for periprocedural complications and economic burden associated with procedure. Future research should focus on refining patient selection criteria, assessing long-term outcomes, and comparing TAVI with other treatment modalities to further optimize management of severe aortic stenosis

However, our study has some limitations worth mentioning including relatively short duration of follow-up period after TAVI procedure, and lack of investigation for effects of TAVI on specific subgroups, such as patients with irreversible myocardial damage or those with preserved LV function which could provide insights into appropriate candidate selection for procedure.

CONCLUSION

The present study demonstrated that TAVI can significantly improve LV function and induce mass regression in patients with severe aortic stenosis supporting TAVI as an effective intervention for enhancing cardiac performance and reducing burden of aortic stenosis.

CONFLICT OF INTERESTS

There are no conflicts of interest.

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CONTRIBUTION

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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تأثير زراعة الصمام الأورطي عن طريق القسطرة على وظيفة انقباض البطين الأيسر وكتلته في المرضى الذين يعانون من الضيق الحاد

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الخلفية: يعد التضيق التنكسي للصمام الأبهري أكثر أمراض صمامات القلب شيوعًا والتي تتطلب تدخلًا، خصوصًا في الفئات العمرية المتقدمة. يشكل إعادة تشكيل البطين الأيسر واختلال وظيفته من المضاعفات الهامة المرتبطة بالتضيق الأبهري المصحوب بأعراض، مما يستدعي الحاجة إلى خيارات علاجية فعالة مثل زرع الصمام الأبهري عبر القسطرة.

هدف الدراسة: تقييم تأثير إجراء زرع الصمام الأبهري عبر القسطرة على استعادة الوظيفة البطينية اليسرى وتراجع الكتلة القلبية في المرضى الذين يعانون من تضيق أبهري شديد.

الطرق: شملت هذه الدراسة الرصدية المستعرضة 50 مريضًا يعانون من تضيق أبهري شديد مصحوب بأعراض وخضعوا لإجراء زرع الصمام الأبهري عبر القسطرة. تم تسجيل المعايير التخطيطية القلبية الأساسية وبعد 6 أشهر من الإجراء، بما في ذلك كتلة البطين الأيسر، مؤشر كتلة البطين الأيسر، كسر القذف البطيني الأيسر، حجم الأذين الأيسر، مساحة الصمام الأبهري، وسرعة التدفق الأقصى عبر الصمام الأبهري. تم إجراء التحليل الإحصائي باستخدام اختبار "t" المقترن، وتحليل الارتباط بين مؤشر كتلة البطين الأيسر قبل الإجراء والمعايير بعده.

النتائج: تضمنت الخصائص الأساسية متوسط مساحة سطح الجسم 1.86 ± 0.17، حيث كان لدى 74٪ من المرضى ارتفاع في ضغط الدم و98٪ يعانون من ضيق في التنفس. بعد إجراء زرع الصمام الأبهري، لوحظت تحسينات كبيرة في كتلة البطين الأيسر (من 300.12 ± 85.24 إلى 193.36 ± 44.71) وحجم الأذين الأيسر (من 45.64 ± 4.84 إلى 42.56 ± 3.73). كما انخفضت سرعة التدفق الأقصى عبر الصمام الأبهري من 4.35 ± 0.59 إلى 1.64 ± 0.45، بينما زادت مساحة الصمام الأبهري بشكل كبير. تحسن كسر القذف البطيني الأيسر بعد الإجراء ولكن بدون دلالة إحصائية.

الاستنتاجات: تبين أن إجراء زرع الصمام الأبهري عبر القسطرة أدى إلى تحسن ملحوظ في وظيفة البطين الأيسر وتراجع في الكتلة القلبية لدى المرضى الذين يعانون من تضيق أبهري شديد.