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Left Ventricular Outflow Tract Anatomy as a Predictor of Conduction Disturbances after Transcatheter Aortic Valve Implantation

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Abstract

Background: Transcatheter aortic value implantation (TAVI) has transformed the management of elderly aortic stenosis patients (AS). However, conduction disturbances post-TAVI remain a concern.

Objective: To explore the influence of left ventricular outflow tract (LVOT) anatomical and morphological characteristics assessed by cardiac CT on the progression of conduction disturbances after TAVI.

Methods: This prospective observational study recruited 60 symptomatic severe AS patients scheduled for trans-femoral TAVI. Pre-procedural cardiac CT evaluated various LVOT morphological characteristics. Pre- and post-operative ECGs were conducted, and procedural details were recorded, including implantation depth, balloon pre/post-dilation, and transcatheter heart valve (THV) oversizing.

Results: Patients with a composite endpoint of new left bundle branch block at discharge or permanent pacemaker placement exhibited significantly lower weight than those without the endpoint (77 kg ±11 vs. 88 kg ±16, respectively, p = 0.009). Flared LVOT anatomy was less common in patients with the composite endpoint (10.5%) versus those without (37.5%) (p = 0.033). Multivariate analysis revealed weight (OR = 0.938, 95% CI = 0.884–0.996, p = 0.036) and implantation depth (OR = 1.976, 95% CI = 1.108–3.523, p = 0.021) as significant predictors of the composite endpoint.

Conclusion: Weight and implantation depth are significant predictors of post-TAVI conduction disturbances. Additionally, while not reaching statistical significance, the Euroscore II and flared anatomy trended towards being a potential predictor, showing the need for further investigation into their role in post-TAVI conduction disturbances.

Keywords: Left Ventricular Outflow Tract; Anatomy; Conduction Disturbances; Transcatheter Aortic Valve Implantation

1. Introduction

C evere symptomatic aortic stenosis (AS) is

D connected to very high mortality if left untreated. Transfemoral aortic valve implantation (TAVI) enhances overall survival, causes reversal of LV remodeling, and ameliorates symptoms. ¹

Currently, the era of TAVI has changed the demographic as well as clinical characteristics of AS Patients towards elderly sick patients. TAVI has surfaced as a viable substitute for surgical aortic valve replacement (SAVR) in the management of symptomatic severe AS patients who are considered unsuitable for surgical intervention or who have a heightened likelihood of adverse procedure results.²

A few years ago, TAVI was exclusive to highrisk surgical patients, but now, it is also being applied to low and intermediate-surgical risk groups. TAVI indications have expanded to include younger, less morbid patients with relatively longer life expectancies, making it necessary to optimize TAVI outcomes and minimize its drawbacks. ^{3,4}

Conduction disturbances and permanent pacemaker requirements remain relatively common complications of TAVI. ⁵

Several risk factors for conduction abnormalities after TAVI. such as prior conduction defects and implantation depth, have been established over the last decade.^{6,7,8} However, the function of LVOT anatomy in the pathogenesis of conduction disturbances still needs to be thoroughly examined.

Thus, this research aimed to examine the influence of LVOT anatomical and morphological characteristics assessed by cardiac C.T. on the progression of conduction disturbances after TAVI.

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2. Patients and methods

2.1.Study Design and Patients:

This research was a prospective observational research initiative aimed at enrolling patients from Nasr City Health Insurance Hospital, As-Salam International Hospital, and Al-Azhar University Hospitals. Every executed procedure adhered to the ethical guidelines set forth by the institutional and national committees responsible for human testing, the Helsinki Declaration of 1964, and subsequent editions. All participants provided informed consent to be enrolled in the research.

Eligible cases included those diagnosed with senile tricuspid symptomatic severe AS who are scheduled for trans femoral TAVI by a multidisciplinary heart team.

A pre-procedural C.T. evaluation of the aortic valve and root was conducted.

Exclusion criteria were pre-existing pacemaker placement, severe renal impairment, acute infective endocarditis, left ventricle apical thrombus, and insufficient cardiac C.T. data.

2.2.Methods

Upon obtaining written informed consent, all cases were subjected to:

Pre-operative ECG: Baseline ECG analysis to determine rate, rhythm, P.R. interval, QRS, QTc, and baseline conduction abnormalities.

Pre-operative MSCT aortography: Utilized to assess LVOT morphological features (Figures 1 and 2) and investigate their correlation with post-TAVI conduction disturbances, exploring:

Membranous septum length, Minimum LVOT diameter (anteroposterior diameter), Maximum LVOT diameter (transverse diameter), LVOT eccentricity index (1 – minimum diameter / maximum diameter), Perimeter-derived LVOT diameter, Area-derived LVOT diameter, LVOT perimeter, Valve oversizing by LVOT perimeter, LVOT area (mm), Valve oversizing by LVOT area, k)Calcification analysis using calcium score and l) Annular dimension / LVOT dimension to classify LVOT morphology into tapered vs. tubular:

The delta value that exists between the mean diameter of the left ventricular outflow tract (LVOTd) and the mean diameter of the aortic annulus (AAd) were measured and split into two groups: Group 1 - patients with AAd > LVOTd & tapered LVOT morphology; Group 2 - patients with tubular (AAd = LVOTd) or flared (AAd < LVOTd) LVOT morphology.

Procedural characteristics: Assessment of implantation depth, the necessity for balloon predilatation or post-dilatation, and transcatheter heart valve (THV) oversizing.

Postoperative ECG (at 48-72 hrs): This is an ECG analysis to determine changes in rate, rhythm, P.R. interval, QRS, and QTc.

2.3. Statistical analysis:

Statistical analysis and data management were conducted with SPSS version 28 (IBM, Armonk, New York, United States). In order to examine the normality of the quantitative data, the Shapiro-Wilk test, Kolmogorov-Smirnov test, and direct visualization techniques were utilized. data Ranges, means, standard deviations, and medians were utilized to summarise quantitative data by the principle of normality. In order to summarize categorical data, percentages, and numbers were utilized. Based on the composite endpoint, quantitative data were contrasted utilizing the Mann-Whitney U test for non-normally distributed variables and the Independent t-test for normally distributed variables. The chi-square and Fisher's exact tests were employed to contrast categorical data. The composite endpoint was predicted through the implementation of multivariate logistic regression. The odds ratios were computed, accompanied by confidence intervals of 95%. Every statistical test has two sides. Significance was attributed to P values below 0.05.

3. Results

3.1.Demographic characteristics according to the composite endpoint

The study comprised 60 patients. At discharge, fifty-nine patients were alive and classified according to the presence or absence of a composite endpoint of a new left bundle branch block at discharge or permanent pacemaker. Patients' weight was the only variable showing a significant difference between groups. Patients who met the composite endpoint had a mean weight of 77 kg (±11 S.D.), which was significantly less than those who did not meet the endpoint, who had a mean weight of 88 kg (±16 S.D.), with a p-value of 0.009 (Table 1).

Other variables, including age (P = 0.614), gender (P = 0.206), height (P = 0.524), BMI (P = 0.053), diabetes mellitus (P = 0.441), antidiabetic treatment (P = 0.834), hypertension (P = 0.359), dyslipidemia (P = 0.85), smoking (P = 1), chronic obstructive pulmonary disorder (COPD) (P = 0.653), and other chronic diseases (P = 0.532), did not show significant associations with the composite endpoint (Table 1).

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		COMPOSITE ENDP	OINT	
	Total	Yes (n = 19)	No (n = 40)	P-value
AGE (YEARS)	73 ±6	73 ±4	73 ±7	0.614
GENDER				
MALES	19 (32.2)	4 (21.1)	15 (37.5)	0.206
FEMALES	40 (67.8)	15 (78.9)	25 (62.5)	
HEIGHT (CM)	165 ±9	164 ±5	166 ±10	0.524

WEIGHT (KG)	84 ±15	77 ±11	88 ±16	0.009*
BODY MASS INDEX	30.8 ±6.2	28.6 ±5.4	31.9 ±6.3	0.053
DIABETES MELLITUS	26 (44.1)	7 (36.8)	19 (47.5)	0.441
ANTIDIABETIC TREATMENT	26 (44.1)	8 (42.1)	18 (45)	0.834
HYPERTENSION	39 (66.1)	11 (57.9)	28 (70)	0.359
DYSLIPIDEMIA	29 (49.2)	9 (47.4)	20 (50)	0.850
SMOKING	7 (11.9)	2 (10.5)	5 (12.5)	1.0
COPD	6 (10.2)	1 (5.3)	5 (12.5)	0.653
OTHER CHRONIC DISEASE	37 (62.7)	13 (68.4)	24 (60)	0.532

Data were presented as Mean \pm standard deviation (SD), *Significant at P < 0.05; COPD: Chronic obstructive pulmonary disease

3.2.Clinical characteristics according to the composite endpoint

The EURO score II significantly differed according to the composite endpoint, with a median value of 6.1% (range 1.5%-22.4%) in the group with the composite endpoint compared to 2.5% (range 0.8%-20.6%) in the group without the composite endpoint (P = 0.039) (Table 2).

All other variables assessed, including shortness of breath (P = 0.544), angina (P = 0.132), syncope (P = 0.376), prior myocardial infarction (MI) (P = 0.074), peripheral vascular disorder (PVD) (P = 0.322), coronary artery bypass grafting (CABG) (P = 0.078), percutaneous

intervention (PCI) (P coronary 0.182), ischemic cerebrovascular accident/transient attack (CVA/TIA) (P = 0.588), hemoglobin levels (P= 0.832), total leukocyte count (TLC) (P = 0.954), international normalized ratio (INR) (P = 0.399), creatinine levels (P = 0.396), glomerular filtration rate (GFR) (P = 0.581), New York Heart Association (NYHA) class (P = 0.222), various cardiac rhythm and conduction disturbances, mean pressure gradient (MPG) (P = 0.268), peak pressure gradient (PPG) (P = 0.474), left ventricular ejection fraction (LVEF) (P = 0.242). The aortic valve area (A.V. area) (P = 0.526) was not significantly connected to the composite endpoint in our research (Table 2).

Table 2. Clinical characteristics according to the composite endpoint

		COMPOSITE ENDPOINT		
	Total	Yes (n = 19)	No (n = 40)	P-value
SHORTNESS OF BREATH	57 (96.6)	18 (94.7)	39 (97.5)	0.544
ANGINA	32 (54.2)	13 (68.4)	19 (47.5)	0.132
SYNCOPE	6 (10.2)	3 (15.8)	3 (7.5)	0.376
PRIOR MI	16 (27.1)	8 (42.1)	8 (20)	0.074
PVD	1 (1.7)	1 (5.3)	0 (0)	0.322
CABG	6 (10.2)	4 (21.1)	2 (5)	0.078
PCI	18 (30.5)	8 (42.1)	10 (25)	0.182
CVA/TIA	4 (6.8)	2 (10.5)	2 (5)	0.588
HEMOGLOBIN	11.8 ±1.4	11.8 ±1.1	11.9 ±1.5	0.832
TLC	7 (3.5 - 20)	7 (3.5 - 11)	7 (4 - 20)	0.954
INR	1.1 (0.9 - 3.5)	1 (0.9 - 2.1)	1.1 (0.9 - 3.5)	0.399
CREATINE	1 (0.45 - 3.5)	1 (0.6 - 1.3)	1 (0.45 - 3.5)	0.396
GFR (ML/MIN)	64.8 (19.7 - 150)	64 (40 - 127)	66.9 (19.7 - 150)	0.581
EURO SCORE II (%)	2.75 (0.84 - 22.4)	6.13 (1.46 - 22.4)	2.49 (0.84 - 20.6)	0.039*
NYHA CLASS				
Ι	3 (5.1)	2 (10.5)	1 (2.5)	0.222
Ш	8 (13.6)	2 (10.5)	6 (15.0)	
Ш	30 (50.8)	7 (36.8)	23 (57.5)	
IV	18 (30.5)	8 (42.1)	10 (25.0)	
PREVIOUS VALVE SURGERY	1 (1.7)	0 (0)	1 (2.5)	1
RHYTHM				
SINUS	52 (88.1)	18 (94.7)	34 (85)	0.411
AF	7 (11.9)	1 (5.3)	6 (15)	
PR (MSEC)	168 ±37	177 ±40	163 ±34	0.20
QRS (MSEC)	88 ±20	91 ±23	87±18	0.493
QT (MSEC)	371 ±43	372 ±56	370 ±36	0.896
QTC (MSEC)	407 ±41	401 ±57	410 ±32	0.528
1ST DEGREE HB	7 (11.9)	3 (15.8)	4 (10)	0.670
RBBB	2 (3.4)	1 (5.3)	1 (2.5)	0.544
LBBB	7 (11.9)	4 (21.1)	3 (7.5)	0.197
PREOP ECHO MPG	54 ±14	57 ±17	53 ±13	0.268
PREOP ECHO PPG	89 ±23	93 ±26	88 ±21	0.474
LVEF	62 ±10	60 ±11	63 ±10	0.242
AV AREA (CM ²)	0.76 ±0.13	0.75 ±0.15	0.77 ±0.13	0.526

Data were presented as n (%), Mean ± standard deviation (SD)and Median (range), *Significant at P < 0.05; PVD: Peripheral Vascular Disease; CABG: Coronary Artery Bypass Grafting; PCI: Percutaneous Coronary Intervention; CVA: Cerebrovascular Accident; TIA: Transient Ischemic Attack; TLC: Total Leukocyte Count; INR: International Normalized Ratio; GFR: Glomerular Filtration Rate; ml/min: milliliters per minute; EURO score II: European System for Cardiac Operative Risk Evaluation II; NYHA: New York Heart Association; AF: Atrial Fibrillation; QT: QT Interval; QTC: Corrected QT Interval; HB: Heart Block; MPG: Mean Pressure Gradient; PPG: Peak Pressure Gradient; LVEF: Left Ventricular Ejection Fraction; AV: Aortic Valve; cm2: square centimeters.

3.3.Anatomical characteristics according to the composite endpoint

LVOT anatomy significantly differed according to

the composite endpoint (P =0.033), with the flared type being higher in those without the composite endpoint (37.5%) compared to those with the

composite endpoint (10.5%) (Table 3).

All remaining measurements were not significantly associated with the composite end point, including A.V. morphology (P = 0.445), A.V. calcification (P = 0.868), LVOT calcification (P = 0.365), minimum LVOT diameter (P = 0.831), maximum LVOT diameter (P = 0.745), eccentricity index LVOT (P = 0.859), area derived LVOT (P = 0.998), LVOT perimeter (P = 0.686), perimeter derived LVOT (P = 0.621), LVOT area (P = 0.649), *Table 3. Anatomical characteristics of the aortic va* mean LVOT diameter (P = 0.541), minimum aortic annulus diameter (P = 0.495), maximum aortic annulus diameter (P = 0.914), mean aortic annulus diameter (P = 0.67), annular eccentricity index (P = 0.302), perimeter derived aortic annulus (P = 0.748), area derived aortic (P = 0.748), aortic annulus perimeter (P = 0.538), aortic annulus area (P = 0.586), aortic root angulation (P = 0.763), and membranous septum length (P = 0.291) (Table 3, Figure 1).

Table 3. Anatomical characteristics of the aortic valve and left ventricular outflow tract according to the composite endpoint in the studied patients

		COMPOSITE ENDPOIN	Т	
	Total	Yes (n = 19)	No (n = 30)	P-value
AV MORPHOLOGY				
BICUSPID	4 (6.8)	2 (10.5)	2 (5.0)	0.445
TRILEAFLET	55 (93.2)	17 (89.5)	38 (95.0)	
AV CALCIFICATION				
MILD	7 (11.9)	2 (10.5)	5 (12.5)	0.868
MODERATE	30 (50.8)	9 (47.4)	21 (52.5)	
SEVERE	22 (37.3)	8 (42.1)	14 (35)	
LVOT CALCIFICATION				
NO	26 (44.1)	10 (52.6)	16 (40)	0.365
MILD	27 (45.8)	7 (36.8)	20 (50)	
MODERATE	5 (8.5)	1 (5.3)	4 (10)	
SEVERE	1 (1.7)	1 (5.3)	0 (0)	
MINIMUM LVOT DIAMETER	20.1 ±3.2	20.2 ±2.7	20 ±3.4	0.831
MAXIMUM LVOT DIAMETER	27.6 ±3.1	27.8 ±3.3	27.5 ±3	0.745
ECCENTRICITY INDEX LVOT	0.27 ±0.08	0.28 ±0.07	0.27 ±0.09	0.859
AREA DERIVED LVOT	23.6 ±3	23.6 ±2.6	23.6 ±3.2	0.998
LVOT PERIMETER	77.5 ±9.8	78.2 ±9.5	77.1 ±10	0.686
PERIMETER DERIVED LVOT	24.6 ±3.1	24.9 ±3.1	24.5 ±3.1	0.621
LVOT AREA	439.6 ±113.7	429.7 ±110.9	444.3 ±116.2	0.649
MEAN LVOT DIAMETER	23.8 ±3	24.1 ±2.9	23.6 ±3	0.541
MINIMUM AORTIC ANNULUS DIAMETER	20.7 ±2.5	21 ±2.4	20.6 ±2.6	0.495
MAXIMUM AORTIC ANNULUS DIAMETER	27.1 ±2.7	27.1 ±2.6	27 ±2.8	0.914
MEAN AORTIC ANNULUS DIAMETER	23.9 ±2.5	24.1 ±2.3	23.8 ±2.6	0.670
ANNULAR ECCENTRICITY INDEX	0.24 (0.03 - 0.7)	0.21 (0.13 - 0.36)	0.24 (0.03 - 0.7)	0.302
PERIMETER-DERIVED AORTIC ANNULUS	24.4 ±2.5	24.5 ±2.2	24.3 ±2.6	0.748
AREA DERIVED AORTIC	24 ±2.5	24.1 ±2.2	23.9 ±2.7	0.748
AORTIC ANNULUS PERIMETER	76.6 ±7.6	77.4 ±6.9	76.1 ±7.9	0.538
AORTIC ANNULUS AREA	449 ±87.6	458.1 ±79	444.7 ±92.1	0.586
LVOT ANATOMY				
FLARED	17 (28.8)	2 (10.5)	15 (37.5)	0.033*
NOT FLARED	42 (71.2)	17 (89.5)	25 (62.5)	
AORTIC ROOT ANGULATION	42 ±7	42 ±5	42 ±7	0.763
MEMBRANOUS SEPTUM LENGTH	8.3 ±1.1	8.6 ±1.4	8.2 ±0.9	0.291

Data were presented as n (%), Mean ± standard deviation (SD) and Median (range), AV: Aortic Valve; LVOT: Left Ventricular Outflow Tract; SD: Standard Deviation; mm: millimeters; cm: centimeters; SD: Standard Deviation.



Figure 1. LVOT anatomy according to the composite endpoint

3.4.Procedural characteristics according to the composite endpoint

Implantation depth showed a notable difference between patients with the composite endpoint (median depth of 5 mm) and those without (median depth of 4 mm), indicating significance (P = 0.008). Similarly, the magnitude of the postdilation balloon was significantly more significant in the group with the composite endpoint (mean size of 24 ± 2) compared to the group without (mean size of 22 ± 2), with a P-value of 0.036 (Table 4).

Other variables did not demonstrate statistical significance. These include device type, valve oversizing (P = 0.805) device size (P = 0.323), predilatation (P = 0.063), size of pre-dilatation balloon (P = 0.648), post-dilatation (P = 0.182), vascular complications (P = 0.656), any in-hospital CVA (P = 0.322), paravalvular leak or A.R. grade (P = 0.448), postoperative echo PPG (P = 0.196), and postoperative echo MPG (P = 0.141) (Table 4).

Table 4. Procedural characteristics according to the composite endpoint in the studied patients

	COMPOSITE ENDPOINT		
Total	Yes (n = 19)	No (n = 30)	P-value
59 (0)	19 (100%)	40 (100%)	-
2 (10.5)	2 (10%)	11 (28%)	0.323
13 (68.4)	13 (68%)	21 (52%)	
4 (21.1)	4 (21%)	8 (20%)	
22 ±6	22 ± 6	21 ± 5	0.805
5 (2 - 10)	5 (2-10)	4 (1-7)	0.008*
16 (84.2)	16 (84%)	24 (60%)	0.063
21 ±2	21 ± 2	20 ± 2	0.648
8 (42.1)	8 (42%)	10 (25%)	0.182
24 ±2	24 ± 2	22 ± 2	0.036*
16 (84.2)	16 (84%)	31 (78%)	0.656
3 (15.8)	3 (16%)	6 (15%)	
0 (0)	0 (0%)	3 (8%)	
1 (5.3)	1 (5%)	0 (0%)	0.322
7 (36.8)	7 (37%)	11 (28%)	0.448
6 (31.6)	6 (32%)	16 (40%)	
5 (26.3)	5 (26%)	13 (32%)	
1 (5.3)	1 (5%)	0 (0%)	
15 (10 - 24)	15 (10-24)	14 (7.5-38)	0.196
7 (4.5 - 13)	7 (4.5-13)	6.2 (4-20)	0.141
	$\begin{array}{c} {\rm Total} \\ 59 \ (0) \\ 2 \ (10.5) \\ 13 \ (68.4) \\ 4 \ (21.1) \\ 22 \ \pm 6 \\ 5 \ (2 - 10) \\ 16 \ (84.2) \\ 21 \ \pm 2 \\ 8 \ (42.1) \\ 24 \ \pm 2 \\ \end{array}$ $\begin{array}{c} 16 \ (84.2) \\ 3 \ (15.8) \\ 0 \ (0) \\ 1 \ (5.3) \\ \end{array}$ $\begin{array}{c} 7 \ (36.8) \\ 6 \ (31.6) \\ 5 \ (26.3) \\ 1 \ (5.3) \\ 1 \ (5.3) \\ 1 \ (5.10 - 24) \\ 7 \ (4.5 - 13) \end{array}$	COMPOSITE END Total Yes (n = 19) 59 (0) 19 (100%) 2 (10.5) 2 (10%) 13 (68.4) 13 (68%) 4 (21.1) 4 (21%) 22 ±6 22 ± 6 5 (2 - 10) 5 (2-10) 16 (84.2) 16 (84%) 21 ±2 21 ± 2 8 (42.1) 8 (42%) 24 ± 2 24 ± 2 16 (84.2) 16 (84%) 3 (15.8) 3 (16%) 0 (0) 0 (0%) 1 (5.3) 1 (5%) 7 (36.8) 7 (37%) 6 (31.6) 6 (32%) 5 (26.3) 5 (26%) 1 (5.3) 1 (5%) 7 (36.8 7 (37%) 6 (31.6) 6 (32%) 5 (26.3) 5 (26%) 1 (5.3) 1 (5%) 15 (10 - 24) 15 (10 - 24) 7 (4.5 - 13) 7 (4.5 - 13)	COMPOSITE ENDPOINT Total Yes (n = 19) No (n = 30) 59 (0) 19 (100%) 40 (100%) 2 (10.5) 2 (10%) 11 (28%) 13 (68.4) 13 (68%) 21 (52%) 4 (21.1) 4 (21%) 8 (20%) 22 ±6 22 ± 6 21 ± 5 5 (2 - 10) 5 (2-10) 4 (1-7) 16 (84.2) 16 (84%) 24 (60%) 21 ±2 21 ± 2 20 ± 2 8 (42.1) 8 (42%) 10 (25%) 24 ± 2 24 ± 2 22 ± 2 16 (84.2) 16 (84%) 31 (78%) 3 (15.8) 3 (16%) 6 (15%) 0 (0) 0 (0%) 3 (8%) 1 (5.3) 1 (5%) 0 (0%) 7 (36.8) 7 (37%) 11 (28%) 6 (31.6) 6 (32%) 16 (40%) 5 (26.3) 5 (26%) 13 (32%) 1 (5.3) 1 (5%) 0 (0%) 15 (10 - 24) 15 (10 - 24) 14 (7.5-38) 7 (4.5 - 13) 7 (4.5 - 13)

Data were presented as n (%), Mean ± standard deviation (SD) and Median (range), *Significant at P < 0.05; PPG: Peak Pressure Gradient; MPG: Mean Pressure Gradient; AR: Aortic Regurgitation; CVA: Cerebrovascular Accident.

3.5.LVOT anatomy in the prediction of the composite endpoint

Multivariate logistic regression analysis was done to predict the composite endpoint. The model included all significant and borderline significant variables (up to a P-value of 0.1) on the univariate level.

BMI (P =0.053 on the univariate level) was not included to avoid multicollinearity with weight. In addition, size post-dilatation (P = 0.036 on the univariate level) was not included as only 18 patients had postdilatation, limiting the total number of cases in the model and leading to a significant loss of information.

The model revealed that weight (OR = 0.938, 95% CI = 0.884 - 0.996, P = 0.036) and implantation depth (OR = 1.976, 95% CI = 1.108 - 3.523, P = 0.021) were significant predictors for the composite endpoint. Additionally, the Euroscore II (OR = 1.161, 95% CI = 0.99 - 1.362, P = 0.067) and flared anatomy (OR = 0.106, 95% CI = 0.01 - 1.114, P = 0.061) revealed borderline significance as predictors of the composite endpoint (Table 5).

Table 5. Multivariate logistic regression analysis to predict the composite endpoint

	OR (95% CI)	P-VALUE
WEIGHT (KG)	0.938 (0.884 - 0.996)	0.036*
PRIOR MI	0.684 (0.076 - 6.166)	0.735
CABG	1.609 (0.158 - 16.43)	0.688
EURO SCORE II (%)	1.161 (0.99 - 1.362)	0.067
FLARED ANATOMY	0.106 (0.01 - 1.114)	0.061
IMPLANTATION DEPTH (MM)	1.976 (1.108 - 3.523)	0.021*
PREDILATATION	4.594 (0.615 - 34.302)	0.137

*Significant at P < 0.05; MI: Myocardial infarction; CABG: Coronary artery bypass graft; OR: Odds Ratio; CI: Confidence Interval; † Tubular was the reference category.

4. Discussion

Specific LVOT anatomies have been linked to an increased risk of complications like stroke⁹, PVR¹⁰, PPI¹¹, or annular rupture¹². Thus, evaluating a composite clinical endpoint is also reasonable. This research aimed to investigate the effect of LVOT anatomical and morphological characteristics on the progression of conduction disturbances after TAVI.

The study included 60 patients. At discharge, fifty-nine patients were alive and classified according to the presence or absence of a composite endpoint of a new left bundle branch block at discharge or permanent pacemaker. We found that the weight was significantly reduced in the patients who met the composite endpoint compared to those who did not (77±11 vs. 88 ±16 kg). This might be attributed to potential anatomical variations or procedural differences associated with body size. An Asian study by Nakashima et al. found that a problematic TAVI surgery is predicted by smaller body size because of specific anatomical difficulty and adverse outcomes such as annulus rupture, acute coronary blockage, and vascular problems.¹³

The EURO score II significantly differed according to the composite endpoint, with a median value of 6.1% (range 1.5%-22.4%) in the group with the composite endpoint compared to 2.5% (range 0.8%-20.6%) without the composite endpoint. Contrasting our findings, Waldschmidt et al. investigated The effect of LVOT characteristics on results after TAVI. They

reported no significant difference regarding estimated surgical risk by using EuroSCORE II. This discrepancy may be the focus of the investigation solely on LVOT calcification and its direct correlation with mortality and device success. Additionally, differences in the application of EuroSCORE II could contribute to these varying results.¹⁴

In the current study, LVOT anatomy significantly differed according to the composite endpoint, with the flared type being higher in those without the composite endpoint (37.5%) than those with the composite endpoint (10.5%). However, all remaining measurements were not significantly associated with the composite endpoint.

This variation in LVOT anatomy, particularly the higher prevalence of the flared type among patients without the composite endpoint, indicates a potential association between this anatomical variation improved and cardiovascular Flared LVOT outcomes. configuration might contribute to more favourable hemodynamics, reduced flow resistance during ventricular ejection, preserved ventricular function, efficient cardiac output, and possibly better aortic valve function. These factors collectively suggest that specific LVOT morphologies, such as the flared type, could create a more conducive environment for cardiac potentially function, mitigating adverse cardiovascular events. 15, 16

In contrast, Jilaihawi and colleagues reported that the Annulus area, mean annulus diameter, LVOT perimeter, and LVOT area were significantly lower in no permanent pacemaker implantation (PPMI) patients compared to PPMI. This may be attributed to a larger sample of patients in their study. ¹⁷

Implantation depth showed notable а difference between patients with the composite endpoint and those without, indicating significance. Similarly, the size of the postwas significantly dilation balloon more significant in the group with the composite endpoint compared to the group without.

Studies by Mouillet et al.¹⁸ andToutouzas et noted a relationship between deeper al.19 prosthesis implantation and the occurrence of delayed high-grade A.V. block or the requirement for novel permanent pacemaker insertion (PPI). Mouillet et al.¹⁸ discovered that patients experiencing delayed high-grade A.V. block had deeper prosthesis implantation $(12 \pm 4 \text{ vs. } 9 \pm 5 \text{ s})$ Toutouzas et al.¹⁹ demonstrated mm). significantly greater implantation depth in patients requiring new PPI. Conversely, Giannini et al.²⁰ highlighted that the repositionable Evolut R, as opposed to CoreValve, allowed for shallower implantation depths, leading to lower rates of paravalvular leak and new permanent pacemaker insertion. The Evolut R's recommended optimal implantation depth (3–5 mm) is notably shallower than the CoreValve (4–6 mm), indicating potential advantages in reducing complications associated with deeper implantation depths.

Previously, pre-dilatation and post-dilatation were considered essential procedures during TAVI to assure adequate valve expansion and allow device crossing, particularly for THVs with low radial force. Conduction disturbances observed during TAVI have been attributed to predilatation²¹. Despite this, the extent of its influence on the occurrence of periprocedural conduction disturbance or PPI remains uncertain, as mounting evidence indicates that it has no significant effect.⁵

From the electrocardiological point of view, Left bundle branch block (LBBB) is the most prevalent complication following TAVI, affecting 13.3–39% of patients.^{22,23} LBBB was reported in 7 (28.6%) cases.

LBBB are the most prevalent conduction abnormalities following TAVI. This is due to the regression of procedure-associated traumatic inflammation and oedema, which causes postprocedural conduction anomalies to diminish or settle with time, eliminating the necessity for PPI in nearly half of the instances.²⁴ Variable percentages of TAVI using first-generation valves have been reported to have new-onset LBBB (4 % to 65 %). The high degree of reporting variability, the many valve types, and the various periods taken into account throughout the research all contribute to the wide reported range. ⁵

According to earlier research, the majority of conduction abnormalities following TAVI are newonset LBBB, which can occur in as many as 50– 70% of cases (with a wide range of 25% to 85% after implantation of the CoreValve system and from 8 % to 30 % after the implantation of an Edwards Sapien valves), and third-degree A.V. block, necessitating PPI in the range of 5.7 to 42.5 % (with a median of 28 % for the Medtronic CoreValve System and 6 per cent for the Edwards Sapien valves). $^{6, 25-30}$

By performing multivariate logistic regression, our study identified weight and implantation depth as significant predictors of the composite endpoint post-TAVI. Euroscore II and flared anatomy also showed borderline significance in predicting this endpoint.

In their respective analyses, Ziad et al. ³¹ studied different outcomes and distinct anatomical factors as they revealed several independent predictors of NP-LBBB in Evolut post-TAVI. Their findings highlighted significant associations between NP-LBBB occurrence and specific anatomical factors: shorter membranous septum length (OR = 0.82 per mm septum, 95%CI = 0.68 to 0.98, p = 0.030, higher LVOT eccentricity (OR = 1.04 per %, 95% CI = 1.01 to 1.06, p = 0.002), and deeper implant depth at the noncoronary cusp (NCC) (OR = 1.28 per mm ventricular, 95% CI = 1.11 to 1.48, p = 0.001). et al. found an independent Katsanos relationship between the necessity for a pacemaker or new-onset LBBB and the depth of frame into the left ventricular outflow system (odds ratio 1.401, 95 % confidence interval 1.066 to 1.770, p = 0.010).³² Van der Boon and colleagues observed through the multivariate analysis that TAVI-LBBB was significantly predicted by the depth of implantation (OR [95% C.I.]: 1.16 [1.10-1.24], p < 0.001). ³³

Reaching the left subendocardial side of the interventricular septum, the left bundle branch emerges from the His bundle close to the NCC, a few millimetres below the aortic annulus. The most frequently documented predictor of new conduction disturbances is implant depth within the LVOT due to its anatomical position. ³²⁻³⁴

In Guo et al.'s study, the LVOT perimeter, in particular, has the most miniature aortic root architecture and the highest negative predictive value for new-onset conduction disturbances (NOCDs). An increased oversizing ratio by LVOT perimeter, which could result in more significant radial stresses on the conduction system, was more likely in cases where the LVOT perimeter was smaller. A11 multivariate regression models demonstrated the significance of either oversizing by LVOT perimeter or LVOT perimeter. According to Jilaihawi's research, permanent pacemaker implantation may be decreased by minimizing implantation depth as directed by infra-annular membranous septum depth (PPMI). 17

Interestingly, Increased BMI may be linked to a higher likelihood of conduction problems resulting in permanent pacemaker implantation (PPI) after TAVI, according to data from the Netherlands Heart Registry.³⁵

Finally, this study has some limitations: Its relatively small sample size limits the extent to which its results may be applied to larger Additionally, the exclusion of populations. patients with pre-existing pacemakers, severe impairment, and acute infective renal endocarditis might limit the extrapolation of results to these specific patient cohorts. Moreover, the lack of sustained follow-up data impedes the assessment of sustained effects beyond the immediate postoperative period, necessitating further research to confirm the durability of these predictive associations.

4. Conclusion

LVOT anatomy, precisely the flared type, demonstrated a significant association with reduced occurrence of post-TAVI conduction disturbances. Additionally, implantation depth and post-dilation balloon size emerged as crucial factors influencing these complications.

Disclosure

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