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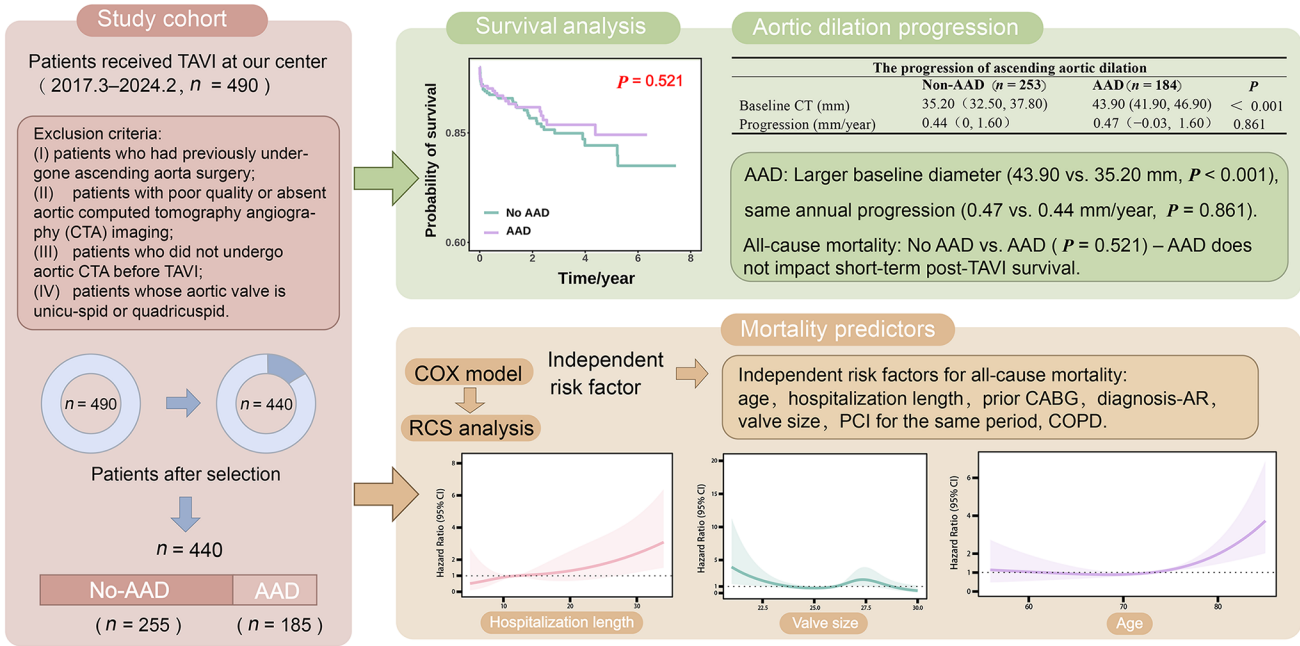
CARDIAC RESEARCH

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# Short-term and 3-year follow-up clinical outcomes after transcatheter aortic valve implantation in patients with aortic valve disease, with or without ascending aortic dilation

Haoyang Li, MM[a], Yuehuan Li, MD[b], Jiawei Zhou, MD[b], Zhang Chen, MM[b] Haibo Zhang, PhD[b,*]

## Graphical abstract




TAVI appears to be safe for patients with aortic valve disease accompanied with ascending aorta dilation. This study found that age, hospitalization length, and valve size may provide a new basis for the risk assessment of all-cause mortality in patients undergoing TAVI.

Supplemental Digital Content is available for this article.

[a] Department of Cardiac Surgery, Beijing Anzhen Hospital, Capital Medical University, Beijing, China, [b] Department of Cardiac Surgery, Beijing Anzhen Hospital, Capital Medical University, Beijing, China.

* Correspondence: Haibo Zhang, Department of Cardiac Surgery, Beijing Anzhen Hospital, Capital Medical University, No.2, Anzhen Road, Chaoyang District, Beijing, China (e-mail: dr_zhanghaibo@163.com).

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**Abstract**

**Background:** Patients with aortic valve disease with ascending aortic dilation (AAD) are not uncommon in clinical practice; however, currently, research on the prognosis of such patients after transcatheter aortic valve implantation (TAVI) surgery in the Asian region is still insufficient. We aimed to compare short-term outcomes and 3-year follow-up clinical outcomes in patients with aortic valve disease who underwent TAVI with or without AAD.

**Methods:** Patients with aortic valve disease who underwent TAVI at Beijing Anzhen Hospital between March 2017 and February 2024 were continuously enrolled and divided into dilated (maximum ascending aorta width ≥ 40 mm) and nondilated groups. The short-term results were analyzed using logistic regression. Kaplan–Meier curves illustrated the survival differences, and the log-rank test was used for evaluation. The independent risk factors affecting 3-year follow-up clinical outcomes were analyzed using a Cox regression model and restricted cubic spline (RCS) analysis. We used patient follow-up data to investigate the rate of progression of AAD.

**Results:** A total of 440 patients were included in this study, including 185 in the AAD group and 255 in the non-AAD group. There was no statistically significant difference in the perioperative complication rate or perioperative mortality between the two groups. The overlap of Kaplan–Meier curves was high in the two groups, and there was no difference in the log-rank test ($P = 0.521$). Restricted cubic spline analysis revealed a nonlinear relationship between age and valve size for predicting all-cause mortality (nonlinear coefficients = 0.0489 and 0.0194). The median follow-up time was 1,051 days (484, 1,541) in the non-AAD group and 961 days (488, 1,672) in the AAD group. Follow-up data showed that there was no significant difference in the rate of progression of AAD between the two groups: 0.44 mm/year (0, 1.60) in the non-AAD group and 0.47 (–0.03, 1.60) in the AAD group ($P = 0.861$). During the follow-up process, no patients in the whole cohort developed ascending aortic dissection.

**Conclusions:** This study found no significant difference in short-term outcomes or 3-year follow-up clinical outcomes between TAVI patients with AAD and those without; therefore, TAVI appears to be safe for patients with aortic valve disease accompanied by ascending aorta dilation. This study found that age, hospitalization length, and valve size may provide a new basis for the risk assessment of all-cause mortality in patients undergoing TAVI, suggesting that individualized management strategies should be developed for age, length of hospital stay, and valve size.

**Keywords:** aortic regurgitation, aortic stenosis, ascending aortic dilation, restricted cubic spline analysis, transcatheter aortic valve implantation

**Nonstandard Abbreviations and Acronyms**

| | |
|---|---|
| **AAD** | Ascending aortic dilation |
| **AR** | Aortic regurgitation |
| **AS** | Aortic stenosis |
| **BAV** | Bicuspid aortic valve |
| **BEV** | Balloon-expandable valves |
| **BMI** | Body mass index |
| **CABG** | Coronary artery bypass grafting |
| **COPD** | Chronic obstructive pulmonary disease |
| **CPB** | Cardiopulmonary bypass |
| **CTA** | Computed tomography angiography |
| **HR** | Hazard ratio |
| **K–M** | Kaplan–Meier |
| **LVEDD** | Left ventricular end-diastolic diameter |
| **LVEF** | Left ventricular ejection fraction |
| **MR** | Mitral regurgitation |
| **NYHA** | New York Heart Association |
| **OR** | Odds ratio |
| **PCI** | Percutaneous coronary intervention |
| **PPI** | Permanent pacemaker implantation |
| **PVL** | Paravalvular leakage |
| **RCS** | Restricted cubic spline |
| **SAVR** | Surgical aortic valve replacement |
| **SEV** | Self-expanding valves |
| **TAVI** | Transcatheter aortic valve implantation |
| **TAV** | Tricuspid aortic valve |
| **TR** | Tricuspid regurgitation |

**Highlights**

- Focusing on the prognosis of patients with aortic valve disease complicated by ascending aortic dilation after transcatheter aortic valve implantation (TAVI), this study systematically analyzed their 3-year follow-up clinical outcomes, filling the gap in medium- and long-term research in this field.
- By comparing the short-term and 3-year prognosis of patients with and without ascending aortic dilation after TAVI, this study confirmed that the former had no significant differences in short-term safety and mid-term efficacy of TAVI.
- Aortic regurgitation patients were innovatively included to fully reflect the anatomical complexity in real-world practice, and aortic regurgitation was identified as an independent risk factor for all-cause mortality after TAVI.
- Restricted cubic spline analysis revealed a nonlinear correlation of age, length of hospital stay, and valve size with all-cause mortality after TAVI, providing a new quantitative basis for risk stratification.
- No ascending aortic dissection occurred during the follow-up of the entire cohort, which confirmed that optimized surgical strategies, such as individualized access selection, could effectively reduce the risk of aortic adverse events in AAD patients after TAVI.

## 1. Introduction

Transcatheter aortic valve implantation (TAVI) has become one of the most important treatments for

patients with severe aortic stenosis (AS). TAVI for pure aortic regurgitation (AR) is less well established, and, in fact, it was previously labeled as a relative contraindication. However, recent prospective trials of TAVI in the treatment of high-risk symptomatic autologous AR have also yielded promising results.[1] Patients with isolated aortic valve disease will have varying degrees of ascending aortic dilation (AAD),[2] and AAD is a common comorbidity in patients with AS.[3] According to the 2021 European Society of Cardiology/European Association for Cardio-Thoracic Surgery (ESC/EACTS) guidelines for the management of valvular heart disease, AR can be caused by primary disease of the aortic valve cusps and/or abnormalities of the aortic root and ascending aortic geometry;[4] therefore, AR patients with AAD are not uncommon.

With the potential for TAVI to be further extended to younger populations in the future, an increasing number of aortic valve disease patients with AAD are undergoing TAVI. However, there is little evidence of the potential impact of AAD on the prognosis of TAVI. The 2021 European guidelines recommend that for patients who have an indication for aortic valve surgery, an aortic diameter ≥ 45 mm is considered to indicate concomitant surgery of the aortic root or tubular ascending aorta.[4] However, TAVI cannot solve the problem of AAD simultaneously. Despite the problem of AAD, the ascending aorta remains intact during TAVI compared with surgical aortic valve replacement (SAVR); therefore, guideline recommendations do not apply to TAVI. In the long term, it is still possible that the ascending aorta will continue to dilate after TAVI, with associated risks of ascending aortic dissection. Therefore, the safety and feasibility of TAVI in patients with aortic valve disease and AAD are worth discussing. The purpose of this study was to discuss the short-term outcomes and 3-year follow-up clinical outcomes after TAVI in aortic valve disease patients with AAD.

To address existing research gaps, this study sets two core objectives: First, based on short-term (perioperative) and long-term (3-year) follow-up data of TAVI patients, identify independent predictors associated with mortality (with short-term outcomes focusing on perioperative mortality and long-term outcomes focusing on all-cause mortality), clarify key risk variables (such as postoperative complications, baseline comorbidities, and procedure-related indicators), and provide a reference basis for clinical risk stratification and individualized intervention. Second, by comparing the clinical outcomes of aortic valve disease patients with AAD and those without AAD after TAVI—including the incidence of short-term complications, 3-year all-cause mortality, and the progression rate of AAD—verify whether AAD is an important factor affecting the safety and long-term prognosis of TAVI, thereby providing evidence-based support for the feasibility of selecting TAVI treatment in aortic valve disease patients with AAD.

## 2. Materials and methods

### 2.1. Patient population

This was a single-center, retrospective study comparing short-term and 3-year follow-up clinical outcomes after TAVI in patients with aortic valve disease and AAD or without AAD. As shown in Fig. 1, patients who underwent TAVI at Beijing Anzhen Hospital from March 2017 to February 2024 were included in this study. The establishment of TAVI indications relies on the consensus reached by the institutional cardiac team, which comprises cardiac surgeons, cardiologists, and anesthesiologists. The following patients were excluded: patients who had previously undergone ascending aorta surgery; patients with poor quality or absent aortic computed tomography angiography (CTA) imaging; patients who did not undergo aortic CTA before TAVI; patients whose aortic valve is unicuspid or quadricuspid. We reviewed a total

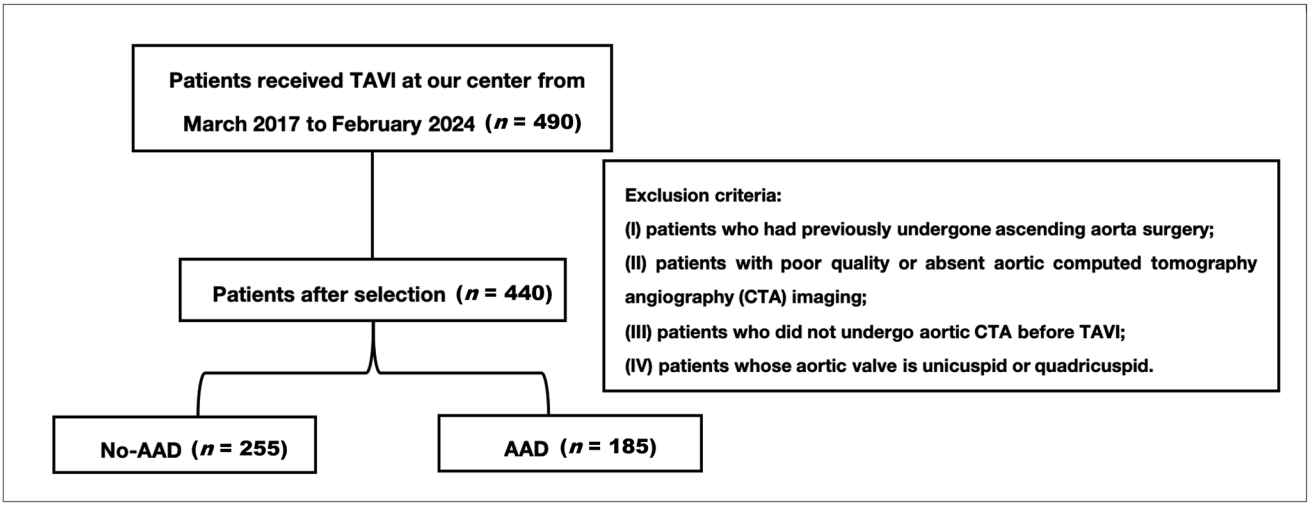



Figure 1. Flow diagram showing the numbers of individuals at each stage of the study. AAD = ascending aortic dilation.

of 490 patients, and after excluding those who met the above criteria, 440 patients were included in the study. This study was approved by the Medical Ethics Review Committee of Beijing Anzhen Hospital (Ethics Registry Number: 2025074x) and was exempt from the requirement of informed consent due to its nature.

### 2.2. Echocardiography

Echocardiographic images were acquired using a standard ultrasound system. Ultrasound specialists performed baseline and postoperative TAVI echocardiographic evaluations following guidelines established by the Society of Echocardiography.[5,6] Severe AS is defined as follows: aortic valve area (AVA) < 1.0 $cm^2$; or peak aortic jet velocity ≥ 4 m/s; or a mean gradient ≥ 40 mmHg. The quantitative definitions of severe AR are as follows: regurgitant fraction (%) > 50; regurgitant volume (mL/beat) > 60.

### 2.3. Aortic CTA

Patients included in the study were assessed for ascending aorta diameter by transthoracic echocardiography (TTE) and aortic CTA preoperatively, as recommended by guidelines. Measurement of the aortic root and ascending aorta in two-dimensional (2D) is performed at four levels: annulus, sinuses of Valsalva, sinotubular junction, and tubular ascending aorta.[4] The maximum of the four values was taken as the patient's ascending aorta diameter. All the CTA images were evaluated using the 3mensio (10.6) workstation and analyzed by a dedicated core laboratory. AAD is defined as the maximum ascending aorta diameter ≥ 40 mm. We recommend that all patients have a repeat aortic CTA at our hospital within 3 months to 1 year after TAVI to assess the morphology and function of the prosthetic valve and the presence of microthrombosis.

### 2.4. Study endpoints and follow-up

All study endpoints were reported according to the Valve Academic Research Consortium-3 criteria (the definition of the endpoint outcomes can be found in the materials, Supplemental Digital Content, https://links.lww.com/CARES/A24). The primary endpoint was all-cause mortality. The secondary endpoints included cardiovascular mortality, perioperative mortality, early mortality, late mortality, technical success, device success, and procedural complications. Procedural complications included postoperative stroke, permanent pacemaker implantation (PPI), postoperative moderate paravalvular leakage (PVL) (no patients in the whole cohort had a paravalvular leak more severe than a moderate paravalvular leak), postoperative vascular and access-related complications, postoperative kidney damage, and coronary obstruction. A total of 437 patients completed the follow-up in this study (with three cases lost to follow-up). For the AAD group, the median follow-up duration was 961 days (interquartile range [IQR]: 484–1,672 days), corresponding to a total number of regular follow-ups ranging from 3 to 10 (median: 6). For the non-AAD group, the median follow-up duration was 1,051 days (IQR: 484–1,541 days), with the total number of regular follow-ups ranging from 3 to 9 (median: 6). There was no statistically significant difference in the distribution of total follow-up times between the two groups (Mann–Whitney *U* test, $P = 0.654$).

### 2.5. Transcatheter aortic valve implantation

The cardiology team performs TAVI in a hybrid operating room, where the patient is placed under general anesthesia and monitored by anesthesiologists. All patients underwent TAVI after discussion with the multidisciplinary cardiology team, and those who met the criteria were treated with either a transfemoral or transapical approach.

Patients undergoing TAVI surgery at our center are generally of advanced age, and many may concurrently present with functional mitral regurgitation (MR) or tricuspid regurgitation (TR). For patients with mild-to-moderate MR or TR, no specific interventions were administered for these valvular regurgitations. For those with severe MR or TR, we attempted to reduce the severity to mild-to-moderate through oral medications or intravenous infusions before TAVI surgery.

### 2.6. Data collection

We manually and retrospectively recorded baseline characteristics, procedural data, and hospitalization information from the electronic medical record system and entered it into a dedicated database.

### 2.7. Statistical analysis

Categorical variables were presented as numbers and percentages and compared using a chi-square test or Fisher exact test as well as the Mann–Whitney *U* test. The Shapiro–Wilk test was used to test the normality of all continuous variables, and the continuous variables that met normality were presented as means ± standard deviations and were compared by *t* tests; continuous variables that did not conform to normality were presented as medians and IQR s and were compared with the Mann–Whitney *U* test. Univariate and multivariate logistic regression models were used to analyze short-term outcomes in the cohort. In the study, 3-year follow-up clinical outcomes were visually represented by Kaplan–Meier curves (K–M curves) and statistically tested using the log-rank test. Univariate and multivariate Cox regression models were used to identify the independent risk factors for all-cause mortality in the entire cohort. Restricted cubic spline (RCS) analysis was employed for the continuous variables among the independent risk factors. Analyses were then performed in different subgroups to determine whether the effect of ascending aorta width on all-cause mortality differed among the subgroups. All tests were 2-sided, and *P* values < 0.05 were considered statistically significant. All statistical analyses were performed using SPSS

26 and RStudio (*R*), and the results were plotted in *R* and GraphPad Prism 10.

## 3. Results

### 3.1. Baseline characteristics

A total of 440 patients with aortic valve disease who underwent TAVI were included in this study and were divided into the AAD ($n$ = 185) group and the non-AAD group ($n$ = 255) according to whether the ascending aorta was ≥ 40 mm before TAVI. The baseline characteristics of the included patients are shown in Table 1. The mean age was 72.5 ± 7.8 years in the non-AAD group and 71.7 ± 7.7 years in the AAD group. There were 60 (23.7%) patients with bicuspid aortic valve (BAV) in the non-AAD group and 61 (33.0%) in the AAD group. There were 132 (51.8%) male patients in the non-AAD group and 129 (69.7%) in the AAD group. There was a statistically significant difference in AAD/body mass index between the two groups, with 1.8 (1.6, 2.1) in the AAD group and 1.5 (1.3, 1.6) in the non-AAD group ($P$ < 0.001). In addition, compared with the non-AAD group, patients in the AAD group had a larger left ventricular end-diastolic diameter (LVEDD), lower left ventricular ejection fraction (LVEF), a lower incidence of pulmonary hypertension, and a shorter hospital stay, as shown in Table 1.

### 3.2. Procedural details and clinical outcomes

As shown in Table 1, the majority of patients underwent the operation via trans-femoral access: 189 (74.1%) in the non-AAD group and 116 (62.7%) in the AAD group ($P$ = 0.010). The use of balloon-expandable valves was lower in both groups (5.1% vs. 5.4%), and the majority of patients used self-expanding valves (SEV) (94.9% vs. 94.6%). There was no statistically significant difference in the type of valve implantation between the two groups ($P$ = 0.886). There was a statistically significant difference in the size of the implanted valves between the two groups ($P$ = 0.003). The median sizes of the implanted valves in the non-AAD group and the AAD group were 26 mm and 27 mm, respectively. There were no statistically significant differences in percutaneous coronary intervention (PCI) for the same period, intraoperative cardiopulmonary bypass, second valve implantation, or valve detachment between the two groups ($P$ values were 0.282, 0.366, 0.633, and 0.247, respectively).

Regarding the clinical outcomes, there were no statistically significant differences in poststroke, post-PPI, moderate PVL, postvascular and access-related complications, or postkidney damage between the two groups, with $P$ values of 0.477, 0.191, 0.074, > 0.999, and 0.846, respectively. Coronary obstruction did not occur in either group. There were no cases of iatrogenic aortic dissection in the entire study cohort. The technical success, as defined by Valve Academic Research Consortium-3 criteria, was 198 (77.6%) in the non-AAD group and 149 (80.5%) in the AAD group, with no statistically significant difference between the two groups ($P$ = 0.397); there was no statistically significant difference in device success between the two groups, with 185 (72.5%) in the non-AAD group and 143 (77.3%) in the AAD group ($P$ = 0.219).

The results of postoperative echocardiography revealed statistically significant differences in LVEF, LVEDD, and pulmonary hypertension between the two groups ($P$ values were 0.007, < 0.001, and 0.024, respectively). The AAD group had lower LVEF, greater LVEDD, and a lower incidence of pulmonary hypertension compared with the non-AAD group. There was no statistically significant difference for the other variables, as shown in Table 1.

There was no statistically significant difference in all-cause mortality between the two groups (13.7% in the non-AAD group vs. 11.4% in the AAD group, $P$ = 0.461). Cardiovascular mortality was slightly higher in the AAD group than in the non-AAD group (4.9% vs. 4.3%), although there was no statistically significant difference between the two groups ($P$ = 0.764). There were no statistically significant differences in periprocedural mortality, early mortality, or late mortality between the two groups, with $P$ values of 0.938, 0.571, and 0.111, respectively.

### 3.3. Short-term outcomes

As previously stated, there was no statistically significant difference in periprocedural mortality between the two groups. To determine the independent risk factors for perioperative mortality in the whole cohort, some variables in the secondary endpoints were first included in the univariate logistic model analysis, and the variables with $P$ values < 0.2 were included in the multivariate logistic model analysis. The odds ratio (OR) values and $P$ values were calculated, and the results are shown in Table 2. As shown in Table 2, the $P$ values of poststroke, postkidney damage, and post-LVEF were < 0.2 in the univariate logistic model. After the multivariate logistic model analysis, the $P$ values for poststroke and postkidney damage were statistically significant ($P$ < 0.05). Therefore, poststroke and postkidney damage were independent risk factors for periprocedural mortality in the whole cohort in this study.

### 3.4. 3-year follow-up clinical outcomes

The medians and IQRs of survival time in this study were 877 days (415, 1,382) in the non-AAD group and 842 days (432, 1,435) in the AAD group, with no statistically significant difference between the groups ($P$ = 0.872). As shown in Table 1, there were 56 all-cause deaths and 20 cardiovascular deaths. The all-cause mortality was 35 (13.7%) in the non-AAD group and 21 (11.4%) in the AAD group, with no significant difference between the two groups ($P$ = 0.461). There were no statistically significant differences in cardiovascular mortality, early mortality, or late mortality between the two groups, with $P$ values of 0.764, 0.571, and 0.111, respectively.

The results of the survival analysis of the two groups are shown as K–M curves in Fig. 2. As shown in Fig. 2,

**Table 1**

**Baseline characteristics, procedural details, and clinical outcomes of patients with and without AAD**

| Variables | Non-AAD ($n = 255$) | AAD ($n = 185$) | P |
|---|---|---|---|
| Age | 72.5 ± 7.8 | 71.7 ± 7.7 | 0.271 |
| Male | 132 (51.8) | 129 (69.7) | < 0.001 |
| BMI | 23.6 ± 5.2 | 24.0 ± 5.0 | 0.377 |
| BAV | 60 (23.7) | 61 (33.0) | 0.032 |
| Hospitalization length | 13 (9.0, 18.0) | 11 (8.0, 16.0) | 0.045 |
| Prior history | | | |
| CAD | 108 (42.4) | 73 (39.5) | 0.543 |
| Hypertension | 135 (52.9) | 92 (49.7) | 0.506 |
| Atrial fibrillation | 30 (11.8) | 16 (8.6) | 0.292 |
| COPD | 13 (5.1) | 13 (7.0) | 0.403 |
| Myocardial infarction | 8 (3.1) | 5 (2.7) | 0.790 |
| Stroke | 24 (9.4) | 15 (8.1) | 0.635 |
| Echocardiographic assessment | | | |
| Diagnosis | | | 0.022 |
| AS | 100 (39.2) | 51 (27.6) | |
| AR | 53 (20.8) | 54 (29.2) | |
| AS and AR | 102 (40.0) | 80 (43.2) | |
| LVEF | 60 (50.0, 65.0) | 58 (46.5, 62.0) | 0.007 |
| LVEDD | 50 (44.0, 56.0) | 55 (48.0, 59.0) | < 0.001 |
| Maximum velocity | 4 (3.8, 5.0) | 4 (3.6, 5.0) | 0.082 |
| Mean gradient | 49 (31.0, 63.0) | 45 (28.5, 59.0) | 0.087 |
| Peak gradient | 79 (56.0, 101.0) | 76 (46.5, 98.0) | 0.077 |
| CT characteristics | | | |
| AAD/BMI | 1.5 (1.3, 1.6) | 1.8 (1.6, 2.1) | < 0.001 |
| Ascending aorta diameter | 35 (32.5, 37.8) | 43 (41.9, 46.9) | < 0.001 |
| Procedural details | | | |
| Access | | | 0.010 |
| Trans-femoral | 189 (74.1) | 116 (62.7) | |
| Trans-apical | 66 (25.9) | 69 (37.3) | |
| Valve type | | | 0.886 |
| BEV | 13 (5.1) | 10 (5.4) | |
| SEV | 242 (94.9) | 175 (94.6) | |
| Valve size | 26 (23.0, 29.0) | 27 (26.0, 29.0) | 0.003 |
| Clinical outcomes | | | |
| Poststroke | 6 (2.4) | 2 (1.1) | 0.477 |
| Post-PPI | 22 (8.6) | 10 (5.4) | 0.191 |
| Moderate PVL | 0 | 3 (1.6) | 0.074 |
| Postoperative echocardiographic assessment | | | |
| Post-LVEF | 60 (52.0, 64.0) | 57 (47.5, 62.0) | 0.007 |
| Post-LVEDD | 47 (43.0,52.0) | 51 (46.0, 56.0) | < 0.001 |
| Postmaximum velocity | 2 (1.8, 2.5) | 2 (1.7, 2.4) | 0.987 |
| Postpeak gradient | 16 (12.0, 24.0) | 17 (13.0, 25.0) | 0.172 |
| postpulmonary hypertension | 56 (22.0) | 25 (13.5) | 0.024 |
| Technical success | 198 (77.6) | 149 (80.5) | 0.397 |
| Device success | 185 (72.5) | 143 (77.3) | 0.219 |
| All-cause mortality | 35 (13.7) | 21 (11.4) | 0.461 |
| Cardiovascular mortality | 11 (4.3) | 9 (4.9) | 0.764 |
| Periprocedural mortality | 12 (4.7) | 9 (4.9) | 0.938 |
| Early mortality | 6 (2.4) | 6 (3.2) | 0.571 |
| Late mortality | 17 (6.7) | 6 (3.2) | 0.111 |

Only selected contents are presented herein. The remaining data can be found in Table S2, Supplemental Digital Content, https://links.lww.com/CARES/A24)

AAD = ascending aortic dilation; AR = aortic regurgitation; AS = aortic stenosis; BAV = bicuspid aortic valve; BEV = balloon-expandable valves; BMI = body mass index; CAD = coronary artery disease; COPD = chronic obstructive pulmonary disease; LVEDD = left ventricular end-diastolic diameter; LVEF = left ventricular ejection fraction; PPI = permanent pacemaker implantation; PVL = periovalvular leakage; SEV = self-expanding valves.

the *P* value of the log-rank test was 0.521, which is greater than 0.05, indicating no significant difference between the two groups. In the entire cohort, 21 patients died during the perioperative period. After excluding these 21 patients, we redrew the survival analysis K–M chart for the remaining 419 patients, as shown in Figure S1, Supplemental Digital Content, https://links.lww.com/CARES/A24 (see Supplementary materials). As indicated in Supplementary Figure S1, Supplemental Digital Content, https://links.lww.com/CARES/A24, the *P* value is 0.387, suggesting that after excluding patients who died perioperatively, there is still no significant statistical difference in overall mortality between the two groups of patients. To determine the independent risk factors

**Table 2**

**Predictive factors of periprocedural mortality and all-cause mortality in the total cohort**

**Logistic-periprocedural mortality**

| | Univariate model | | Multivariate model | |
|---|---|---|---|---|
| **Variables** | **OR (95% CI)** | ***P*** | **OR (95% CI)** | ***P*** |
| Poststroke | 13.800 (3.057, 62.290) | 0.001 | 9.854 (1.490, 65.145) | 0.018 |
| Postkidney damage | 34.167 (11.514, 101.390) | < 0.001 | 27.567 (8.798, 86.373) | < 0.001 |
| Post-LVEF | 0.962 (0.929, 0.996) | 0.027 | 0.973 (0.935, 1.013) | 0.187 |

**COX-All-cause mortality**

| | Univariate model | | Multivariate model | | Residual test |
|---|---|---|---|---|---|
| **Baseline characteristics** | **HR (95% CI)** | ***P*** | **HR (95% CI)** | ***P*** | ***P*** |
| Age | 1.060 (1.021, 1.101) | 0.003 | 1.054 (1.013, 1.096) | 0.009 | 0.739 |
| BMI | 0.943 (0.875, 1.017) | 0.126 | 0.991 (0.914, 1.074) | 0.828 | 0.836 |
| NYHA class | | 0.013 | | 0.054 | |
| I (Reference variable) | | | | | |
| II | 407.561 (0, $7.209 \times 10^{51}$) | 0.917 | 157.952 (0, $9.261 \times 10^{51}$) | 0.931 | 0.714 |
| III | 481.162 (0, $8.504 \times 10^{51}$) | 0.915 | 192.256 (0, $1.126 \times 10^{52}$) | 0.928 | 0.777 |
| IV | 1489.207 (0, $2.636 \times 10^{52}$) | 0.900 | 607.933 (0, $3.569 \times 10^{51}$) | 0.913 | 0.454 |
| BAV | 0.856 (0.468, 1.568) | 0.615 | | | |
| Hospitalization length | 1.061 (1.034, 1.087) | < 0.001 | 1.053 (1.020, 1.086) | 0.001 | 0.513 |
| Atrial fibrillation | 1.864 (0.940, 3.695) | 0.075 | 2.084 (0.885, 4.908) | 0.093 | 0.705 |
| COPD | 2.702 (1.223, 5.968) | 0.014 | 3.351 (1.421, 7.906) | 0.006 | 0.186 |
| Prior CABG | 2.748 (0.668, 11.307) | 0.161 | 6.855 (1.480, 31.751) | 0.014 | 0.671 |
| Prior cardiac surgery without CABG | 2.169 (0.865, 5.435) | 0.099 | 1.155 (0.334, 3.986) | 0.820 | 0.118 |
| Diagnosis | | 0.008 | | < 0.001 | |
| AS (reference variable) | | | | | |
| AR | 1.560 (0.868, 2.801) | 0.137 | 3.167 (1.586, 6.321) | 0.001 | 0.654 |
| AS and AR | 0.497 (0.241, 1.023) | 0.058 | 0.512 (0.243, 1.078) | 0.078 | 0.846 |
| Moderate to severe TR | 1.686 (0.921, 3.088) | 0.091 | 0.783 (0.347, 1.764) | 0.555 | 0.942 |
| Ascending aorta diameter | 0.990 (0.948, 1.033) | 0.637 | | | |
| AAD/BMI | 1.618 (0.835, 3.137) | 0.154 | 1.223 (0.517, 2.888) | 0.647 | 0.256 |
| Procedural details | | | | | |
| Valve size | 0.878 (0.788, 0.979) | 0.020 | 0.833 (0.733, 0.946) | 0.005 | 0.447 |
| PCI for the same period | 1.851 (0.833, 4.115) | 0.131 | 2.993 (1.219, 7.350) | 0.017 | 0.547 |
| Intraoperative CPB | 3.842 (1.526, 9.668) | 0.004 | 2.009 (0.691, 5.842) | 0.200 | 0.370 |

Only selected contents are presented herein. The remaining data can be found in Supplementary Table 4 and Supplementary Table 5 https://links.lww.com/CARES/A24.

AAD = ascending aortic dilation; AR = aortic regurgitation; AS = aortic stenosis; BAV = bicuspid aortic valve; BMI = body mass index; CABG = Coronary artery bypass grafting; CAD = coronary artery disease; CI = confidence interval; COPD = chronic obstructive pulmonary disease; CPB = cardiopulmonary bypass; HR = hazard ratio; LVEDD = left ventricular end-diastolic diameter; LVEF = left ventricular ejection fraction; NYHA = New York Heart Association; OR= odds ratio; PCI = percutaneous coronary intervention; TR = tricuspid regurgitation.

for all-cause mortality in the entire cohort, Cox analysis was performed on variables associated with baseline characteristics and procedural details. The univariate Cox model was first performed, and variables with *P* values < 0.2 were included in the multivariate Cox model. The hazard ratio (HR) and *P* values were then calculated, and the results are shown in Table 2.

As shown in Table 2, the independent risk factors for all-cause mortality in the whole cohort were age, hospitalization length, chronic obstructive pulmonary disease, prior coronary artery bypass grafting, diagnosis of AR, valve size, and percutaneous coronary intervention for the same period, where valve size was a protective factor, HR was 0.833 (0.733, 0.946), and the remaining variables were risk factors.

The results of the RCS analysis for the continuous variables age, hospitalization length, and valve size are presented in Supplementary Table S1, Supplemental Digital Content, https://links.lww.com/CARES/A24 (see Supplementary materials) and Fig. 3. The bias of other independent risk factors for all-cause mortality was corrected for each RCS analysis, and the number of nodes was set to four. The nonlinearity of age and valve size is < 0.05, so the corresponding image of the two is nonlinear, while the nonlinearity of hospitalization length is > 0.05, so the corresponding image is more likely to be a linear image. When HR = 1, age = 63.54, 73.04; hospitalization length = 12; valve size = 23.76, 26, 28.9. Therefore, for age, age < 63.54 or > 73.04 is a risk factor, and age between 63.54 and 73.04 is a protective factor. The image becomes steeper when age > 73.04. For hospitalization length, a hospitalization length of more than 12 days is a risk factor. For valve size, it is a risk factor when the valve size is < 23.76 or between 26 and 28.9 and a protective factor when the valve size is between 23.76 and 26 or > 28.9.

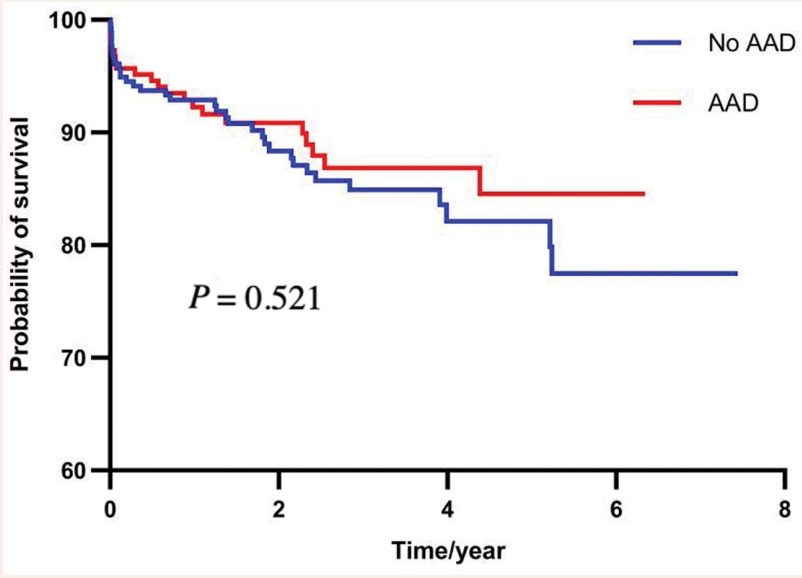



Figure 2. Survival analysis for the overall patients. AAD = ascending aortic dilation.

### 3.5. Subgroup analysis

The results of the subgroup analysis are shown in Figure S2, Supplemental Digital Content, https://links.lww.com/CARES/A24 (see Supplementary materials). As shown in Figure S2, Supplemental Digital Content, https://links.lww.com/CARES/A24, the *P* for interaction was > 0.05 in each subgroup, so no difference in the effect of ascending aorta diameter on all-cause mortality was observed across the different subgroups. In particular, no difference in the effect of ascending aortic diameter on all-cause mortality was observed in the BAV, tricuspid aortic valve (TAV), AS, or AR subgroups.

### 3.6. Progression of AAD

In the entire cohort, the follow-up aortic CTA data for three individuals could not be accessed because of issues with the electronic medical record system; therefore, a total of 437 patients completed the follow-up for aortic CTA (253 in the non-AAD group and 184 in the AAD group), and the results are shown in Table 3. As shown in Table 3, during the follow-up period, the aortic CTA results for the AAD group were significantly broader than those for the non-AAD group (43.9 mm vs. 35.2 mm, $P < 0.001$); the median follow-up time for the non-AAD group was longer than that for the AAD group, but there was no statistical difference. The postprocedural ascending aortic diameter in the AAD group was greater than that in the non-AAD group (1.2 mm vs. 0.8 mm). However, there was no statistically significant difference between the two groups ($P = 0.635$). In terms of the annual growth of the ascending aorta (0.44 mm/year vs. 0.47 mm/year), there was also no statistical difference between the two groups ($P = 0.861$). Throughout the follow-up period, no patients in the entire cohort experienced ascending aortic dissection.

## 4. Discussion

This study focused on the prognosis of patients with aortic valve disease and AAD undergoing TAVI. Our results revealed no significant differences in short-term outcomes or 3-year follow-up clinical outcomes in aortic valve disease patients with or without AAD who underwent TAVI. This study provides crucial insights for the application of TAVI in patients with AAD.

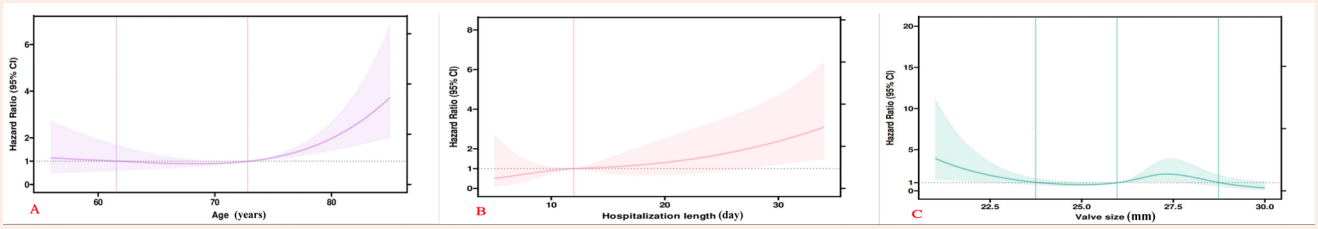



Figure 3. Restriction cubic spline plots of age (A), hospitalization length (B), and valve size (C), with the shaded portion representing the 95% CI of the hazard ratio.

**Table 3**

**The progression of AAD**

| | Overall patients | | |
|---|---|---|---|
| | Non-AAD ($n$ = 253) | AAD ($n$ = 184) | $P$ |
| Baseline CT (mm) | 35.20 (32.50, 37.80) | 43.90 (41.90, 46.90) | < 0.001 |
| Follow-up CT (mm) | 36.00 (33.00, 38.35) | 44.70 (42.40, 47.40) | < 0.001 |
| Time interval (year) | 2.00 (0.88, 3.00) | 1.63 (0.81, 3.00) | 0.601 |
| Change (mm) | 0.80 (0.00, 1.65) | 1.20 (–0.10, 1.60) | 0.635 |
| Progression (mm/year) | 0.44 (0.00, 1.60) | 0.47 (–0.03, 1.60) | 0.861 |

AAD = ascending aortic dilation.

### 4.1. BAV patients

Previous studies have shown that nearly 1/4 of patients treated with TAVI are diagnosed with AAD.[7] However, in our study, the proportion of patients with AAD was even higher, accounting for approximately 42%. The incidence of AAD in patients with AS is between 1/5 and 1/4.[8] The incidence of AAD in AS patients in our cohort was 33.8%, which was also higher than that reported in previous studies. Considering that AAD often co-occurs with BAV, in our cohort, BAV was more prevalent in AAD patients than in non-AAD patients, which aligns with previous findings. In the multivariate Cox analysis, BAV was not an independent risk factor for all-cause mortality in the entire cohort. In the subgroup analyses, we observed no impact of ascending aortic diameter on all-cause mortality in patients with BAV, which is consistent with the findings of Feng et al.[9] However, Jia et al.[10] found that a maximal ascending aorta diameter > 45 mm was associated with a 4.4-fold increased risk of mortality following TAVI. This difference may be due to the fact that in this study, the average ascending aorta diameter in BAV patients was 40.5 mm, which did not reach the 45 mm studied by Jia et al.[10] However, since only 24 patients in the study by Jia et al.[10] had a maximum ascending aortic diameter > 45 mm, these results were adjusted only for sex and age. Furthermore, cardiovascular mortality and adverse aortic events were not reported; thus, a cautious approach should be taken toward this conclusion. He et al.[11] identified the ascending aorta diameter as the sole predictor of 1-year mortality in patients with type 0 BAV, with an optimal cutoff of 42 mm; therefore, further studies are needed to ascertain whether the ascending aorta diameter impacts long-term prognosis in patients with BAV.

### 4.2. Patients with AR were not excluded from this study

Previous studies on AAD and prognosis after TAVI have focused on patients with isolated AS;[8,9,12] however, this study innovatively assessed the outcomes of patients with AR (isolated AR or AS and AR), providing a more comprehensive reflection of the anatomical complexity in the real world and thus filling this gap. AR can be caused by primary disease of the aortic valve cusps and/or abnormalities of the aortic root and ascending aortic geometry;[4] however, AS is more likely associated with valve calcification. The mechanism of AAD may differ between the two groups; for example, patients with AR may have worse aortic wall elasticity and a higher risk of postoperative dilation. A recent study indicated that AR itself negatively influences the smooth muscle cell phenotype in the ascending aortic wall.[13] Patients with AR experience left ventricular dilation caused by long-term volume overload, while those with AS primarily suffer from pressure overload, which may affect the extent of hemodynamic improvement after TAVI and the process of ascending aorta remodeling. Compared with patients with pure AS, those with AR face unique challenges in TAVI treatment. Patients with AR often have symmetrical dilation of the aortic root, a larger aortic annulus area, and a larger prosthetic valve for adequate anchoring. Therefore, it is reasonable not to exclude AR patients. Moreover, this study concluded that AR is an independent risk factor for all-cause mortality in the entire cohort, with an HR value and 95% confidence interval (CI) of 3.167 (1.586, 6.321), which inversely proves that it is reasonable not to exclude AR patients.

### 4.3. Short-term outcomes

Our results revealed no significant difference in short-term outcomes, consistent with the study of Yu et al.,[8] that reported that there was no difference in the 30-day mortality between AAD patients and non-AAD patients (2.4% vs. 1.4%, $P$ = 0.609). It is worth noting that AR patients often require larger prosthetic valves due to aortic root dilation and complex annular geometry (median valve size 27 mm in the AAD group vs. 26 mm in the non-AAD group in this study). However, there was no difference in the incidence of moderate PVL after TAVI in this study (1.6% vs. 0.0%, $P$ = 0.074). A recent international registry study on patients with AR (ALIGN-AR) also demonstrated that SEV has excellent sealing properties in the AR population, with a rate of moderate or greater PVL occurring within 30 days postoperatively being < 1%,[1] which supports the findings of this study. Notably, although the baseline left ventricular function in the AAD group was worse (lower LVEF and larger LVEDD), the extent of postoperative hemodynamic improvement was

comparable to that in the non-AAD group, suggesting that the immediate hemodynamic corrective effect of TAVI on patients with concurrent AAD was not affected by the ascending aortic diameter. Multivariate logistic regression further revealed that poststroke (OR = 9.854, 95% CI, 1.490–65.145) and postkidney damage (OR = 27.567, 95% CI, 8.798–86.373) were independent risk factors for perioperative mortality; however, the diameter of the ascending aorta itself was not included in the risk model, which may be related to the fact that TAVI maneuvers focus on the aortic root and avoid direct intervention in the ascending aorta. In addition, the proportion of the transfemoral approach in this study was not low (62.7% in the AAD group and 74.1% in the non-AAD group). However, the incidence of postvascular and access-related complications was low, suggesting that modern image-guided technology can optimize the choice of approach.

### 4.4. 3-year follow-up clinical outcomes

In the case of a median follow-up period exceeding 2 years (non-AAD group 1,051 days; AAD group 961 days), there was no significant difference in all-cause mortality between the AAD group and the non-AAD group (11.4% vs. 13.7%, $P = 0.461$) or in cardiovascular mortality (4.9% vs. 4.3%, $P = 0.764$). Survival analysis revealed a high degree of overlap in the K–M curves of both groups (log-rank $P = 0.521$), further supporting the long-term safety of TAVI in the AAD population. This finding is consistent with the 10-year follow-up data from the Nordic Aortic Valve Intervention (NOTION) trial, which showed no difference in 10-year all-cause mortality between patients undergoing TAVI and SAVR.[14] However, Ochiai et al.[12] reported that 2-year cumulative all-cause mortality was significantly higher in the AAD group in patients with TAV. The reason for this difference may be that Ochiai et al.[12] studied the vast majority of patients with TAV and the absence of SEV in the type of valve implanted. According to a previous study,[15] a larger AAD and a smaller degree of oversizing were confirmed to be the most relevant predictors of unsuccessful device implantation following SEV implantation. In theory, the SEV needs to be closely attached to the valvular–aortic complex at both the inflow and outflow levels;[15] moreover, the use of the SEV theoretically increases the complexity of transcatheter crossing and potential injury to the ascending aorta.[9] In our study, although the majority of patients were treated with SEV, there was no significant difference in short-term outcomes or 3-year follow-up clinical outcomes between the AAD group and the non-AAD group. Additionally, the type of valve was not an independent risk factor for all-cause mortality in the entire cohort. According to our research, the use of SEV for AAD patients appears to be feasible; however, since our study did not entirely randomize the selection of the types of implanted valves, and many factors affect the device success of TAVI, such as the depth of valve implantation, more rigorous studies are needed in the future to verify.

In addition, the annual growth rate of the ascending aorta after TAVI in the AAD group was slightly higher (0.47 mm/year vs. 0.44 mm/year). However, the difference was not statistically significant ($P = 0.635$), and no occurrence of ascending aortic dissection was observed during the follow-up period. This is similar to the findings of Rylski et al.,[7] who reported that the short-term risk of adverse events in the aorta after TAVI is manageable; however, long-term monitoring for high-risk populations (such as those with a baseline ascending aorta diameter ≥ 45 mm) should not be overlooked. According to previous studies,[16,17] the risk of adverse aortic events after SAVR is low, so a conservative therapy strategy for AAD at the time of aortic valve surgery is recommended.

Furthermore, this study revealed the complex associations between age, hospitalization length, and valve size and all-cause mortality through RCS analysis, providing new evidence for TAVI risk assessment. The results indicate that there is a "U-shaped" relationship between age and the risk of death: when the age is < 63.54 years or > 73.04 years, the risk significantly increases (HR > 1). In comparison, the risk relatively decreases between 63.54 and 73.04 years (HR < 1). This phenomenon may be related to the differences in physiological characteristics across different age groups. According to a previously published study, the burden of atherosclerotic cardiovascular disease (ASCVD) caused by metabolic risk factors such as high body mass index and high fasting plasma glucose has significantly increased among the young population.[18] In elderly patients, due to the more complex preoperative comorbidities, relatively weaker constitutions, and decreased multiorgan functional reserves, there is an evident increase in the risk of mortality. It suggests the need for personalized management strategies tailored to different age groups.

When the length of hospital stay exceeds 12 days, the risk of death increases linearly with the number of days. This finding shows that shortening the length of hospital stay under safe conditions can reduce the mortality of TAVI patients. The valve size exhibits nonlinear fluctuations in relation to the risk of death. This finding supports the concept of "individualized size selection," which necessitates a comprehensive decision-making process that integrates preoperative aortic CTA measurements of the annular area, calcification distribution, and geometric morphology of the aortic root.

### 4.5. Association between patient selection bias and ascending aortic diameter threshold

The 2021 European guidelines recommend that for patients who have an indication for aortic valve surgery, an aortic diameter ≥ 45 mm is considered to indicate concomitant surgery of the aortic root or tubular ascending aorta.[4] In the AAD group of this study, the median ascending aortic diameter was 43.9 mm (IQR: 41.9–46.9 mm), with 72 patients (16.4% of the total cohort, 38.9% of the AAD group) having a diameter

≥ 45 mm. This distribution aligns with our hospital practice: patients with a diameter ≥ 45 mm are prioritized for "SAVR combined with ascending aortic repair"; TAVI is only considered after multidisciplinary evaluation for those intolerant to surgery (e.g., age ≥ 70 years, severe chronic obstructive pulmonary disease, chronic kidney disease stage 4–5). Thus, though 72 patients with a diameter ≥ 45 mm were included in the TAVI cohort, they were all high-risk for surgery and accounted for a limited proportion—this bias may explain the lack of significant differences in short-term complications and 3-year all-cause mortality between the AAD and non-AAD groups.

### 4.6. Interpretation boundaries of the study's "negative results"

Based on the inclusion of 72 patients with an ascending aortic diameter ≥ 45 mm, the conclusion of this study that "there are no significant differences in short-term and 3-year outcomes after TAVI" has its applicability boundaries: All 72 patients were high-risk individuals intolerant to surgery, and their baseline characteristics differ significantly from those of patients with an ascending aortic diameter ≥ 45 mm who can tolerate SAVR, so the results cannot be directly generalized; Although no severe complications such as ascending aortic dissection occurred during the follow-up period, the sample size is insufficient to rule out low-probability high-risk events. Additionally, the median follow-up duration (961 days) does not cover the risk window for aortic-related adverse events, so the potential impact of an ascending aortic diameter ≥ 45 mm on the long-term prognosis of TAVI cannot be negated.

### 4.7. Future research directions based on existing data

For the 72 patients with diameter ≥ 45 mm, future research can focus on two aspects: Conduct long-term follow-up (≥ 5 years) and subgroup analysis for this cohort to evaluate the annual ascending aortic diameter growth rate and dissection incidence, clarifying long-term TAVI safety in this high-risk population; Expand the sample size via multicenter registry studies to compare TAVI with "SAVR combined with ascending aortic repair," focusing on aortic remodeling and long-term cardiovascular mortality, to provide evidence-based support for surgical strategy selection in patients with diameter ≥ 45 mm in guidelines.

### 4.8. Limitations

This study has the following limitations. First, as a single-center retrospective study, selection bias is difficult to avoid completely. Second, there was a lack of a control group undergoing SAVR combined with ascending aorta repair, making it impossible to directly compare the advantages and disadvantages of TAVI and SAVR in patients with AAD. Last, in this study, there were only 22 patients with an ascending aortic diameter > 50 mm, with a median follow-up duration of 961 days (approximately 2.6 years). Although no aortic-related complications occurred in this subgroup during the follow-up period, these two limitations mean that the current data cannot support the conclusion that "TAVI is safe in this type of patient." From the perspective of sample size, the scale of 22 cases is insufficient to rule out the possibility of low-probability high-risk events (such as aortic dissection), and in particular, it is impossible to conduct stratified analysis on the association between AAD and prognosis within this subgroup. From the perspective of follow-up duration, previous studies have shown that the risk peak of AAD-related complications (such as aortic dissection) mostly occurs more than 5 years after surgery. The current follow-up period has not covered this critical risk window, which may lead to the omission of long-term safety hazards.

Therefore, the conclusion of this study that "there is no significant association between AAD and long-term TAVI prognosis" cannot be generalized to patients with an ascending aortic diameter >50 mm, nor can it be used as a safety basis for such patients to choose TAVI. In the future, further verification through multicenter, large-sample, and long-term follow-up studies will be required.

## 5. Conclusions

In this study, we found no significant difference in short-term outcomes or 3-year follow-up clinical outcomes in aortic valve disease patients with AAD after TAVI. This study found that age, hospitalization length, and valve size may provide a new basis for the risk assessment of all-cause mortality in patients undergoing TAVI, suggesting that individualized management strategies should be developed for age, length of hospital stay, and valve size.

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## Ethical statement

This study was approved by the Medical Ethics Review Committee of Beijing Anzhen Hospital (Ethics Registry Number: 2025074x) and was exempt from the requirement of informed consent due to its nature. This study was conducted in accordance with the Declaration of Helsinki.

## Conflicts of interest

The authors have no conflicts of interest to disclose.

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## Data availability statement

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

## Author contributions

Writing—original draft, Software, Methodology, Formal analysis, Data curation: Haoyang Li.

Writing—review & editing, Conceptualization: Yuehuan Li.

Formal analysis, Data curation: Jiawei Zhou and Zhang Chen.

Writing—review & editing, Supervision, Project administration, Conceptualization: Haibo Zhang.

## AI statement

No generative AI or AI-assisted technologies were used in the preparation of this manuscript. All content was written and reviewed exclusively by the authors, who take full responsibility for the accuracy, integrity, and originality of the work.

## References


[1] Vahl TP, Thourani VH, Makkar RR, et al. Transcatheter aortic valve implantation in patients with high-risk symptomatic native aortic regurgitation (ALIGN-AR): a prospective, multic entre, single-arm study. *Lancet*. 2024;403:1451–9.

[2] Roberts WC, Vowels TJ, Ko JM, et al. Comparison of the structure of the aortic valve and ascending aorta in adults having aortic valve replacement for aortic stenosis versus for pure aortic regurgitation and resection of the ascending aorta for aneurysm. *Circulation*. 2011;123:896–903.

[3] Messner B, Bernhard D. Bicuspid aortic valve-associated aortopathy: where do we stand? *J Mol Cell Cardiol*. 2019;133:76–85.

[4] Vahanian A, Beyersdorf F, Praz F, et al.; ESC/EACTS Scientific Document Group. 2021 ESC/EACTS Guidelines for the management of valvular heart disease. *Eur Heart J*. 2022;43:561–632.

[5] Zoghbi WA, Chambers JB, Dumesnil JG, et al.; American Society of Echocardiography's Guidelines and Standards Committee. Recommendations for evaluation of prosthetic valves with echocardiography and doppler ultrasound: a report From the American Society of Echocardiography's Guidelines and Standards Committee and the Task Force on Prosthetic Valves, developed in conjunction with the American College of Cardiology Cardiovascular Imaging Committee, Cardiac Imaging Committee of the American Heart Association, the European Association of Echocardiography, a registered branch of the European Society of Cardiology, the Japanese Society of Echocardiography and the Canadian Society of Echocardiography, endorsed by the American College of Cardiology Foundation, American Heart Association, European Association of Echocardiography, a registered branch of the European Society of Cardiology, the Japanese Society of Echocardiography, and Canadian Society of Echocardiography. *J Am Soc Echocardiogr*. 2009;22:975–1014.

[6] Baumgartner H, Hung J, Bermejo J, et al. Recommendations on the echocardiographic assessment of aortic valve stenosis: a focused update from the European Association of Cardiovascular Imaging and the American Society of Echocardiography. *J Am Soc Echocardiogr*. 2017;30:372–92.

[7] Rylski B, Szeto WY, Bavaria JE, et al. Transcatheter aortic valve implantation in patients with ascending aortic dilatation: safety of the procedure and mid-term follow-up. *Eur J Cardiothorac Surg*. 2014;46:228–33.

[8] Yu J, Wang W. Short- to mid-term outcomes after transcatheter aortic valve replacement in patients with ascending aorta dilation: a single-centre retrospective analysis. *BMC Cardiovasc Disord*. 2023;23:31.

[9] Feng D, Zhao J, Niu G, et al. Outcomes for patients undergoing transcatheter aortic valve replacement with ascending aorta dilation. *Int J Cardiol*. 2024;405:131948.

[10] Jia Y, Tirado-Conte G, Montarello N, et al. Prognostic impact of ascending aortic dilatation in bicuspid TAVR patients. *JACC Cardiovasc Interv*. 2023;16:3057–9.

[11] He J, Xiong T-Y, Yao Y-J, et al. Outcomes following transcatheter aortic valve replacement for aortic stenosis in patients with Type 0 Bicuspid, Type 1 Bicuspid, and Tricuspid Aortic Valves. *Circ Cardiovasc Interv*. 2023;16:e013083.

[12] Ochiai T, Yoon S-H, Sharma R, et al. Prevalence and prognostic impact of ascending aortic dilatation in patients undergoing TAVR. *JACC Cardiovasc Imaging*. 2020;13:175–7.

[13] Balint B, Bernstorff IGL, Schwab T, Schäfers H-J. Aortic regurgitation provokes phenotypic modulation of smooth muscle cells in the normal ascending aorta. *J Thorac Cardiovasc Surg*. 2023;166:1604–16.e1.

[14] Thyregod HGH, Jørgensen TH, Ihlemann N, et al. Transcatheter or surgical aortic valve implantation: 10-year outcomes of the NOTION trial. *Eur Heart J*. 2024;45:1116–24.

[15] Maeno Y, Yoon S-H, Abramowitz Y, et al. Effect of ascending aortic dimension on acute procedural success following self-expanding transcatheter aortic valve replacement. *Int J Cardiol*. 2017;244:100–5.

[16] Longi F, Orelaru F, Clemence J, Naeem A, Wu X, Yang B. Outcomes of bicuspid aortic valve thoracic aorta (4.0–4.5 cm) after aortic valve replacement. *Ann Thorac Surg*. 2021;113:1521–8.

[17] Mckellar SH, Michelena HI, Li Z, Schaff HV, Sundt TM. Long-term risk of aortic events following aortic valve replacement in patients with bicuspid aortic valves. *Am J Cardiol*. 2010;106:1626–33.

[18] Li Z, Yang Y, Wang X, et al. Comparative analysis of atherosclerotic cardiovascular disease burden between ages 20–54 and over 55 years: insights from the Global Burden of Disease Study 2019. *BMC Med*. 2024;22:303.