Long-term Outcomes of Intervention between Open Repair and Endovascular Aortic Repair for Descending Aortic Syndrome: A Propensity-Matched Analysis

Shin-Ah Son
Kyungpook National University Hospital https://orcid.org/0000-0003-3317-6857
Hanna Jung
Kyungpook National University Hospital
Joon Yong Cho (sina0217@naver.com)
Kyungpook National University Hospital

Research article

Keywords: Descending aorta, endovascular repair, graft replacement, outcomes, reintervention

Posted Date: August 3rd, 2020

DOI: https://doi.org/10.21203/rs.3.rs-46690/v1

Version of Record: A version of this preprint was published on November 3rd, 2020. See the published version at https://doi.org/10.1186/s12893-020-00923-4.

Abstract

Background

The long-term complication rates of open repair and thoracic endovascular aortic repair (TEVAR) have not yet been determined. Therefore, this study aimed to compare long-term outcomes and aortic reintervention rates between open repair and TEVAR in patients with descending thoracic aortic diseases.

Methods

Between January 2002 and December 2017, 230 patients with descending thoracic aortic disease underwent surgery. Among them, 45 patients underwent open repair and 91 underwent TEVAR treatment at Kyungpook National University Hospital. The primary end points were in-hospital mortality, and short-term complications. The secondary end points were long-term mortality and reintervention rates. Based on the propensity score matching, 35 patients who underwent open repair were matched to 35 patients who underwent TEVAR (ratio = 1:1).

Results

The mean follow-up period was 70.2 ± 51.9 months. Shorter intensive care unit and hospital stay were seen in the TEVAR group than in the open repair group ($p < 0.001$ and $p < 0.001$, respectively). However, in-hospital mortality, and spinal cord ischemia were not significantly different among the two groups ($p = 0.068$ and $p = 0.211$ before matching, $p = 0.303$ and $p = 0.314$ after matching, respectively). The cumulative all-cause death and aorta-related death showed no significant difference ($p = 0.709$ and $p = 0.734$ before matching, $p = 0.888$ and $p = 0.731$ after matching, respectively). However, aortic reintervention rates were higher in the TEVAR group than in the open repair group before and after propensity score matching ($p = 0.006$ and $p = 0.013$, respectively).

Conclusion

The TEVAR group was superior in short-term recovery results but had higher reintervention rates compared to the open repair group. However, there was no significant differences between the groups in long-term survival.

Background

The treatment of descending thoracic aortic syndromes is challenging and depends on its pathologies. Since the introduction of thoracic endovascular aortic repair (TEVAR), many reports have demonstrated that it is a safe and feasible alternative to the conventional open repair [1–3]. Typically, the success of this procedure is dictated by its favorable outcomes and ease of use [4]. Although the use of TEVAR has rapidly increased due to improved perioperative morbidity rates, significant postoperative complications associated with TEVAR, such as endoleak, stent- graft migration, retrograde type aortic dissection, new-onset dissection, and stent- graft infection, contribute to relatively poor results of TEVAR [3, 5, 6]. In early clinical results with open repair versus TEVAR covered in previous reports [3, 6, 7], there are no long-term data comparing two procedures. In particular, the durability and long-term complication rates of open repair and TEVAR have not yet been determined. In this study, the primary end points were in-hospital mortality, and short-term complications. The secondary end points were long-term mortality and reintervention rates.

The purpose of this study was to compare long-term outcomes and reintervention rates between open repair and TEVAR in patients with descending aortic syndromes. To neutralize the effects of confounding independent variables such as unbalanced numbers (45:91) and age discrepancy, a propensity matched subsample of patients was created for an adequately powered analysis.

Methods

Study population

Between January 2002 and December 2017, 512 patients were diagnosed with descending thoracic aortic syndromes including dissection, pseudoaneurysm and penetrating atherosclerotic ulcer (PAU) at Kyungpook National University. The study design flowchart is demonstrated in Fig. 1. 230 patients underwent surgery. Among them, 136 patients were included for the study. 45 patients underwent open repair, and 91 underwent TEVAR. We retrospectively reviewed the medical records and undertook telephonic clinical assessments of these patients.

Definitions

Acute dissections was defined when the occurrence developed in 14 days from the first symptom. Complicated aortic syndromes were defined as the presence of one or more of the following conditions: aortic maximum size > 5.5 cm; resistant hypertension despite adequate medical therapy; recurrent or refractory pain; impending rupture; rupture with end-organ malperfusion; and extension of dissection. Aortic reintervention was defined as the need for any surgical or endovascular interventions following the initial procedure during follow-up. In the TEVAR group, an endoleak was defined as radiological evidence of blood flow outside the stent -graft according to published guidelines [8].

Operative strategies

The treatment modality was decided collaboratively by the cardiologist and cardiac surgeons who were involved in the patients’ care; decision was based on the patients’ co-morbidities, functional status, anatomical feature of the lesion, and the appropriate of vascular access [9].

Open aortic repair

Open aortic repair was generally performed via left thoracotomy or median sternotomy. All procedures employed with sequential clamping to minimize ischemic times. In case of left atrial-left femoral artery partial bypass (LHB), blood was drained from the left atrium via the inferior pulmonary vein and returned through the femoral artery. In case of circulatory arrest, bypass was initiated via the femoral artery and vein or the ascending aorta and right atrium.

TEVAR

Preoperative CT scans were reviewed for the preoperative assessment of access routes for the feasibility of TEVAR and to measure the bilateral vertebral artery for assessing the subclavian steal syndrome with a policy of selective subclavian artery revascularization. All TEVAR procedures were performed via the transfemoral approach under general anesthesia. Perioperative anticoagulation with heparin was prescribed at a dose of 3000–5000 units. The proximal landing zones in the aortic arch were classified as 0 to 4 according to Ishimaru's classification [10].

Statistical analysis

When comparing continuous variables, the Student's t-test and Wilcoxon test were used for parametric and nonparametric data, respectively, and are presented with the mean ± standard deviation (SD) or as median and interquartile range (IQR). Categorical variables were reported as absolute numbers or percentages and the Fisher's exact test or Chi-square test was used for comparison. The Kaplan-Meier method was used to estimate survival. For statistical analyses, p-values < 0.05 were deemed significant. Univariate and multivariate logistic regression models were utilized to determine independent risk factors. Hazard ratios (HRs) are presented with 95% confidence intervals (CIs).

To reduce the effect of selection bias and potential confounding in this retrospective cohort study, estimated propensity scores were used to match two groups. This was computed for each patient using a logistic regression model including the following variables: age, proximal maximal aortic size, aortic pathology, and proximal aortic tear site. The propensity score model was well-calibrated (Hosmer-Lemeshow goodness-of-fit test; $p = 0.784$) with good discrimination (c-statistic = 0.712). To neutralize the effects of confounding variables, 35 patients in the open repair group were matched to 35 patients who underwent TEVAR using propensity score matching (PSM). The data were analyzed using SAS/STAT software, v. 9.4 (SAS Institute Inc., NC, USA) and SPSS 25 (IBM, Armonk, NY, USA).

Results

Baseline characteristics of patients

Baseline characteristics of patients who underwent open repair and TEVAR are shown in Table 1. The mean follow-up duration was 70.2 ± 51.9 months (range, 0.0–212.0 months). The median ages of the patients were 56.0 (range, 43.0–64.0) years, and 65.0 (range, 57.0–72.0) years for the open repair and TEVAR groups, respectively, which was significantly different between the two groups ($p < 0.001$).

Moreover, connective tissue diseases were observed at a significantly higher rate in the open repair group than in the TEVAR group ($p < 0.001$). After PSM, the baseline characteristics of the patients in each group exhibited no significant difference.

Table 1
Baseline characteristics of patients

Characteristics	Overall series			After matching		
	Open repair N = 45	TEVAR N = 91	p-value	Open repair N = 35	TEVAR N = 35	p-value
Age (years)	56.0 (43.0–64.0)	65.0 (57.0–72.0)	< 0.001	60.0 (49.0–69.0)	59.0 (51.0–68.0)	0.967
Men	31 (30.1)	72 (69.9)	0.279	25 (71.4)	30(85.7)	0.145
Height (cm)	167 ± 10.0	166 ± 9.0	0.623	164.8 ± 10.1	168.3 ± 7.6	0.112
Weight (kg)	66.1 ± 13.2	64.9 ± 11.0	0.584	65.7 ± 12.9	68.9 ± 12.4	0.283
Initial SBP (mmHg)	133.5 ± 34.2	141.3 ± 29.3	0.208	135.7 ± 37.3	145.5 ± 27.1	0.215
Hypertension	30 (66.7)	66 (72.5)	0.480	26 (74.3)	25 (71.4)	0.788
Diabetes	5 (11.1)	12 (13.2)	0.731	5 (14.3)	1 (2.9)	0.088
Current smoking	20 (44.4)	36 (39.6)	0.586	16 (45.7)	16 (45.7)	1.000
Obesity	2 (4.4)	3 (3.3)	0.738	2 (5.7)	2 (5.7)	1.000
CAOD	4 (8.9)	10 (11.0)	0.705	3 (8.6)	6 (17.1)	0.284
PAOD	4 (18.2)	18 (81.8)	0.105	4 (11.4)	5 (14.3)	0.721
COPD	2 (4.4)	4 (4.4)	0.990	2 (5.7)	2 (5.7)	1.000
Cerebrovascular accident	1 (2.2)	11 (12.1)	0.056	1 (2.9)	4 (11.4)	0.164
Acute kidney injury	2 (4.4)	5 (5.5)	0.794	2 (5.7)	1 (2.9)	0.555
Chronic renal failure	4 (8.9)	7 (7.7)	0.810	2 (5.7)	3 (8.6)	0.643
Previous cardiac operation	10 (22.2)	18 (19.8)	0.740	3 (8.6)	6 (17.1)	0.284
Connective tissue disease	8 (17.8)	1 (1.1)	< 0.001	2 (5.7)	0 (0.0)	0.151
Preoperative status						
Shock	7 (15.6)	5 (5.5)	0.052	5 (14.3)	3 (8.6)	0.654
Hemoptysis	6 (13.3)	11 (12.1)	0.836	4 (11.4)	4 (11.4)	1.000
Persistent pain	37 (82.2)	73 (80.2)	0.780	30 (85.7)	27 (77.1)	0.356
Neurologic deficit	3 (6.7)	3 (3.3)	0.368	2 (5.7)	0 (0.0)	0.151

Characteristics	Overall series			After matching		
	Open repair N = 45	TEVAR N = 91	p-value	Open repair N = 35	TEVAR N = 35	p-value

Values are presented as the median (interquartile range), mean ± standard deviation or number of patients (%)

TEVAR: thoracic endovascular aortic repair; SBP: systemic blood pressure; CAOD: coronary artery occlusive disease; PAOD: peripheral artery occlusive disease; COPD: chronic obstructive pulmonary disease.

Descending thoracic aortic diseases details of patients are shown in Table 2. No variables showed significant difference between the groups, before and after PSM, respectively.

Table 2
Descending thoracic aortic diseases details of patients

Characteristics	**Overall series**			**After matching**		
	Open repair N = 45	**TEVAR N = 91**	**p-value**	**Open repair N = 35**	**TEVAR N = 35**	**p-value**
Acute	15 (33.3)	20 (22.0)	0.154	13 (37.1)	7 (20.0)	0.112
Rupture	19 (42.2)	21 (23.1)	0.021	16 (45.7)	9 (25.7)	0.081
Preoperative diagnosis						
Dissection	10 (22.2)	27 (29.7)	0.358	8 (22.9)	10 (28.6)	0.584
PAU	2 (4.4)	1 (1.1)	0.211	2 (5.7)	0 (0.0)	0.151
Pseudoaneurysm	33 (73.3)	63 (69.2)	0.621	25 (71.4)	25 (71.4)	1.000
Arch involvement	12 (26.7)	14 (15.4)	0.115	9 (25.7)	4 (11.4)	0.124
Maximal aortic size (mm)	54.9 ± 4.7	51.5 ± 4.5	0.471	52.8 ± 4.8	51.8 ± 3.7	0.985
Aortic tear site						
Arch	5 (11.1)	4 (4.4)	0.138	4 (11.4)	1 (2.9)	0.164
Isthmus	8 (17.8)	34 (37.4)	0.020	6 (17.1)	12 (34.3)	0.101
Descending	32 (71.1)	53 (58.2)	0.145	25 (71.4)	22 (62.9)	0.445
Malperfusion	5 (11.1)	3 (3.3)	0.068	3 (8.6)	0 (0.0)	0.077
CSF drainage	14 (31.1)	17 (18.7)	0.104	8 (22.9)	7 (20.0)	0.771
Emergency	17 (37.8)	28 (30.8)	0.414	16 (45.7)	9 (25.7)	0.081

Values are presented as mean ± standard deviation or number of patients (%)

TEVAR: thoracic endovascular aortic repair; PAU: penetrating atherosclerotic ulcer; CSF: cerebrospinal fluid.

Operative details are showed in Table 3. In the open repair group, circulatory arrest perfusion was performed most frequently (60.0%), and thoracotomy was the most common approach (60.0%). In the TEVAR group, zone 3 TEVAR was performed most frequently (40.7%).

Table 3
Operative details

Open repair	(N = 45)
Perfusion method	
Left atrium- Femoral artery partial bypass	15 (33.3)
Partial Shunt *	3 (6.67)
Circulatory arrest	27 (60.0)
Femoral artery-Femoral vein	9 (20.0)
Aorta-Right atrium	18 (40.0)
Operative Approach	
Median sternotomy	10 (22.2)
Median sternotomy + Thoracotomy	8 (17.7)
Thoracotomy	27 (60.0)
Cardiopulmonary bypass time (min)	191.1 ± 104.3
Cross clamp time (min)	60.5 ± 49.9
Circulatory arrest time (min)	25.5 ± 31.4
TEVAR	**(N = 91)**
Number of devices	1.2 ± 0.4
Proximal stent size (mm)	35.1 ± 4.7
Stent Length (mm)	140.2 ± 28.2
Zone 0	6 (6.6) Total debranching bypass with TEVAR
Zone 1	1 (1.1) BCA to LCCA and LCCA to LSCA bypass
Zone 2	25 (27.5) LCCA to LSCA bypass in 5 patients
Zone 3	37 (40.7)
Zone 4	22 (24.2)

Open repair	(N = 45)
Values are presented as mean ± standard deviation or number of patients (%)	
* Graft placed proximal and distal to the injury site	
TEVAR: thoracic endovascular aortic repair; BCA: brachiocephalic artery; LCCA: left common carotid artery; LSCA: left subclavian artery.	

Postoperative outcomes and complications

Postoperative outcomes and complications are shown in Table 4. The patients in the open repair group significantly required more operative time, needed longer ventilator care, stayed longer in the intensive care unit, and had longer periods of hospitalization than those in the TEVAR group (all $p < 0.001$, respectively), before and after PSM, respectively.

Table 4
Postoperative outcomes and complications

Characteristics	Overall series			After matching		
	Open repair N = 45	TEVAR N = 91	p-value	Open repair N = 35	TEVAR N = 35	p-value
Outcomes						
Operative time	420.6 ± 182.9	149.3 ± 92.9	< 0.001	386.7 ± 124.5	150.9 ± 88.7	< 0.001
Postop Hospital stay (day)	41.5 ± 42.7	18.3 ± 16.0	< 0.001	34.2 ± 26.5	15.1 ± 8.6	< 0.001
Total ICU stay (day)	19.6 ± 31.2	5.7 ± 14.0	< 0.001	17.7 ± 24.6	3.7 ± 5.4	0.002
Total ventilator care (min)	145.7 ± 334.1	33.6 ± 158.2	0.009	111.9 ± 225.4	11.9 ± 48.6	0.008
Reintubation	7 (15.6)	4 (4.4)	0.025	5 (14.3)	1 (2.9)	0.088
Complications						
Acute kidney injury	19 (42.2)	18 (19.8)	0.006	15 (42.9)	6 (17.1)	0.019
Dialysis	6 (13.3)	6 (6.6)	0.192	3 (8.6)	1 (2.9)	0.303
Hematologic complications	1 (2.2)	2 (2.2)	0.993	1 (2.9)	0 (0.0)	0.314
Bleeding	9 (20.0)	3 (3.3)	0.001	6 (17.1)	2 (5.7)	0.133
Spinal cord injury	2 (4.4)	1 (1.1)	0.211	1 (2.9)	0 (0.0)	0.314
Stroke	3 (6.7)	1 (1.1)	0.071	2 (5.7)	0 (0.0)	0.151
Pulmonary complications	9 (20.0)	7 (7.7)	0.036	7 (20.0)	2 (5.7)	0.074
Wound complications	12 (26.7)	6 (6.6)	0.001	9 (25.7)	3 (8.6)	0.057
In-hospital mortality	5 (11.1)	3 (3.3)	0.068	3 (8.6)	1 (2.9)	0.303
30-day mortality	5 (11.1)	4 (4.4)	0.138	3 (8.6)	2 (5.7)	0.643

Values are presented as mean ± standard deviation or number of patients (%)

TEVAR: thoracic endovascular aortic repair; Postop: postoperative; ICU: intensive care unit.

Acute kidney injury (AKI) was significantly higher in the open repair group than those in the TEVAR group, before and after PSM, respectively. Bleeding, pulmonary and wound complications were significantly more observed in the open repair group; however, there was no statistical difference after PSM. There was no statistical difference in-hospital mortality and 30-day mortality between the two groups, before and after PSM, respectively. In-hospital mortality was observed in five patients in the open repair group (11.1%), of whom four and one died of acute aorta-related complications and hospital-acquired pneumonia, respectively. In the TEVAR group, among three patients (3.3%), two patients died of delayed rupture after stent insertion and one patient died of aspiration pneumonia.

Reintervention

Reintervention data associated with specific treatments are depicted in Table 5. In the open repair group, five patients (11.1%) required reinterventions; and four patients experienced new onset aortic expansion. Among these patients, secondary open repair surgery was performed in three patients, and TEVAR was performed in one patient. In the TEVAR group, 22 patients (48.9%) required reintervention; twelve patients had endoleaks and four patient developed fistula, such as aortobronchial fistula (ABF) or aortoesophageal fistula (AEF).

Table 5
Aortic reintervention details

Cause of Reintervention	Open repair (N = 5, 11.1%)	TEVAR (N = 22, 48.9%)
New onset dissection/expansion	Aorta replacement:3 TEVAR:1	Aorta replacement:2 TEVAR:1
Fistula formation	Aorta replacement and lobectomy:1	Aorta replacement and lobectomy:4 Aorta replacement and esophagotomy:1 TEVAR:1
Endoleak		TEVAR :12 LSCA plug obliteration:1
Infection		Aorta replacement:1
TEVAR: thoracic endovascular aortic repair; LSCA: left subclavian artery.		

In the overall series, freedom from aortic reintervention at 1, 5, and 10 years in the open repair group was 97.1% ± 0.1%, 94.2% ± 0.1%, and 87.1% ± 0.0%, respectively. The corresponding rates in the TEVAR group were 93.2% ± 0.1%, 68.4% ± 0.1%, and 62.2% ± 0.0%. After PSM, the freedom from aortic reintervention at 1, 5, and 10 years in the open group was 100% ± 0.0%, 96.4% ± 0.1%, and 92.2% ± 0.1%, respectively. In the TEVAR group, the corresponding values were 91.0% ± 0.1%, 78.1% ± 0.1%, and 66.8% ± 0.1%. Freedom from aortic reintervention (Fig. 2) was lower in the TEVAR group than the open repair group before and after PSM ($p = 0.006$ and $p = 0.013$).

A multivariable Cox proportional hazard model is shown in Table 6.

Table 6
Cox proportional hazard regression analysis for reintervention

Variable	Overall series				After matching			
	Univariate		Multivariate		Univariate		Multivariate	
	HR (95% CI)	p-value	HR (95% CI)	p-value	HR (95% CI)	p-value	HR (95% CI)	p-value
Male	2.0 (0.6–5.8)	0.191			0.2 (0.0-2.2)	0.230		
Hemoptysis	2.8 (1.3–6.9)	0.026	3.8 (1.2–11.3)	0.016	8.5 (2.4–29.3)	0.001	6.8 (1.3–34.7)	0.021
Hoarseness	2.2 (0.8-9.0)	0.102			1.6 (0.2–13.2)	0.619		
Hypertension	0.2 (0.5–2.9)	0.595			4.4 (0.5–34.9)	0.151	7.9 (0.8–73.1)	0.048
Diabetes	2.0 (0.7–5.4)	0.148			1.3 (0.2–10.7)	0.766		
CAOD	2.5 (0.9–6.7)	0.059			2.2 (0.5–10.6)	0.210		
PAOD	1.7 (0.7–4.4)	0.210			1.9 (0.4–8.9)	0.400		
COPD	3.4 (1.0-11.4)	0.046			4.4 (0.9–21.2)	0.063		
Cerebrovascular accident	2.2 (0.7–6.4)	0.144			1.7 (0.2–14.2)	0.579		
Acute kidney injury	1.1 (0.1–7.4)	0.994			5.1 (0.6–41.2)	0.124		
Previous cardiac operation	1.1 (0.6–3.4)	0.388			3.2 (0.8–12.4)	0.082	6.81 (1.3–34.9)	0.021

HR: hazard ratio; CI: confidence interval; CAOD: coronary artery occlusive disease; PAOD: peripheral artery occlusive disease; COPD: chronic obstructive pulmonary disease.

Change of aorta size after the repair

Figure 3 shows the changes in the aorta size after the repair. During the following 5 years, the maximal aortic diameter was reduced in the open repair group compared to that in the TEVAR group before and after PSM ($p = 0.004$ and $p = 0.05$). The maximal aortic diameter decreased from 55.4 ± 18.2 mm to 40.07 ± 11.0 mm in the open repair group as we expect. Meanwhile the maximal aortic diameter increased from 52.0 ± 14.7 mm to 56.8 ± 19.1 mm in the TEVAR group.

Survival

In the open repair group, overall mortality was 42.2% (19/45), and aorta-related mortality was 17.8% (8/45) during follow-up. Aorta-unrelated death secondary to cancer occurred in three patients and three patients died of pneumonia. Late deaths secondary to unknown causes occurred in four patients. In the TEVAR group, overall mortality was 35.2% (32/91), and aorta-related mortality was 12.1% (11/91) during follow-up. Aorta-unrelated death secondary to cancer occurred in three patients and two patients died of pneumonia. Late deaths secondary to unknown causes occurred in 10 patients.

The cumulative survival of all-cause death was not statistically different, either before or after PSM, Additionally, the cumulative survival rates from aorta-related deaths were also not significantly different, before and after PSM, respectively (Fig. 4A,B).

A multivariable Cox proportional hazard model identified that age > 80 years, systolic blood pressure < 90 mmHg, diabetes, preoperative chronic renal failure, and aortic arch involvement were predictive factors in the in overall series; after PSM analysis, age > 80 years, and aortic arch involvement ($p = 0.024$ and $p = 0.048$, respectively) were independent predictors of aorta-related mortality (Table 7).

Table 7
Cox proportional hazard regression analysis for aorta-related mortality

	Overall series				**After matching**			
Variable	**Univariate**		**Multivariate**		**Univariate**		**Multivariate**	
	HR (95% CI)	p-**value**	HR (95% CI)	p-**value**	HR (95% CI)	p-**value**	HR (95% CI)	p-**value**
Age > 80 years	5.9 (1.3–25.9)	0.018	16.7 (2.7-104.3)	0.002	9.9 (1.2–83.4)	0.031	24.0 (1.5-378.1)	0.024
SBP < 90 mmHg	2.9 (0.9–8.9)	0.058	6.0 (1.6–22.4)	0.008	2.6 (0.5–13.3)	0.241		
Hemoptysis	2.5 (0.9-7.0)	0.077			5.2 (1.2–22.0)	0.023		
Dissection	0.7 (0.3–1.4)	0.356			0.4 (0.0-3.3)	0.392		
Pseudoaneurysm	1.4 (0.7–2.6)	0.333			2.8 (0.3–23.2)	0.329		
Arch involvement	3.5 (1.4–8.7)	0.007	5.4 (1.8–16.1)	0.002	5.1 (1.3–20.9)	0.022	14.5 (1.0-211.5)	0.048
Maximal aortic size > 50 mm	3.1 (0.9–10.8)	0.068			3.8 (0.5–31.9)	0.200		
Malperfusion	2.3 (0.5–10.1)	0.210			5.2 (0.6–43.9)	0.128		
Chronic renal failure	7.0 (2.4–20.3)	0.000	5.7 (1.2–26.8)	0.027	8.4 (1.5–46.7)	0.015		
eGFR	1.2 (1.1–1.3)	0.001			1.1 (0.9–1.5)	0.211		
Diabetes	2.8 (1.0-7.8)	0.047	3.1 (0.9–10.7)	0.049	4.2 (0.8–20.9)	0.080		
COPD	2.7 (0.6–11.9)	0.178			0.1 (0.0-34.8)	0.654		

Overall series	After matching
HR: hazard ratio; CI: confidence interval; SBP: systolic blood pressure; eGFR: estimated glomerular filtration rate; COPD: chronic obstructive pulmonary disease.	

Discussion

Since, the report on the first successful open repair of a descending thoracic aortic aneurysm with a prosthetic graft in 1953 by De Bakey and Cooley [11], an open surgical repair for treating descending thoracic aortic disease has been the gold standard for 50 years [11–14]. Despite remarkably improved operative techniques and maximized organ protection, open repair of the descending thoracic aorta is still associated with high complications, including intraoperative and postoperative death, hemorrhage, stroke, and paraplegia [9, 15].

Dake et al. [12] proposed an alternate method of TEVAR which sought to provide better clinical outcomes in patients who were deemed to be at high risk for open repair or were typically considered nonsurgical candidates. Therefore, TEVAR has shown significantly improved early quality of life versus open repair and a general trend toward better short-term perioperative survival and freedom from major complications [1, 3, 4, 16]. However, TEVAR has anatomic restrictions such as severe thoracic aortic tortuosity, short landing and sealing zones, and extensive mural thrombus. These are the limiting factors, although a seemingly infinite variety of debranching and bypass procedures can be applied to extend either the proximal or distal sealing zones [6, 17]. Furthermore, significant complications related to stent - grafts were always implied [3, 5, 15].

Patients who underwent TEVAR have a tendency to have a worse prognosis and older age, with multiple comorbidities, than patients who underwent open repair. Due to the relative lack of data supporting the long-term reliability of TEVAR, open repair procedure has been preferentially offered to younger patients [4, 18]. Therefore, to neutralize the effects of age difference which could potentially unmask a mortality benefit, PSM was performed between the two groups to perform an adequately powered analysis.

In present study, operative time, postoperative length of stay, and procedure-related complications showed better results with TEVAR before and after PSM. Not surprisingly, TEVAR was considering as the procedure involved no aortic cross-clamping, ischemic time, or thoracotomy [4]. In open repair cases, some disadvantages of deep hypothermia, including coagulopathy which caused difficulty in controlling bleeding, retraction injury to a heparinized lung, cold injury to the lung, and a profound inflammatory response from the bypass circuit [19]. For the in-hospital mortality of the open repair group, in present study, one patient died of pneumonia. AKI is another important complication and regarded as a marker of increased early or late morbidity and mortality after open repair or TEVAR [20]. Patients who underwent TEVAR were older and tended to receive larger amounts of contrast agent, which was not safe considering the risk of AKI. In the present study, postoperative AKI was higher in the open repair group (42.2%), and dialysis was performed in 13.3% of patients. Eighteen patients who underwent TEVAR

(19.8%) had an AKI with six requiring dialysis. Before and after PSM, AKI was higher in the open repair group; however, there was no statistical difference in dialysis between the two groups.

Although a short-term hospital outcome is more favorable for TEVAR, aorta-related complications are more frequent for TEVAR. Five patients (11.1%) in the open repair group underwent reintervention, and the most common cause of reintervention was new-onset aortic dissection or expansion. In the case of TEVAR, the most common cause of reintervention was endoleaks. Twenty-two (48.9%) patients who underwent reintervention showed no in-hospital mortality in the TEVAR; however, seven patients showed late mortality, one patient died of ABF and one patient died of sepsis due to stent-graft infection. Ascending aortic replacement was performed in one patient with retrograde aortic dissection, four patients with ABF underwent aorta replacement and lobectomy, and one patient with AEF underwent aorta replacement and esophagotomy, and showed late mortality. Additionally, eight patients who were initially offered TEVAR, later crossed over to open repair due to difficult anatomy or other reasons.

In the open repair cases, high-volume centers reported mortality and neurological morbidity rates ranging from 5.4–7.2% for mortality, 2.1–6.2% for permanent stroke, 5.7% for permanent paraparesis, and 0.8–2.3% for permanent paralysis, respectively [21, 22]. In the TEVAR cases, the perioperative results for the three stent- graft trials showed 1.9–2.1% for mortality, 2.4- 4% for stroke, 4.4–7.2% for permanent paraparesis, and 1.3- 3% for permanent paralysis, respectively [1, 2, 23]. In the present study, the open repair group showed 63.8% of overall 10-year survival rate, 84.3% of aorta-related 10-year survival rate, 6.7% of stroke, and 4.4% of SCI (after PSM, 65.4%, 88.5%, 5.7%, and 2.9%, respectively). In TEVAR, overall 10-year survival rate was 56.5%, aorta-related 10-year survival rate 81.3%, 1.1% stroke, and 1.1% SCI (after PSM, 64.2%, 88.7%, 0.0%, 0.0%, respectively). Although mortality was higher than previous large-scale studies, it was difficult to compare our results because previous reports did not have long-term follow-up data.

Moreover, although long-term survival of the two groups had no significant difference, more reinterventions occurred in the TEVAR group; the costs of additional graft modules to treat endoleaks and of follow-up computed tomography increase hospital cost, attributing to the disadvantage of TEVAR. In addition, TEVAR has less procedure-related complications than that of open repair; patients had more adverse events, such as re-dissection, fistula formation and stent-graft infection should be considered in the choice of approach.

Some authors have proposed that TEVAR does not change the natural history of the disease, and although less invasive, may be inferior to open therapies [24]. In our present study, maximal aortic size decreased more in the open repair group than in the TEVAR group, but not dramatically. This supports the report that it does not alter natural history of aortic pathologies, and, emphasizes the importance of long - term follow up. For this reason, in patients requiring TEVAR, the establishment of a precise TEVAR indication will reduce the requirement for further reintervention; better results are expected with improvements in debranching skills and stent- graft development.

Our study has several limitations. First, it was a single-center retrospective study that included a small number of patients, with a possible selection bias that might have affected our results. Second, the difference of follow-up duration and frequency can affect the survival rate of both groups. We performed PSM attempts to reduce the bias due to confounding variables. However, since TEVAR was introduced in 2007, it has a relatively short follow-up duration, whereas more frequent follow-up to monitor stent- grafts is expected to affect the results. Finally, the functional status of patient information influenced treatment strategy; there was no data interpretation, and studies on cost analysis, which is an increasingly important consideration for treatment strategy, have not been conducted.

Conclusions

In conclusion, our study showed that a long-term comparison between the open repair and TEVAR group demonstrated similar results in patients with descending aortic syndromes. However, patients who underwent TEVAR showed superior short-term recovery results and a higher reintervention rate than the open repair group. Larger multicenter population studies that consider quality of life could support our present study results.

Declarations

Ethics approval and consent to participate

The institutional review board (IRB) of Kyungpook National University Hospital approved this retrospective study and waived the requirement for individual patient consent (IRB approval No. 2019-10-051).

Consent for publication

Not applicable

Availability of data and materials

All data generated or analysed during this study are included in this published article and its supplementary information files.

Competing interests

The authors declare that they have no conflict of interest.

Funding

None of the authors have any financial interests to disclose.

Authors' contributions

SS collected the case data and drafted the original manuscript. JC and HJ operated and treated the patients and critically reviewed the manuscript. All authors read and approved the final manuscript.

Acknowledgements

We certify that this research is our own work and all sources of information used in this research have been fully acknowledged.

References

1. Fairman RM, Criado F, Farber M, Kwolek C, Mehta M, White R, Lee A, Tuchek JM, Investigators V. Pivotal results of the Medtronic Vascular Talent Thoracic Stent Graft System: the VALOR trial. J Vasc Surg. 2008;48(3):546–54.
2. Matsumura JS, Cambria RP, Dake MD, Moore RD, Svensson LG, Snyder S, Investigators TXCT. International controlled clinical trial of thoracic endovascular aneurysm repair with the Zenith TX2 endovascular graft: 1-year results. J Vasc Surg. 2008;47(2):247–57. discussion 257.
3. Makaroun MS, Dillavou ED, Kee ST, Sicard G, Chaikof E, Bavaria J, Williams D, Cambria RP, Mitchell RS. Endovascular treatment of thoracic aortic aneurysms: results of the phase II multicenter trial of the GORE TAG thoracic endoprosthesis. J Vasc Surg. 2005;41(1):1–9.
4. Gopaldas RR, Huh J, Dao TK, LeMaire SA, Chu D, Bakaeen FG, Coselli JS. Superior nationwide outcomes of endovascular versus open repair for isolated descending thoracic aortic aneurysm in 11,669 patients. J Thorac Cardiovasc Surg. 2010;140(5):1001–10.
5. Zhang L, Zhao Z, Chen Y, Sun Y, Bao J, Jing Z, Zhou J. Reintervention after endovascular repair for aortic dissection: A systematic review and meta-analysis. J Thorac Cardiovasc Surg. 2016;152(5):1279–88 e1273.
6. Hua HT, Cambria RP, Chuang SK, Stoner MC, Kwolek CJ, Rowell KS, Khuri SF, Henderson WG, Brewster DC, Abbott WM. Early outcomes of endovascular versus open abdominal aortic aneurysm repair in the National Surgical Quality Improvement Program-Private Sector (NSQIP-PS). J Vasc Surg. 2005;41(3):382–9.
7. Hansen CJ, Bui H, Donayre CE, Aziz I, Kim B, Kopchok G, Walot I, Lee J, Lippmann M, White RA. Complications of endovascular repair of high-risk and emergent descending thoracic aortic aneurysms and dissections. J Vasc Surg. 2004;40(2):228–34.
8. Fillinger MF, Greenberg RK, McKinsey JF, Chaikof EL, Society for Vascular Surgery Ad Hoc Committee on TRS. Reporting standards for thoracic endovascular aortic repair (TEVAR). J Vasc Surg. 2010;52(4):1022–33. 1033 e1015.
9. Lee HC, Joo HC, Lee SH, Lee S, Chang BC, Yoo KJ, Youn YN. Endovascular Repair versus Open Repair for Isolated Descending Thoracic Aortic Aneurysm. Yonsei Med J. 2015;56(4):904–12.
10. Mitchell RS, Ishimaru S, Ehrlich MP, Iwase T, Lauterjung L, Shimono T, Fattori R, Yutani C. First International Summit on Thoracic Aortic Endografting: roundtable on thoracic aortic dissection as an

indication for endografting. J Endovasc Ther. 2002;9(Suppl 2):II98–105.

11. Adams JD, Angle JF, Matsumoto AH, Peeler BB, Arslan B, Cherry KJ, Kern JA, Dake MD. Endovascular repair of the thoracic aorta in the post-FDA approval era. J Thorac Cardiovasc Surg. 2009;137(1):117–23.
12. Dake MD, Miller DC, Semba CP, Mitchell RS, Walker PJ, Liddell RP. Transluminal placement of endovascular stent-grafts for the treatment of descending thoracic aortic aneurysms. N Engl J Med. 1994;331(26):1729–34.
13. Cambria RP, Clouse WD, Davison JK, Dunn PF, Corey M, Dorer D. Thoracoabdominal aneurysm repair: results with 337 operations performed over a 15-year interval. Ann Surg. 2002;236(4):471–9. discussion 479.
14. Huynh TT, Miller CC 3rd, Estrera AL, Porat EE, Safi HJ. Thoracoabdominal and descending thoracic aortic aneurysm surgery in patients aged 79 years or older. J Vasc Surg. 2002;36(3):469–75.
15. Zhang H, Wang ZW, Zhou Z, Hu XP, Wu HB, Guo Y. Endovascular stent-graft placement or open surgery for the treatment of acute type B aortic dissection: a meta-analysis. Ann Vasc Surg. 2012;26(4):454–61.
16. Orandi BJ, Dimick JB, Deeb GM, Patel HJ, Upchurch GR Jr. A population-based analysis of endovascular versus open thoracic aortic aneurysm repair. J Vasc Surg. 2009;49(5):1112–6.
17. Stone DH, Brewster DC, Kwolek CJ, Lamuraglia GM, Conrad MF, Chung TK, Cambria RP. Stent-graft versus open-surgical repair of the thoracic aorta: mid-term results. J Vasc Surg. 2006;44(6):1188–97.
18. Harky A, Kai Chan JS, Ming Wong CH, Bashir M. Open versus Endovascular Repair of Descending Thoracic Aortic Aneurysm Disease: A Systematic Review and Meta-analysis. Ann Vasc Surg. 2019;54:304–15 e305.
19. Coselli JS, Bozinovski J, Cheung C. Hypothermic circulatory arrest: safety and efficacy in the operative treatment of descending and thoracoabdominal aortic aneurysms. Ann Thorac Surg. 2008;85(3):956–63. discussion 964.
20. Huynh TT, van Eps RG, Miller CC 3rd, Villa MA, Estrera AL, Azizzadeh A, Porat EE, Goodrick JS, Safi HJ. Glomerular filtration rate is superior to serum creatinine for prediction of mortality after thoracoabdominal aortic surgery. J Vasc Surg. 2005;42(2):206–12.
21. Wong DR, Parenti JL, Green SY, Chowdhary V, Liao JM, Zarda S, Huh J, LeMaire SA, Coselli JS. Open repair of thoracoabdominal aortic aneurysm in the modern surgical era: contemporary outcomes in 509 patients. J Am Coll Surg. 2011;212(4):569–79. discussion 579–581.
22. Estrera AL, Miller CC 3rd, Chen EP, Meada R, Torres RH, Porat EE, Huynh TT, Azizzadeh A, Safi HJ. Descending thoracic aortic aneurysm repair: 12-year experience using distal aortic perfusion and cerebrospinal fluid drainage. Ann Thorac Surg. 2005;80(4):1290–6. discussion 1296.
23. Bavaria JE, Appoo JJ, Makaroun MS, Verter J, Yu ZF, Mitchell RS, Gore TAGI. Endovascular stent grafting versus open surgical repair of descending thoracic aortic aneurysms in low-risk patients: a multicenter comparative trial. J Thorac Cardiovasc Surg. 2007;133(2):369–77.

24. Elefteriades JA, Farkas EA. Thoracic aortic aneurysm clinically pertinent controversies and uncertainties. J Am Coll Cardiol. 2010;55(9):841–57.

Figures

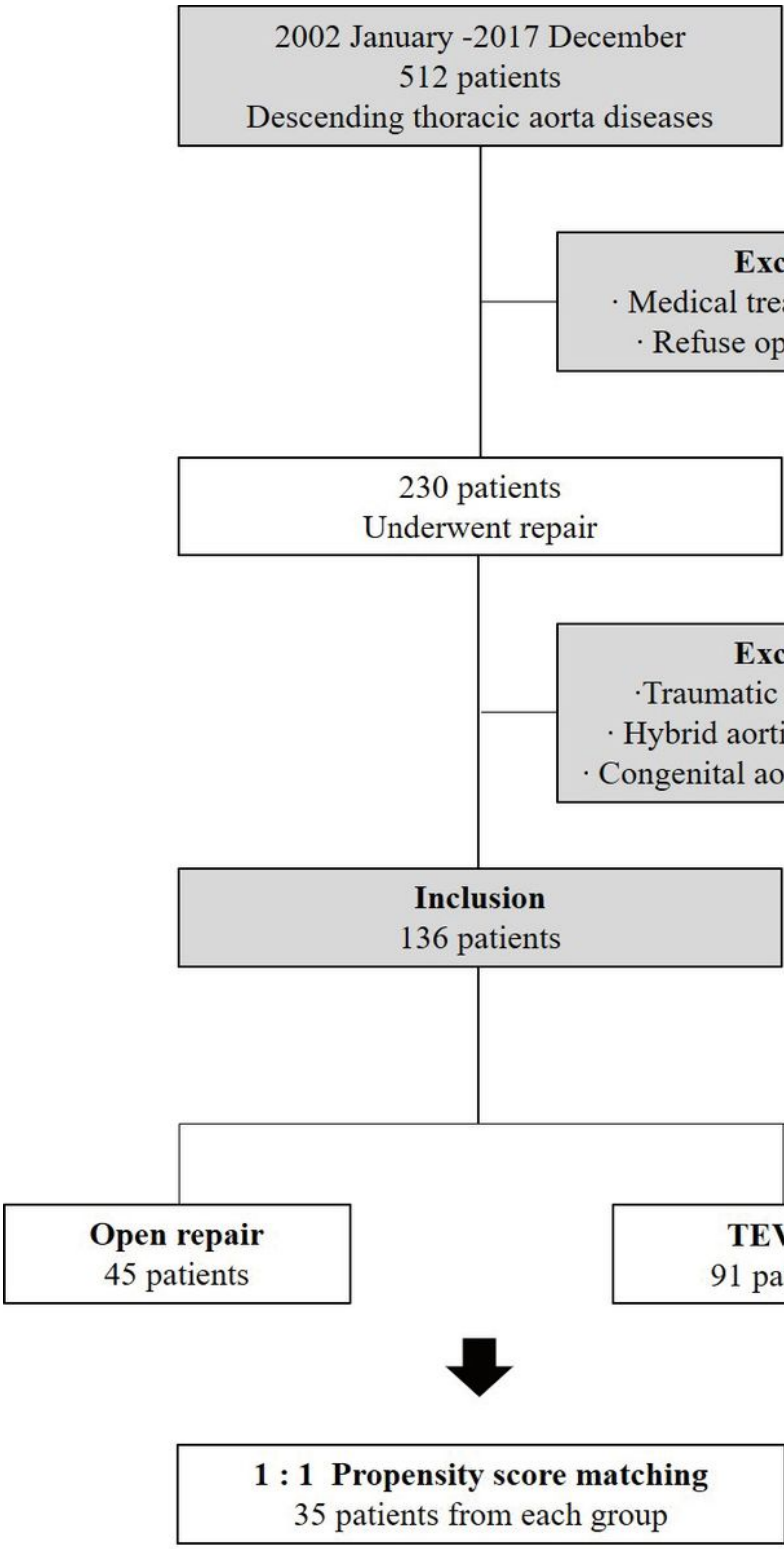

Figure 1

Study design flowchart

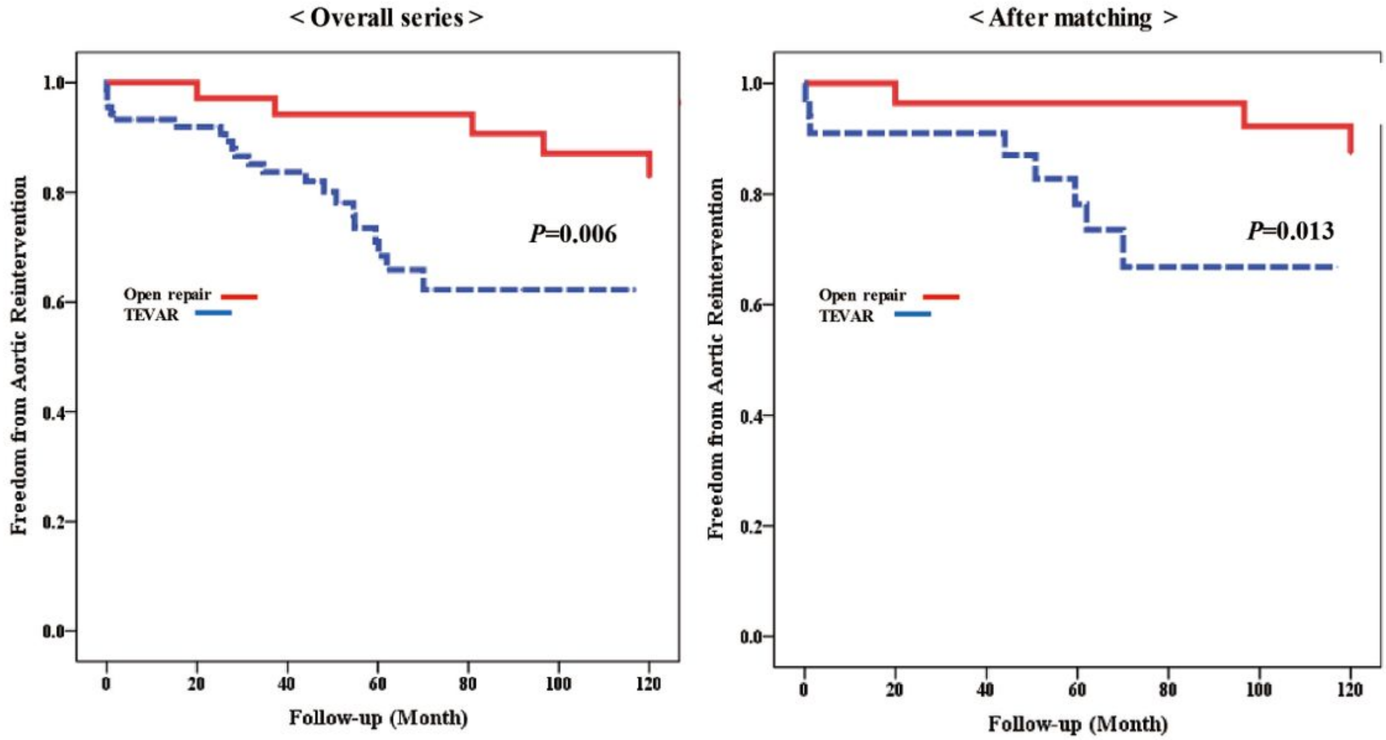

Figure 2

Freedom from aortic reintervention before propensity matching and after propensity matching

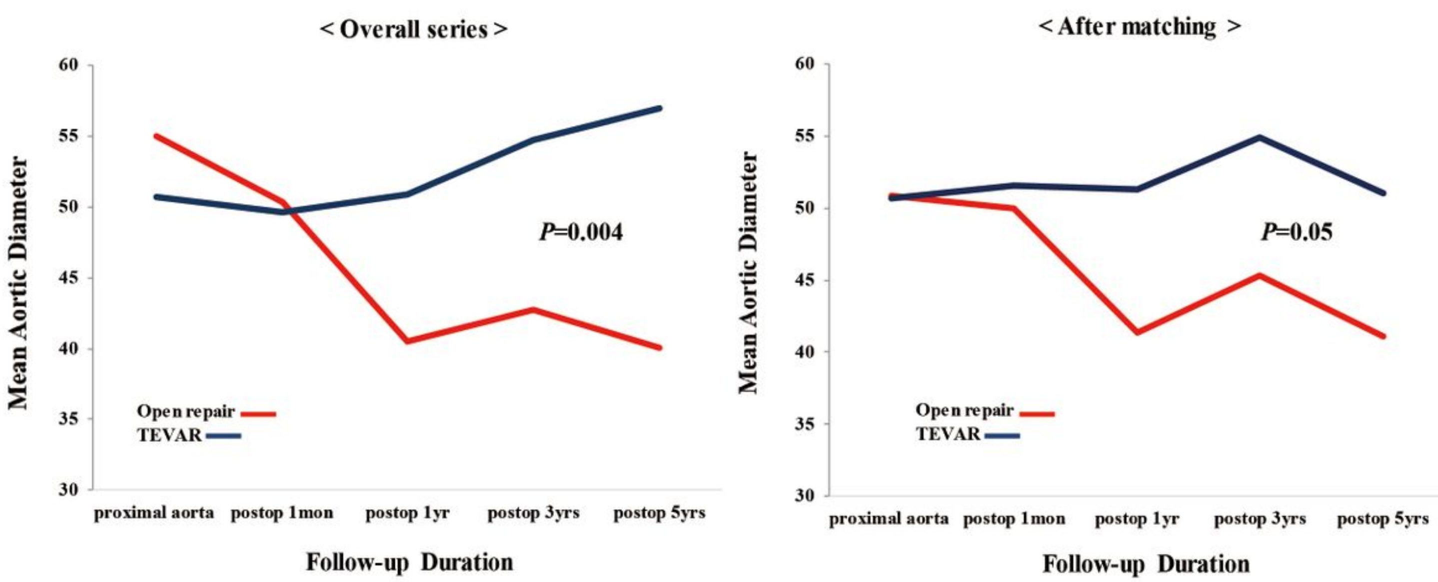

Figure 3

Changes in aortic diameters over time before propensity matching and after propensity matching

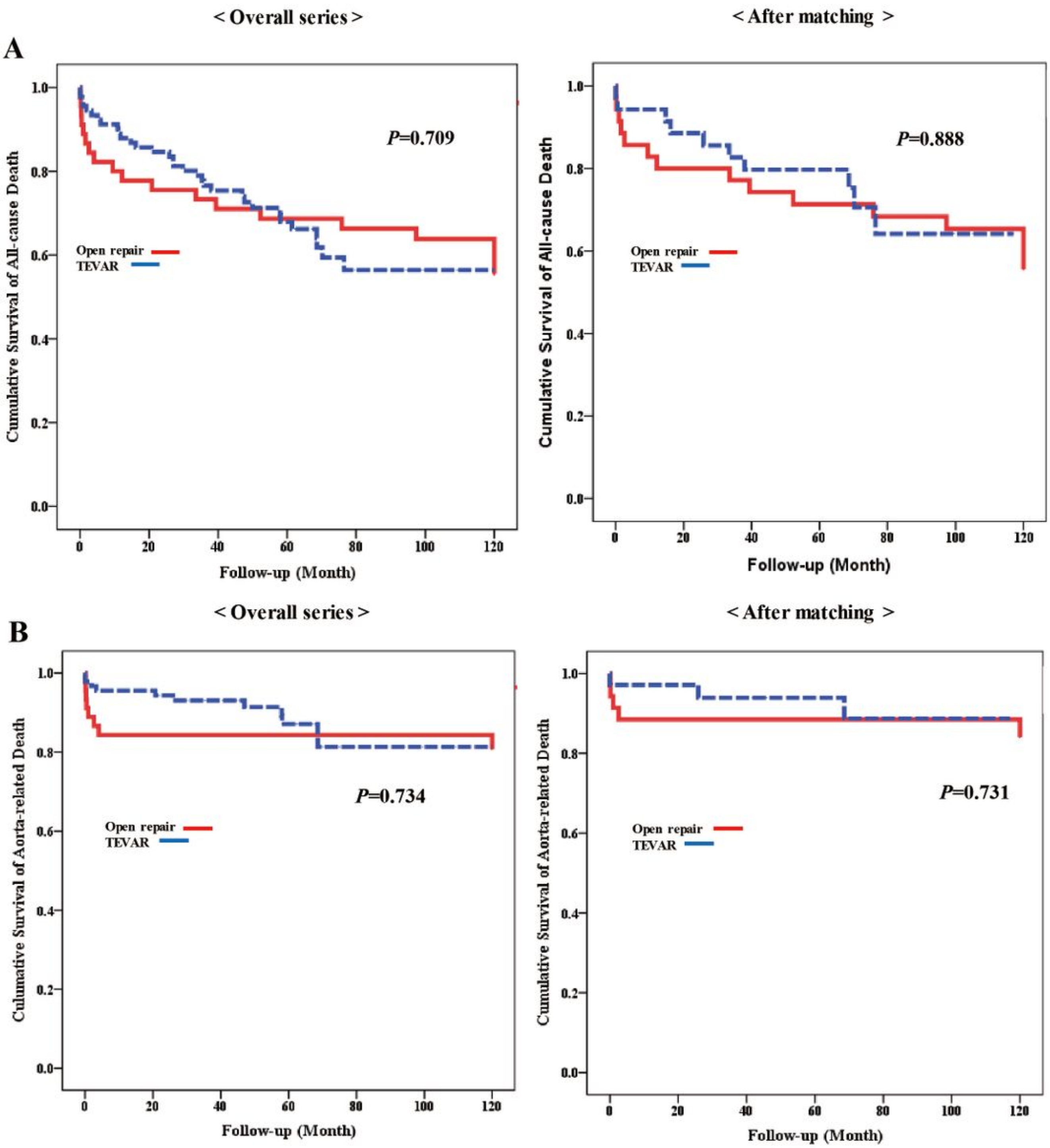

Figure 4

Cumulative survival of all-cause death and aorta-related death