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Original Article

Assessing Aortic Remodeling after Thoracic Endovascular Aortic Repair (TEVAR) in DeBakey IIIb Aortic Dissection: A Retrospective Study

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Purpose: This study analyzed the different levels of aortic remodeling in patients with DeBakey IIIb aortic dissection (AD) after thoracic endovascular aortic repair (TEVAR) at a single center.
Methods: In all, 66 patients with DeBakey IIIb AD who underwent TEVAR in the acute (Group A) or subacute phase (Group SA) from January 2012 to October 2016 were included in the study. The change in aortic lumen (A), true lumen (TL), false lumen (FL), and true lumen index (TLi) at different levels were analyzed.
Results: There was no statistically significant difference in the clinical information and morphologic imaging findings in Groups A and SA. At proximal levels (levels A–C), there was no difference in aortic remodeling parameters, that is, increased TL, decreased FL, and increased TLi at levels B and C and stable A at levels A–C, in both groups. Moreover, the above parameters were illustrated using a box-and-whisker plot, which revealed the unstable acute phase by the larger distribution interval and the median and abnormal values of the right skew distribution in Group A.
Conclusion: Postoperative surveillance is important for patients of both acute and subacute AD.

Keywords: aortic dissection, endovascular, DeBakey IIIb, aortic remodeling

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Introduction

Aortic dissection (AD) is a challenging life-threatening vascular emergency. DeBakey type I or II (Stanford type A) AD involving the ascending aorta is treated by urgent surgical intervention, while DeBakey type III (Stanford type B) AD involving the descending thoracic aorta (DeBakey type IIIa) or thoracoabdominal aorta (DeBakey type IIIb) is managed medically or by surgical or endovascular intervention when it is complicated. In recent years, thoracic endovascular aortic repair (TEVAR) has increasingly been used in the management of DeBakey type III (Stanford type B) AD.[1)] AD was considered acute if TEVAR was performed within 14 days from the onset of symptoms and chronic if TEVAR was performed more than 14 days

from the onset of symptoms. This definition is used in most trials and everyday clinical practice.[2)]

It has been proved that TEVAR is the treatment of choice in complicated acute DeBakey type III AD because of its rapid and obvious effect in the prevention of death from aortic rupture. Moreover, it was reported that there was a subacute phase in this AD type, which may be unstable and thus requires TEVAR.[3)] As TEVAR is widely used, long-term dissection complications are prevented in both acute and subacute dissection. There are a significant proportion of patients presenting with complications more than 14 days after symptom onset who needed to undergo TEVAR.[4)] In subacute DeBakey type III AD, TEVAR has overmatched both surgery for substantial morbidity and mortality and best medical treatment (BMT) for the development of progressive dissection and aortic rupture in the 5-year follow-up.[5,6)] Furthermore, some of the "uncomplicated DeBakey type III AD" benefited from TEVAR;[7)] however, the time when a patient with uncomplicated DeBakey type III AD should undergo TEVAR is unknown. Patterson et al. proposed that factors, such as length of aortic coverage and timing of treatment, should be considered in patient selection and operation time through a systematic review of aortic remodeling after TEVAR.[8)] Therefore, some patients would benefit from a longer time window of the treatment.

Then, what is the effect of TEVAR in the subacute phase? A few studies focused on the remodeling of acute and subacute DeBakey type III AD. A recent report from the VIRTUE Registry Investigators proposed that TEVAR in the subacute phase showed a similar degree of aortic remodeling to TEVAR in the acute phase and patients with acute and subacute dissection exhibited greater aortic plasticity than patients with chronic dissection, by comparing the absolute change in aortic area (mm^2) at different levels and over different periods of follow-up.[2)] The present study investigated the clinical characteristics, morphologic imaging findings, and aortic remodeling before and after TEVAR for acute and subacute DeBakey IIIb AD. Less effect of aortic coverage length of the DeBakey IIIb AD patients, and easily measured parameters of aorta remodeling made the assessment accessible.

Materials and Methods

Patients

Data of all patients who underwent TEVAR (Medtronic, GORE, VIABAHN, Life Tech Scientific Corporation, and MicroPort) from January 2012 to October 2016 were reviewed. In all, 66 patients with DeBakey IIIb AD, who were diagnosed using preoperational computed tomography angiography (CTA) (Discovery CT750 HD; GE Healthcare, Milwaukee, WI, USA), who underwent TEVAR in the acute or subacute phase, and who were followed-up by CTA at our hospital at least twice in 1 and 6 months after the operation separately from January 2012 to October 2016, were included in the study. Patients who underwent open surgery because the vascular condition was not appropriate for TEVAR were excluded. The enrolled patients were divided into the acute group (Group A, 45 patients) and subacute group (Group SA, 21 patients) based on the interval from symptom onset to TEVAR. This study was conducted in accordance with the Declaration of Helsinki. This study was conducted with approval from the Ethics Committee of Qingdao University. Written informed consent was obtained from all participants.

All patients were treated medically at the initial hospital. The indications of TEVAR included persistent or recurrent pain, uncontrolled hypertension despite full medication, early aortic expansion, malperfusion, and signs of rupture, such as hemothorax and increasing periaortic and mediastinal hematoma,[9)] which were the main manifestations of complicated DeBakey IIIb AD. All patients in Group A had complicated DeBakey IIIb AD, while not all patients in Group SA had uncomplicated conditions as complications may develop in the subacute phase. TEVAR was the suggested treatment of complicated DeBakey IIIb AD and generally performed in the acute phase to close the "primary" entry tear and redirect the blood flow to the true lumen (TL). The suggested treatment of uncomplicated DeBakey IIIb AD was optimal medical treatment with close surveillance on blood pressure, heart rate, and other possible signs predicting the progression of dissection. In contrast, once patients with uncomplicated DeBakey IIIb AD develop complications of aortic enlargement, susceptible rupture, and recent extension of the initial dissection, that is, becoming complicated, TEVAR was suggested. However, stable uncomplicated DeBakey IIIb AD could be treated by TEVAR to induce aortic remodeling processes. The data on demographics, comorbidities, and complications were collected from the medical records. Moreover, the CTA images were collected and measured on the picture archiving and communication systems (PACS).

Clinical practice

All operations were performed with the patient in the horizontal position under general anesthesia, with the

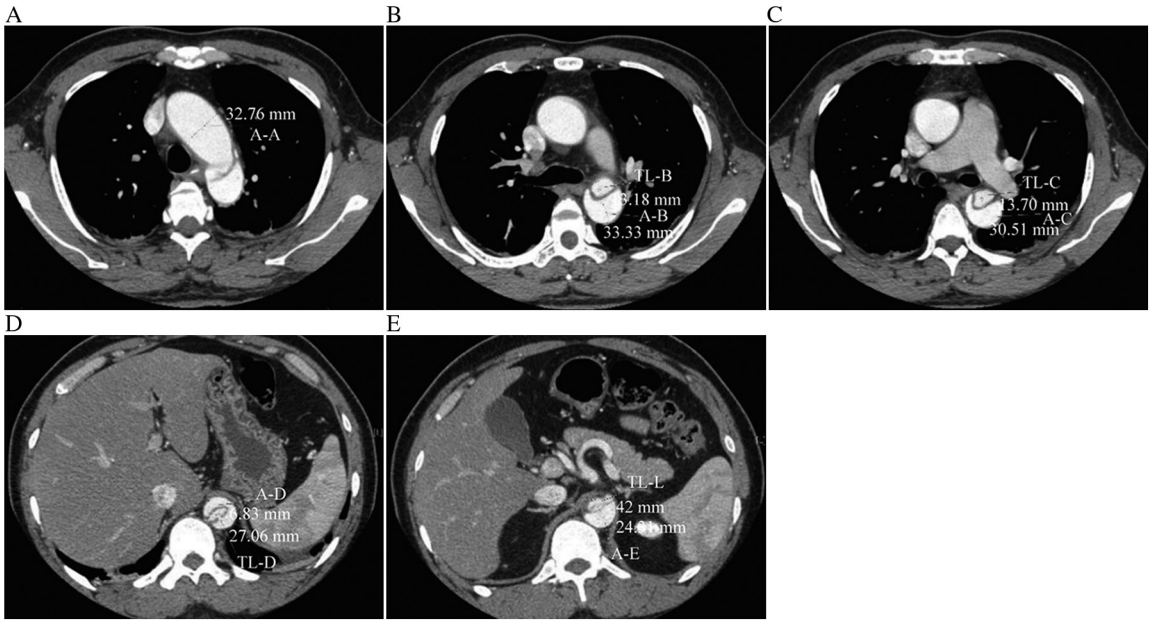

Fig. 1 Morphologic description and measure of A and TL of level A, B, C, D, and E were illustrated. A: aortic lumen; TL: true lumen

contrast pathway being the left brachial artery and the operation route being the femoral artery. The location of the main entry tears, TL, false lumen (FL), and origin of the left subclavian artery (LSA) were reconfirmed by digital subtraction angiography (DSA) through the aortic arch. Oversized devices (10%–15%), relative to the diameter of the undissected aorta proximal to the dissection (measured on preoperational CTA), were placed on the appropriate site to reduce flow into the FL and restore normal TL flow by stenting the aorta. Extremely proximal entry tears (when proximal landing zone <1.5 cm) may necessitate coverage of LSA to secure a safe proximal landing zone. We commonly used 15- to 20-cm-long stent graft devices in these patients, the distal landing zones of which were always above the diaphragm. Technical success was defined as endograft deployment at the intended aortic segment without antegrade flow into the FL.

Clinical information

Several symptoms, such as throbbing chest, abdominal, back, and waist pain and other types of pain, were observed. Smoking and drinking habits and possible comorbidities, such as hypertension, diabetes mellitus, coronary heart disease, cerebrovascular disease, and chronic obstructive pulmonary disease (COPD), were considered. The measured data such as age, height, weight, and Body Mass Index (BMI) were recorded. Morphologic description and measurement of the aorta are illustrated in **Fig. 1**. The development of malperfusion, pericardial effusion, and pleural effusion and the involvement of side branches were observed.

The parameters of aortic remodeling remain controversial until now. The measurements of the TL and FL were used by some studies; however, some of them were complicated and incomprehensible. This study chose the aortic lumen (A), TL, FL, and true lumen index (TLi) as the parameters, which were easy to measure and analyze in clinical work.

Several dimensions were measured on the CTA before the operation (T1) and 1 month (T2) and 6 months after the operation (T3) separately, which were the diameters of the A, TL, FL, and TLi (all diameters were the short diameter of the FL or TL perpendicular to the intimal flaps on the cross section), on the five different levels of the axial images as below: (A) aortic arch (the maximum transverse image of the aortic arch), (B) proximal descending thoracic aorta (the maximum transverse image of the thoracic aorta), (C) mid-descending aorta (level of the pulmonary arteries as easily identifiable landmarks), (D) aortic hiatus (the convergence of the bilateral diaphragm roots), and (E) celiac axis origin. TLi was defined as the percentage of TL accounted for A. At level A, only the maximum diameter of the aortic arch (A-A) was measured. At levels B–E, the diameters of A and TL were measured, and FL and TLi were calculated. The measurements were coded as diameter level. For example, A-A means the diameter of A

on the aortic arch (level A), TL-C means the diameter of TL on the mid-descending aorta (level C), and so on.

As mentioned above, five levels of the transverse image were analyzed in this study. In contrast, because all included patients had DeBakey IIIb AD, the effect of the hemodynamic alterations on the aorta between the aortic root and the initial part of the LSA was not evaluated. Despite the aortic arch level, the other four levels were in accordance with the levels F, G, H, and I suggested by the 2014 guideline and, at the same time, the typical levels used in many typical studies.[4,10] However, it was easier to process the study because of the easy measurement work. A reliable software is indeed needed, which could make the standard collection and large-scale investigation possible.

Statistical analysis

Age, height, weight, and BMI of Groups A and SA were analyzed by Shapiro–Wilk test to determine whether the data were normally distributed. Independent t-test was performed to analyze the normally distributed age and height, and nonparametric statistics-independent Kruskal–Wallis test was performed to analyze the non-normally distributed weight and BMI.

The categorical variables were summarized using frequencies and percentages and analyzed using the chi-square test or when necessary, Fisher's exact test. Normally distributed continuous variables were described using means and standard deviations and analyzed by independent t-test. Moreover, non-normal distributed continuous variables were described using medians and analyzed by nonparametric statistics-independent Kruskal–Wallis test. The difference in variables, such as A, TL, FL, and TLi, at different levels between subgroups and all three follow-up intervals were analyzed by repeated measurements. All statistical analyses were performed using the Statistical Package for Social Sciences, version 22, software (IBM SPSS Statistics; IBM Corporation, Armonk, NY, USA). Furthermore, the above parameters were illustrated in a box-and-whisker plot, in which the distribution interval and the upper part were analyzed.

Results

TEVAR for all patients was successful, and no mortality was noted. There were 62 (93.94%) men and 4 (7.06%) women. The mean age was 49.67 ± 11.61 years (range, 26–73 years). The mean interval from onset of the acute dissection to TEVAR was 11.41 ± 11.98 days (range, 1–75 days). The mean interval from symptom onset to TEVAR was 6.31 ± 3.68 days in Group A and 22.33 ± 15.89 days in Group SA.

Analysis of the clinical information

The chi-square test, or when necessary Fisher's exact test, was performed to analyze the enumerated variables, and there were no significant differences between Groups A and SA ($\alpha = 0.05$). No statistical significance was found ($\alpha = 0.05$) (**Table 1**).

Analysis of the morphologic imaging findings

Chi-square test or, when necessary, Fisher's exact test was performed to analyze these variables, and the difference in Groups A and SA was not statistically significant. Three patients (all in Group A [6.7%]) underwent LSA revascularization. In all, 61 patients had overstented LSA without revascularization (40 in Group A [88.89%], 21 in Group SA [100%]). Only one patient (in Group SA [4.8%]) required complete coverage of the LSA to obtain adequate seal of the entry tear. Two patients (in Group A [4.4%]) underwent bilateral carotid artery bypass. Two patients (one in Group A [2.2%] and another in Group SA [4.8%]) had locally dilated abdominal aorta. One patient (in Group SA [4.8%]) had retrograde type A dissection in the proximal aorta to the stent graft 42 days after TEVAR (**Table 2**).

Analysis of diameters of A, TL, FL, and TLi

The means and standard deviations of maximum short-axis diameter and different follow-up intervals of both groups are shown in **Table 3**. The variables at different levels of the aorta were classified into Groups A and SA and analyzed by repeated measurements. There was no significant difference between the parameters of Groups A and SA ($\alpha = 0.05$) between different surveillance times at each level. A significant increase in TL and TLi and reduction in FL can be observed at levels B and C in both groups according to different surveillance intervals. A significant increase in TL was still obvious while reduction in FL slowed down at level D, that is, FL on T3 reduced significantly than that on T1 and T2 in Group A while that of all the three surveillance intervals remained unchanged in Group SA, which causes the unclear change in FL and TLi at level D in both groups. At level E, TL and TLi on T3 increased significantly than that on T1 and T2 in Group A while the corresponding parameters in Group SA showed no significant change, and FL in both groups showed no significant reduction. A at different levels showed difference: at levels A and B, A showed no

Table 1 Analysis of the clinical information between Group A and Group SA

	Group A (n = 45)	Group SA (n = 21)
Age, y, mean ± SD[b]	50.022 ± 12.228	48.905 ± 10.4685
Male, no. (%)[e]	43 (95.6)	19 (90.5)
Height, cm, mean ± SD[b]	171.500 ± 5.411	171.095 ± 5.486
Weight, kg, median[c]	72.250	80.000
BMI, %, median[c]	25.026	26.670
Onset to TEVAR, d, median[c]	6.000	16.000[a]
Tobacco use, no. (%)[d]	31 (46.7)	15 (71.4)
Alcohol use, no. (%)[d]	20 (44.4)	7 (33.3)
Hypertension, no. (%)[d]	29 (64.4)	17 (81.0)
Diabetes mellitus, no. (%)[e]	4 (8.9)	1 (4.8)
Coronary artery disease, no. (%)[e]	4 (8.9)	0.000
Cerebrovascular disease, no. (%)	0.000	0.000
COPD, no. (%)	0.000	0.000
Chest pain, no. (%)[d]	27 (60.0)	11 (52.4)
Abdominal pain, no. (%)[e]	13 (28.9)	2 (9.5)
Back pain, no. (%)[d]	25 (55.6)	14 (66.7)
Waist pain, no. (%)[e]	9 (20)	3 (14.3)
Other pain, no. (%)[e]	6 (13.3)	3 (14.3)

[a]p <0.05, vs. Group A, [b]independent t-test, [c]Kruskal–Wallis test, [d]chi-square test, [e]Fisher's exact test. TEVAR: thoracic endovascular aortic repair; COPD: chronic obstructive pulmonary disease; BMI: body mass index

Table 2 Morphologic imaging findings of Group A and Group SA , n. (%)

	Group A (n = 45)	Group SA (n = 21)
Malperfusion[b]	0 (0)	2 (9.5)
Pericardial effusion[b]	2 (4.4)	1 (4.8)
Pleural effusion[a]	29 (64.4)	11 (52.4)
Celiac axis involved[a]	12 (26.7)	5 (23.8)
Superior mesenteric artery involved[b]	4 (8.9)	2 (9.5)
Left renal artery involved[a]	13 (28.9)	4 (19.0)
Right renal artery involved[b]	3 (6.7)	5 (23.8)
Left iliac artery involved[a]	17 (37.8)	3 (14.3)
Right iliac artery involved[a]	16 (35.6)	6 (28.6)
Narrow true lumen Branches[b]	6 (13.3)	3 (14.3)

[a]Chi-square test, [b]Fisher's exact test.

change in both groups; at level C, A on T2 increased significantly than that on T1 in Group A, and the corresponding parameters in Group SA showed no significant change. At levels D and E, A on T2 and T3 increased significantly than that on T1 in both groups (**Table 3**).

Moreover, the above parameters are illustrated in a box-and-whisker plot in **Fig. 2**. The outliers in Group A appeared on the upper side more frequently than those in Group SA, mostly for parameters A and FL. At the same time, the distribution interval of Group A was usually larger than that of Group SA, and the median and abnormal values of Group A tended to be right skew distribution, mostly for parameters A and FL at levels B–E, and with respect to the former parameter (A) at levels D and E and at the time of T3, it was more obvious. It may manifest the unstable acute phase, the unstable proximal part, and the unstable period of 6 months to 1 year after TEVAR. At level E, although most parameters did not change significantly over time, the distribution interval of A on T3 (in 6 months) in Group SA after TEVAR was smaller than that in Group A, and the distribution interval of both TL and FL is roughly equal to that of Group A, indicating a similar vascular remodeling of TEVAR in both acute and subacute phases.

Discussion

TEVAR in the acute or subacute phase

Previously, AD was considered as acute if treatment was performed within the first 14 days from symptom

Table 3 Analysis of aortic dimensions following TEVAR

		T1	T2	T3
A-A	Group A	33.58 ± 3.77	32.5 ± 4.00	33.32 ± 3.57
	Group SA	32.89 ± 4.37	32.83 ± 4.44	32.57 ± 3.92
A-B	Group A	40.46 ± 6.36	41.97 ± 8.43	41.04 ± 8.18
	Group SA	43.02 ± 9.76	44.27 ± 11.11	42.28 ± 12.18
TL-B	Group A	16.80 ± 5.82	28.39 ± 6.03[a]	32.70 ± 6.25[a,b]
	Group SA	19.33 ± 6.55	29.23 ± 8.51[a]	33.34 ± 5.19[a,b]
FL-B	Group A	23.65 ± 8.41	13.58 ± 11.62[a]	8.43 ± 10.78[a,b]
	Group SA	23.69 ± 11.80	15.04 ± 12.91[a]	8.94 ± 12.64[a,b]
TLi-B	Group A	0.42 ± 0.15	0.71 ± 0.21[a]	0.83 ± 0.21[a,b]
	Group SA	0.46 ± 0.16	0.69 ± 0.23[a]	0.83 ± 0.20[a,b]
A-C	Group A	36.59 ± 7.35	38.77 ± 8.42[a]	37.45 ± 8.29
	Group SA	36.81 ± 5.91	38.74 ± 6.68	37.06 ± 8.56
TL-C	Group A	14.67 ± 5.63	25.20 ± 4.36[a]	29.13 ± 4.56[a,b]
	Group SA	15.37 ± 5.46	25.16 ± 7.26[a]	28.43 ± 5.46[a,b]
FL-C	Group A	21.92 ± 8.50	13.57 ± 11.03[a]	8.32 ± 10.97[a,b]
	Group SA	21.45 ± 7.30	13.58 ± 9.97[a]	8.63 ± 10.84[a,b]
TLi-C	Group A	0.41 ± 0.15	0.69 ± 0.20[a]	0.82 ± 0.22[a,b]
	Group SA	0.42 ± 0.15	0.67 ± 0.23[a]	0.80 ± 0.23[a,b]
A-D	Group A	31.39 ± 4.82	33.13 ± 4.85[a]	33.66 ± 5.95[a]
	Group SA	30.51 ± 5.23	33.04 ± 5.28[a]	34.05 ± 4.42[a]
TL-D	Group A	12.72 ± 4.73	15.47 ± 5.13[a]	18.50 ± 5.90[a,b]
	Group SA	13.38 ± 3.51	15.74 ± 5.21	18.22 ± 6.66[a,b]
FL-D	Group A	18.67 ± 6.23	17.66 ± 6.51	15.16 ± 8.98[a,b]
	Group SA	17.13 ± 7.47	17.30 ± 6.26	15.83 ± 8.20
TLi-D	Group A	0.41 ± 0.15	0.47 ± 0.17[a]	0.57 ± 0.23[a,b]
	Group SA	0.45 ± 0.14	0.48 ± 0.16	0.54 ± 0.21[b]
A-E	Group A	30.39 ± 5.08	32.14 ± 5.47[a]	32.91 ± 5.29[a]
	Group SA	31.14 ± 5.28	33.12 ± 5.88[a]	33.03 ± 4.01[a]
TL-E	Group A	13.17 ± 5.05	15.52 ± 5.16[a]	16.59 ± 5.62[a]
	Group SA	12.97 ± 4.71	14.78 ± 3.67	16.13 ± 6.49
FL-E	Group A	17.22 ± 6.72	16.62 ± 6.61	16.32 ± 7.65
	Group SA	18.18 ± 8.40	18.34 ± 6.62	16.90 ± 7.28
TLi-E	Group A	0.44 ± 0.18	0.49 ± 0.16[a]	0.52 ± 0.20[a]
	Group SA	0.43 ± 0.03	0.45 ± 0.12	0.49 ± 0.19

[a]$p < 0.05$, vs. T1, [b]$p < 0.05$, vs. T2. A: aortic lumen; TL: true lumen; FL: false lumen; TLi: true lumen index; TEVAR: thoracic endovascular aortic repair

onset and as chronic if treatment is performed after the first 14 days. It is reported that 74% of deaths from complications of any type of AD occurred within 2 weeks.[11] However, it is now accepted to further divide the course of AD into acute (≤14 days), subacute (15–90 days), and chronic (>90 days) phases.[4,12,13] The method was recommended by the 2014 European Society of Cardiology (ESC) guidelines. With the development of TEVAR, more and more patients with DeBakey IIIb AD were saved using this treatment. It was accepted widely that complicated DeBakey IIIb AD should be treated by TEVAR in the acute phase as soon as possible if the condition of the artery is good enough. The effect of TEVAR in the chronic phase was unsatisfactory due to its limited aortic remodeling and lower cumulative survival at 30 months follow-up.[13] However, the treatment and appropriate intervention time of uncomplicated DeBakey IIIb AD remained controversial.

The Investigation of Stent Grafts in Patients with DeBakey type III AD (INSTEAD) trial randomized a total of 140 patients with subacute (>14 days) uncomplicated DeBakey type III AD.[14] Two-year follow-up results indicated that TEVAR is effective (aortic remodeling in 91.3% of patients who underwent TEVAR vs. 19.4% of patients who underwent BMT; $p < 0.001$); however, TEVAR showed no clinical benefit over medical therapy as the difference in survival rates, all-cause deaths, and aorta-related death rate was underpowered. About 4 years later, a subsequent follow-up of this study (INSTEAD-XL) showed that aorta-related mortality and disease progression were significantly lower after 5 years in patients who underwent TEVAR compared with those in patients who

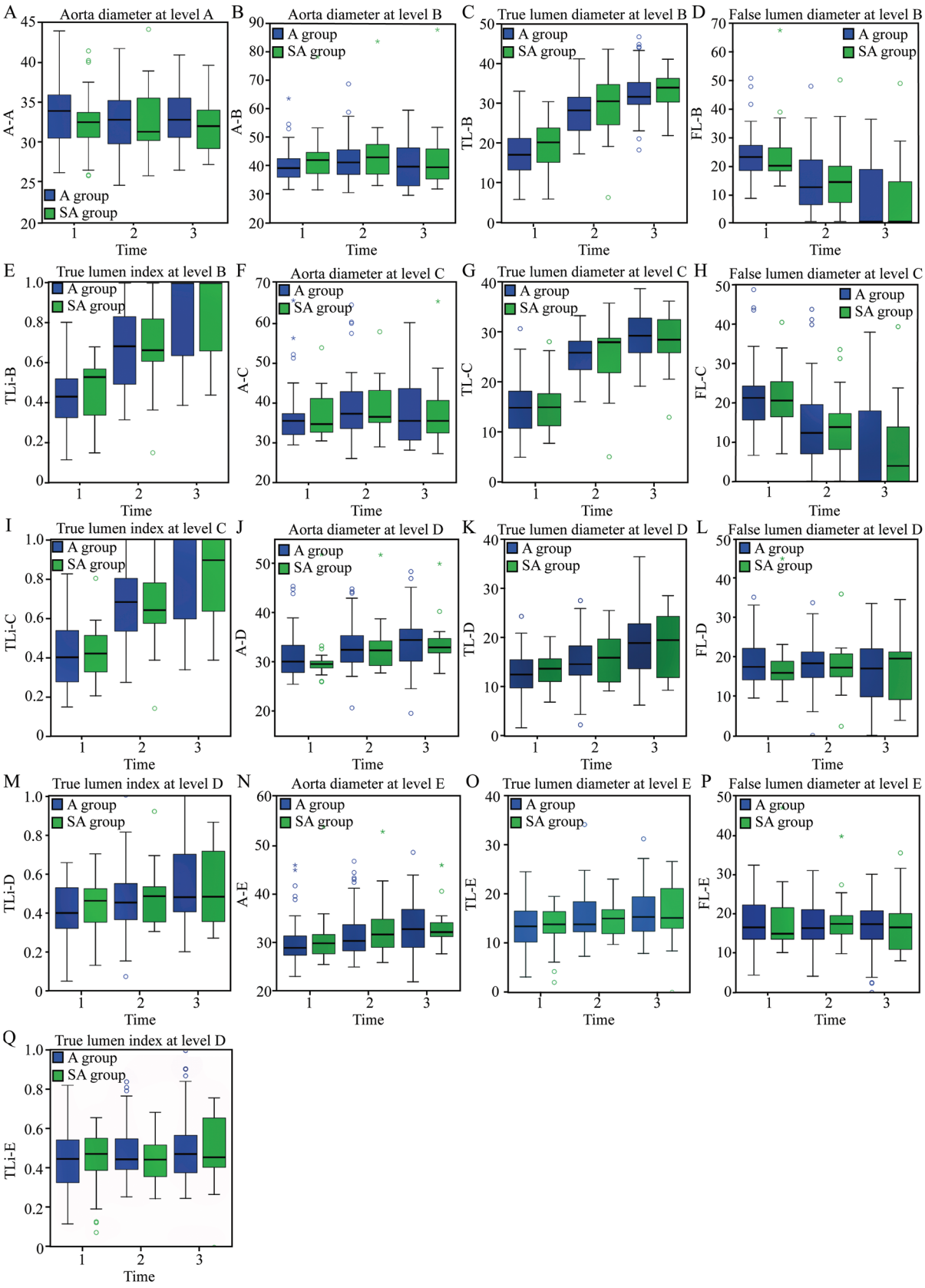

Fig. 2 The A (mm), TL (mm), FL (mm) and TLi of Group A and Group SA, trend at Level B–E along with different surveillant time (time 1, before TEVAR; time 2, 1 months after TEVAR; and time 3, 6 months after TEVAR) in Box-and-whisker Plot. Outliers and distribution interval were illustrated. A: aortic lumen; TL: true lumen; FL: false lumen; TLi: true lumen index; TEVAR: thoracic endovascular aortic repair

underwent BMT only.[15] It is accepted that subacute uncomplicated DeBakey type III AD could benefit from TEVAR than BMT only.

Several other studies compared TEVAR in subacute and acute phases. Desai et al.[16] whose group criterion was a little different from the 2014 guideline, performed 132 TEVAR for DeBakey type III dissection. In all, 70 TEVARs were performed within 48 h of presentation (acute-early), 44 between 48 h and 14 days from presentation (acute-delayed), and 18 between 14 days and 6 weeks from presentation (subacute). They concluded that TEVAR in the subacute phase, if possible, could lower the risk of complications; at the same time, the dissection was not often stable in the subacute phase, so close follow-up was needed. While severe complications were more common in patients in the acute phase than those in the subacute phase, the retrograde type A dissection tended to be more common in the acute-early group. According to the report of the VIRTUE Registry,[2] 3-year all-cause mortality, dissection-related mortality, aortic rupture, and retrograde type A dissection of the subacute group was lower than that of the acute group. While that in the acute phase is important, most patients have to be treated by TEVAR, or they are threatened by death due to serious complications. The heterogeneous cohort may influence the conclusion in an uncertain way.

Sample profile

The present study included 66 patients with DeBakey IIIb AD from January 2012 to October 2016. The clinical characteristics, radiologic findings, and aortic remodeling between the acute (<14 days) and subacute groups (≥14 days, <90 days) were compared to determine if there is any difference. Only patients with DeBakey IIIb AD were included in the study to ensure the homogeneity of the sample as far as possible. The patients were grouped by the interval from symptom onset to TEVAR. The difference in the clinical characteristics and aortic remodeling between the two groups were underpowered except for the interval from symptom onset to TEVAR.

Both acute and subacute DeBakey IIIb ADs have a similar extent of dissection, and acute and subacute TEVAR may stabilize the dissected aorta and prevent late complications by inducing aortic remodeling process equally. It is suggested that in DeBakey IIIb AD, if there are no factors indicating open surgery such as artery disease of the lower extremities and ilium, sharp angulation of the aortic arch, and absence of a healthy proximal landing zone, TEVAR may be the treatment of choice.[12] According to Desai's study,[16] TEVAR in the acute phase means more common perioperative complications, while that in the subacute phase means more safe procedures with possibly new TEVAR indications. We try to explore the difference in aortic remodeling between those in the acute and subacute phases and find if aortic remodeling could support the former opinion. Unlike Desai's study, in the present study, there was one retrograde type A dissection in group SA and no in-hospital mortality. This may be attributed to the limits of this study, which will be discussed later.

Variety of diameters

Diameters at different levels were changing over time. It was reported that the most significant change was in the first postoperative year.[17] Although almost all durations of follow-up in the present study are shorter than 1 year, the same trend as that in the outcome studies was observed.

First, A in both groups was stable at the proximal level (levels A–C) and increased at the distal level (levels D–E), which indicated a similar hemodynamic effect on the whole aorta.

Second, both groups showed a similar increase in TL. Positive TL remodeling was observed at levels B–D in both groups over time while TL greatly increased at T2 and T3 than at T1 at level E only in Group A. As a result, TEVAR in both groups had a much similar extent of positive TL remodeling, and TEVAR in Group A is superior at level E.

Third, FL in both groups decreased at the proximal level (levels B–C) over time, which indicated that entry tear has been closed immediately and hemodynamics have been changed accordingly in both groups. When it came to the distal part, FL remains stable, except for that in Group A it decreased after 6 months (T3 compared to T2 and T1).

Fourth, TLi increased at the proximal level (levels B–C) over time in both groups. At level D, TLi increased more slowly in Group SA than in Group A. At level E, TLi increased only in Group A.

The results of the morphologic change in the aorta in DeBakey type III AD were in accordance with those in previous studies, and some new characteristics were observed. For example, Czermak et al.[18] found that, within 12 months, mean TL volumes showed statistically significant increases in the proximal segments; FL volumes showed a significant decrease in the proximal segment; and no substantial volume changes were observed in the distal segment. Schoder et al.[19] found that, at all levels, the TL diameter increased significantly

after stent graft placement. At 1-year follow-up, the number of thrombosed FL decreased from proximal to distal segments. In the present study, we found that at proximal levels (levels A–C), the parameters on aortic remodeling in both groups showed a similar trend, that is, increased TL, decreased FL, and increased TLi; at distal levels (levels D–E), a more slow increase in TL, decrease in FL, and increase in TLi were observed in both groups; however, the change in Group SA was a unremarkable than that in Group A because the parameters in Group SA showed no statistical change or slower change than those in Group A. When it came to the parameter of A, the trend in both groups was similar: it was stable at the proximal level (levels A and B) and increased at the distal level (levels C–E) after TEVAR (T2 and T3 compared to T1), due to the complex hemodynamic effects.

It should be noted that FL in both groups showed slower or no significant decrease in distal segments. The reason may be that the restored TL had increased blood flow; at the same time, the abdominal aorta in DeBakey IIIb AD usually has one or more distal reentries, which link the TL and FL. Meanwhile, the FL often plays the part of a supply artery of the relative visceral artery; therefore, with no intervention, the FL had to remain in a certain balance. There were 51 patients (77.27%) in this study involving the visceral arteries, which were supplied by the FL. The following treatment is challenging, which should include not only sealing the distal reentries but also reconstructing the involved visceral arteries.

We found that in the box-and-whisker plot (**Fig. 2**), the more frequent outliers and the wider distribution interval of Group A compared with that in Group SA indicated a variety of complex complications. This is consistent with the VIRTUE Registry Investigators' literature reports that patients with subacute dissections after TEVAR had lower 3-year all-cause mortality compared to patients with acute and chronic dissections.[2] Furthermore, the abdominal aorta was unstable like the above analysis indicated, and at the same time, 6 months postoperation (T3) would not always be safe in the clinical practice. The more frequent outliers and the wider distribution interval in both groups at the distal level and in the 6-month postoperative follow-up (T3) warned us of possible sudden change in the abdominal aorta.

Limitations

First, in this study, the patients were followed up for only 6 months, which was not long enough. For one reason, most patients chose to be followed up in the local hospital after 6 months, so CT examination was performed in the local hospital and the imaging data could not be obtained; as the other reason, it is reported that the majority of the aortic remodeling was completed within 6 months of TEVAR in St. George's Vascular Institute.[8] Second, this cohort was included to reveal the difference in aortic remodeling between patients in the acute and subacute phases, so only those who had at least three CTA surveillances (pre-TEVAR, in 1 month, in 6 months) were included. As a result, some patients with aortic rupture or only two CTA surveillances (surveillance pre-TEVAR and in 1 month in our hospital and then maybe in 6 months in the local hospital) were excluded. Third, the number of patients is not large enough. Finally, the development of the technique and device in TEVAR may improve the effect of this treatment, which may also be a source of heterogeneity according to the outcome studies. Therefore, a multicenter prospective follow-up of recent patients is needed.

Conclusion

Proximal aortic remodeling was similar in Groups A and SA while TEVAR in the acute phase was more effective in distal aortic remodeling than that in the subacute phase. However, complications should be considered adequately, which were the factors in the acute phase, abdominal aorta, and 6 months after TEVAR. Patients with complications in either acute or subacute phase should undergo TEVAR if there is any indication. Patients with no complications are not suggested to leave the hospital with the dissection untreated by TEVAR or treated with BMT only because of the unpredictable complications that may develop. And TEVAR should be performed in the subacute phase if possible for patients with no complications. Postoperative surveillance is important for patients of both acute and subacute AD.

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Disclosure Statement

The authors declare no conflict of interest.

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