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Computed Tomography Scan of the Aorta to Predict Type B Aortic Dissection

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Abstract

Background The purpose of this study is to find the high-risk morphological features in type B aortic dissection (TBAD) population and to establish an early detection model.

Methods From June 2018 to February 2022, 234 patients came to our hospital because of chest pain. After examination and definite diagnosis, we excluded people with previous cardiovascular surgery history, connective tissue disease, aortic arch variation, valve malformation, and traumatic dissection. Finally, we included 49 patients in the TBAD group and 57 in the control group. The imaging data were retrospectively analyzed by Endosize (Therevna 3.1.40) software. The aortic morphological parameters mainly include diameter, length, direct distance, and tortuosity index. Multivariable logistic regression models were performed and systolic blood pressure (SBP), aortic diameter at the left common carotid artery (D3), and length of ascending aorta (L1) were chosen to build a model. The predictive capacity of the models was evaluated through the receiver operating characteristic (ROC) curve analysis.

Results The diameters in the ascending aorta and aortic arch are larger in the TBAD group (33.9 ± 5.9 vs. 37.8 ± 4.9 mm, $p < 0.001$; 28.2 ± 3.9 vs. 31.7 ± 3.0 mm, $p < 0.001$). The ascending aorta was significantly longer in the TBAD group (80.3 ± 11.7 vs. 92.3 ± 10.6 mm, $p < 0.001$). Besides, the direct distance and tortuosity index of the ascending aorta in the TBAD group increased significantly (69.8 ± 9.0 vs. 78.7 ± 8.8 mm, $p < 0.001$; 1.15 ± 0.05 vs. 1.17 ± 0.06, $p < 0.05$). Multivariable models demonstrated that SBP, aortic diameter at the left common carotid artery (D3), and length of ascending aorta (L1) were independent predictors of TBAD occurrence. Based on the ROC analysis, area under the ROC curve of the risk prediction models was 0.831.

Conclusion Morphological characteristic including diameter of total aorta, length of ascending aorta, direct distance of ascending aorta, and tortuosity index of ascending aorta are valuable geometric risk factors. Our model shows a good performance in predicting the incidence of TBAD.

Keywords
- aorta/aortic
- anatomy
- computed tomography
- CAT scan

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Introduction

Aortic dissection is the pathological process of aortic wall tearing under the impact of blood flow. It can affect all parts and organs of the whole body. Without appropriate treatment, it might cause an extremely high mortality and complications.[1] Concerning the different location of the primary entry tear, aorta dissection was divided into two types Stanford A and Stanford B. The incidence of type B aortic dissection (TBAD) is approximately 33% of overall aortic dissection patients. There is still a high mortality rate in the populations because of lacking appropriate and time-sensitive interventions.[2]

Therefore, early intervention aiming the high-risk populations becomes much more important.

Recently, several studies demonstrated that the aortic morphological features including diameter, length, and tortuosity are related to the incidence of type A aortic dissection.[3–7] However, rare studies focused on TBAD. We hypothesized that changes in aortic morphology also happened and increased the risk of TBAD. Thus, we designed this study to explore the morphological changes and build a model by comparing TBAD group and control group.

Materials and Methods

Study Design

The study was conducted as a retrospective, single-center, cross-sectional clinical study. Flow diagram of patients selection in the study is presented in ►**Fig. 1**. All the patients who were diagnosed with TBAD at our medical center from June 2018 to February 2022 were subjected to retrospective observational assessment. The inclusion criterion entailed patients who had undergone computed tomography angiography (CTA) at our institution because of chest pain and agreed to be included in our research. The exclusion criteria of the study were connective tissue disease (such as Marfan, Ehlers–Danlos, and Loeys–Dietz syndromes), arch branching variants, cardiac surgery history, bicuspid aortic valve, traumatic dissection, and clinical date losing. The control group consisted of subjects who received CTA with episodes of chest or back pain at our emergency department and were diagnosed with non-aortic disease. Finally, 128 patients met the exclusion criterion, and 106 patients were included in our study.

The study was approved by the institutional review board of our hospital (2020-185-01), and waived the need for individual patient consent.

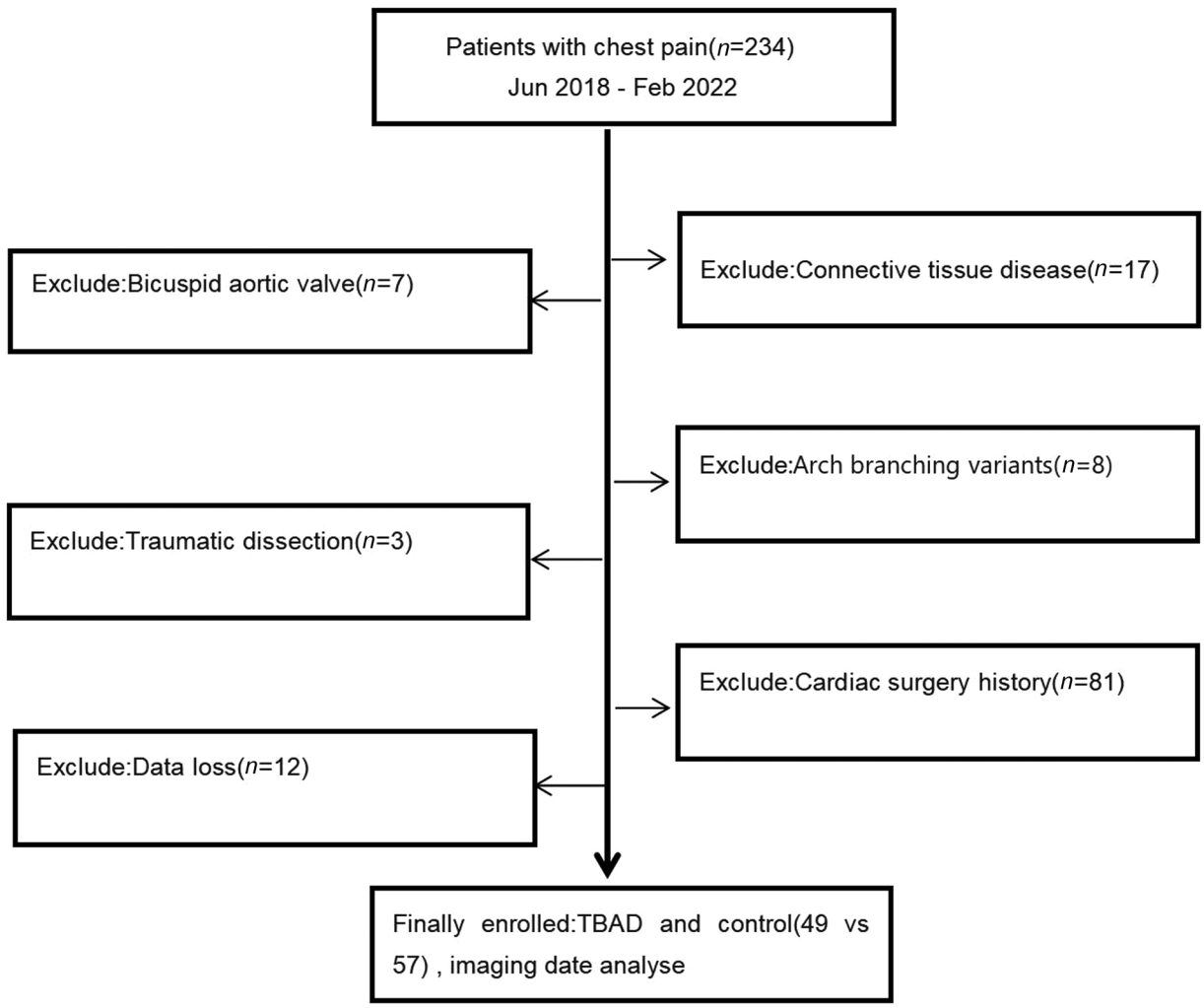

Fig. 1 Flow diagram of the patient selection. TBAD, type B aortic dissection.

Imaging Analysis

Among all the patients in the group, UIH 64-slice spiral CT (UCT 780, United Imaging) or 128-slice spiral CT (Brilliance iCT, Philips) were used for imaging scanning. The scanning mode was dynamic spiral scanning, and data were collected after deep inhalation and breath-holding. All patients were kept supine with advanced feet. Scanning parameters: tube voltage 120 KV, tube current 220 mas; rotate for 0.7 seconds; matrix 512×512; slice thickness 5 mm; and the interval between slices 5 mm. After plain scan, the first-stage enhanced scan is performed. Bronchial bifurcation level is selected as the monitoring level, and region of interest is placed in the middle of the descending aorta, with a threshold of 120 Hu. A high-pressure syringe is used to inject contrast agent Onaipike (350 mg/mL) intravenously at a rate of 1.5 mL/kg, with an injection rate of 4.0 mL/s. After the contrast agent injection, normal saline is routinely added. After scanning, three-dimensional reconstruction of blood vessel imaging results was carried out using ENDOSIZE (Therevna 3.1.40) (►**Fig. 2**). The center-line of blood vessel was extracted. The key points P1, P2, P3, P4, and P5 were marked on the center-line in the specific position. P1 represents the point of the sinus junction plane. P2 represents the point near the distal end of the innominate artery. P3 represents the distal end of the left common carotid artery. P4 represents the point on the distal end of the left subclavian artery. P5 represents the point 2 cm away from the left subclavian artery. This location division is based on the aortic division recommended in the guideline.[8] The morphological parameters were measured by using the measurement function owned by ENDOSIZE, including the following (1) D1, D2, D3, D4, and D5 representing the diameter of aorta at P1, P2, P3, P4, and P5. (2) L1 represents the length of the center-line between P1 and P2. L2 represents the length of the center-line between P2 and P3. L3 represents the length of the center-line between P3 and P4. L4 represents the length of the center-line between P4 and P5. L5 means that the length of the proximal aorta from P1 to P5 includes the ascending aorta and the aortic arch. (3) DD1 represents direct distance between two points P1 and P2. DD2 represents the direct distance between P2 and P3. DD3 represents the direct distance between P3 and P4.

DD4 represents the direct distance between P4 and P5. DD5 represents the direct distance betweenP1 and P5. (4) TI means tortuosity index calculated by dividing the length of the center-line between two points by the straight line distance between two points. It is an index reflecting the bending degree of each area. TI1 represents L1 divided by DD1. Similarly, TI2 represents L2 divided by DD2. TI3 represents L3 divided by DD3. TI4 represents L4 divided by DD4. TI5 represents L5 divided by DD5 (►**Fig. 2**).

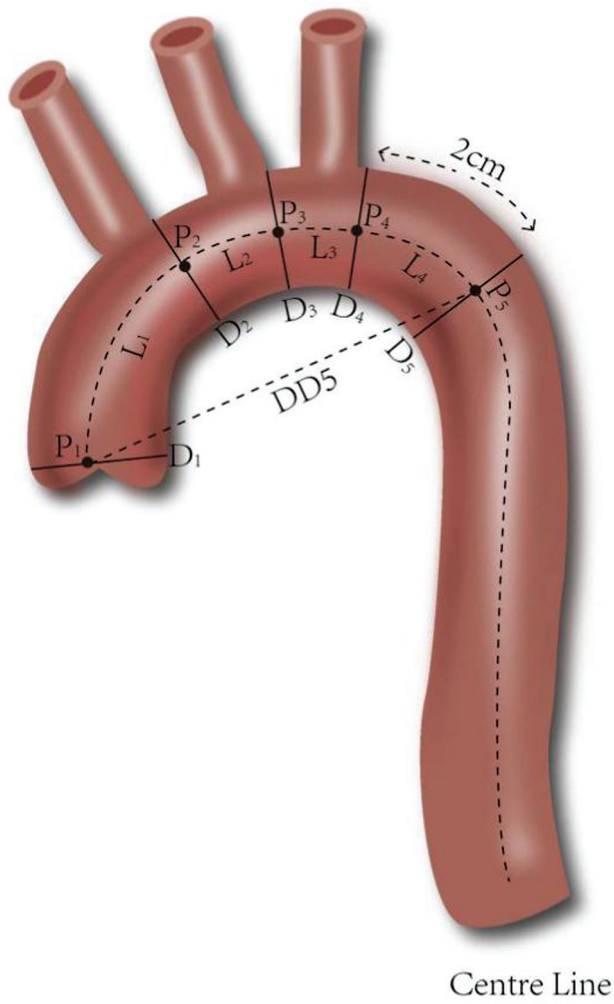

Fig. 2 Schematic of measurement; P1–5: Point 1–5; D1–5: Diameter 1–5; L1–5: Length1–5; DD5: Direct distance from point 1 to point 5.

Statistical Methods

Continuous data were reported as mean ± standard deviation; categorical data were expressed as numbers and percentages. Shapiro–Wilk test was used to assess the normality of the data. Student's *t*-test and Mann–Whitney test were applied to compare the anatomical variables between the TBAD and control groups. The chi-square test and Fisher's exact test were used for comparing the categorical variables between two groups. Multivariable logistic regression models including all baseline characteristics and the significant variables in univariate analysis were used to investigate the independent predictors of TBAD. The risks were expressed as odds ratios with 95% confidence intervals. The receiver operating characteristic (ROC) curve and the area under the ROC curve (AUC) were performed to assess the predictive performance of the risk model. Statistical analysis was performed with SPSS software (version 28.0.1, IBM, Armonk, New York, United States). All *p*-values tested were two-sided.

Results

Baseline Characteristics

The baseline characteristics between the TBAD and control group are summarized in ►**Table 1**. The TBAD group had more males than control group (89.8% vs. 50.9%, $p < 0.001$). Patients in the TBAD group were younger and had a higher body mass index and blood pressure than those in the control group (►**Table 1**).

Table 1 Baseline characteristics in study group and control group

	Controls ($n = 57$)	TBAD ($n = 49$)	p-Value
Male (%)	29 (50.9)	53 (89.8)	$< 0.001^{**}$
Age (y)	56.5 ± 16.8	53.9 ± 9.5	0.325
BMI (kg/m^2)	24.0 ± 4.6	26.8 ± 4.5	0.002^{*}
SBP (mm Hg)	130.4 ± 20.4	144.8 ± 27.0	0.002^{*}
DBP (mm Hg)	76.4 ± 12.8	82.0 ± 14.3	0.036^{*}
WBC (10^9/L)	7.2 ± 2.8	10.3 ± 3.4	$< 0.001^{**}$
RBC (10^12/L)	4.2 ± 0.6	4.3 ± 0.6	0.259
PLT (10^9/L)	208.9 ± 111.5	186.0 ± 63.9	0.207
TG (mmol/L)	1.2 ± 0.7	1.5 ± 1.6	0.242
TC (mmol/L)	3.9 ± 1.0	3.7 ± 0.9	0.154
ALT (U/L)	25.0 ± 20.2	28.7 ± 31.3	0.463
AST (U/L)	28.3 ± 30.8	23.3 ± 20.6	0.332
T-bile (umol/L)	12.9 ± 10.7	11.6 ± 4.7	0.424
Albumin (g/L)	38.5 ± 4.5	38.0 ± 4.0	0.592
eGFR	112.9 ± 38.3	102.9 ± 34.7	0.165
BUN (mmol/L)	11.1 ± 36.4	7.2 ± 5.2	0.459
CR (umol/L)	76.2 ± 87.6	92.1 ± 88.8	0.355

Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; BMI, body mass index; BUN, blood urea nitrogen; CR, creatinine; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; PLT, platelet; RBC, red blood cell; SBP, systolic blood pressure; TBAD, type B aortic dissection; T-bile, total bile acid; TC, total cholesterol; TG, triglyceride; WBC, white blood cell.
Note: Data are n (%) or mean ± standard deviation (SD).
$^{*}p < 0.05$.
$^{**}p < 0.01$.

More TBAD group patients had higher white blood cell. There are no significant difference in red blood cell, platelet, liver and renal function, and state of nutrition.

Morphological Dates

In comparison with the control group, the diameters of the D1–D5 were significantly larger in the TBAD group ($p < 0.001$) (►**Table 2** and ►**Fig. 3A**). The length of L1 and L5 are much longer in the TBAD group ($p < 0.001$), whereas L2, L3, and L4 show no differences (►**Table 2** and ►**Fig. 3B**). Direct distance between points on center-line DD1and DD5 are longer than controls ($p < 0.001$), and the tortuosity index in TI1 is also different ($p = 0.022$). The rest of date DD2–4 (direct distance 2-4) and TI2–5 had no statistical difference (►**Table 2** and ►**Fig. 3C, D**).

Risk Prediction Models for TBAD

According to the results of multivariable regression, we choose L1, systolic blood pressure (SBP), and D3 to build a predictive model. By using Nomogram or Alignment Diagram (►**Fig. 4**), we can get the probability of occurring TBAD in high-risk populations. The AUC is 0.831, showing a better performance verses L1, SBP, and D3, respectively (0.784, 0.655, and 0.776) (►**Fig. 5**).

Discussion

Aortic dissection is a catastrophic vascular disease which has high mortality and morbidity.[1,2] While in our country the prevalence of TBAD is higher than type A aortic dissection,[9] early intervention of high-risk patients through the analysis of aortic morphology has major clinical implications in reducing the mortality associated with TBAD. Our main findings in this study are summarized as follows (1) the dilation of ascending aorta and aorta arch was significant in the TBAD group. (2) The length of the ascending aorta (L1) and total proximal aorta including ascending aorta and aorta arch are longer. (3) The tortuosity index of the ascending aorta was larger.

As stated in the clinical guideline[8] of aortic dissection, hypertension is an important risk factor for aortic dissection. Moreover, the increase of blood pressure is closely related to the change of stress received by aorta. After the contraction of arterioles, the stress of aorta increases and the left ventricular afterload increases. Robicsek and Thubrikar's research[10] on aortic mechanics is a fascinating starting point to understand the relationship between hypertension and aortic dissection, but the research in this field is still not deep enough. In our sample, the SBP and diastolic blood pressure of TBAD are 144.8 ± 27.0 and 82.0 ± 14.3, respectively, which is significantly different from that of normal people at 130.4 ± 20.4 and 76.4 ± 12.8 ($p < 0.001$). During the treatment of these patients in our center, we found that they did not regularly supervise their blood pressure, and even did not take antihypertensive measures. Therefore, improving the monitoring and treatment of hypertension is also a key to prevent this disease. Based on this obvious difference, the randomly measured blood pressure is included in our prediction model.

Table 2 Morphological date of controls and TBAD groups

	Controls ($n = 57$)	TBAD ($n = 49$)	t/chi-square	p-Value
D1 (mm)	33.9 ± 5.9	37.8 ± 4.9	−3.667	< 0.001**
D2 (mm)	30.5 ± 4.1	34.0 ± 3.2	−4.814	< 0.001**
D3 (mm)	28.2 ± 3.9	31.7 ± 3.0	−5.106	< 0.001**
D4 (mm)	26.0 ± 3.5	30.7 ± 4.7	−5.879	< 0.001**
D5 (mm)	26.0 ± 3.6	37.3 ± 8.4	−9.217	< 0.001**
L1 (mm)	80.3 ± 11.7	92.3 ± 10.6	−5.516	< 0.001**
L2 (mm)	9.4 ± 3.1	10.3 ± 2.8	−1.580	0.117
L3 (mm)	14.8 ± 4.3	14.7 ± 3.8	0.124	0.901
L4 (mm)	20.0 ± 0.0	20.0 ± 0.0		
L5 (mm)	124.5 ± 13.2	137.4 ± 12.4	−5.148	< 0.001**
DD1 (mm)	69.8 ± 9.0	78.7 ± 8.8	−5.143	< 0.001**
DD2 (mm)	9.3 ± 3.0	10.3 ± 3.0	−1.728	0.087
DD3 (mm)	14.2 ± 4.5	14.0 ± 4.3	0.040	0.968
DD4 (mm)	18.5 ± 2.1	19.0 ± 1.7	−1.130	0.261
DD5 (mm)	90.0 ± 12.2	98.4 ± 10.0	−3.836	< 0.001**
TI1	1.15 ± 0.05	1.17 ± 0.06	−2.320	0.022*
TI2	1.02 ± 0.13	1.00 ± 0.07	0.429	0.669
TI3	1.06 ± 0.20	1.04 ± 0.09	0.556	0.579
TI4	1.13 ± 0.45	1.07 ± 0.07	0.872	0.385
TI5	1.39 ± 0.10	1.40 ± 0.09	−0.569	0.570

Abbreviation: TBAD, type B aortic dissection.
Note: D1–5: diameter 1–5. L1–5: length 1–5. DD1–5: direct distance 1–5. TI1–5: tortuosity index 1–5.
*$p < 0.05$.
**$p < 0.01$.

Early intervention is an important part of the current treatment. Diameter is a generally accepted intervention standard at present. According to a series of studies,[11–13] when the diameter of aorta is larger than 5.5 cm, the risk of dissection will suddenly increase. Therefore, 5.5 cm is the accepted intervention standard at this stage. When the patient is accompanied by connective tissue disease and symptoms of compression, this standard will be more strictly restricted to 5.0 cm. These series of studies were mostly conducted decades ago. The latest results published by the International Registry of Acute Aortic Dissection[14] show that 60% of people with dissection have a diameter of less than 5.5 cm, and nearly 40% of patients have an aortic diameter of less than 5.0 cm. As for why the aorta dilates, there are many theories trying to explain this phenomenon scientifically. According to O'Rourke and Hashimoto's research,[15] the aorta will become "material fatigue" with age, and its diameter will expand. Koullias et al[16] started from the basic histology and biochemistry, the diameter expansion is also related to the destruction of elastic fibers, the difference of extracellular matrix protein distribution, and the increase of metalloproteinase expression. However, Coady et al[17] showed that the aortic wall suddenly weakened after aortic dissection, and the aortic dissection caused the diameter to expand sharply, that is to say, the diameter would expand in the area involved by aortic dissection. However, the existence of this expansion before the onset of the disease is still controversial. In our results, D1 to D5 are significantly expanded in TBAD, and their diameters are 37.8 ± 4.9, 34.0 ± 3.2, 31.7 ± 3.0, 30.7 ± 4.7, and 37.3 ± 8.4 cm, respectively. In the control group, the diameters of D1 to D5 were 33.9 ± 5.9, 30.5 ± 4.1, 28.2 ± 3.9, 26.0 ± 3.5, and 26.0 ± 3.6 cm, respectively. There was significant statistical difference between the two groups ($p < 0.001$). However, in the control group, the diameter from D1 to D5 gradually decreased, while in the TBAD group D5 increased significantly. We have sufficient evidence to believe that it is due to the occurrence of dissection.

Therefore, when we choose the diameter to be included in the prediction model, we should not only try to avoid the interference of the dissection itself, but also fully explain the aortic dilatation that existed before the onset of the disease. That is why we chose D3. In our study samples, D3 is not involved by dissection, and D3 is close to the site of the dissection, so D3 is a suitable index that we need.

Since Trimarchi et al[18] questioned it, more and more evidences[14,19] show that it is inappropriate to use diameter alone. Many scholars began to try to find new intervention indicators. Heuts et al and Krüger et al[7,20] have established the prediction model of type A dissection among people with type A dissection. However, as far as we know, there are few researches on TBAD. Consistent with the research results of Shirali et al, Lescan et al, and Sun

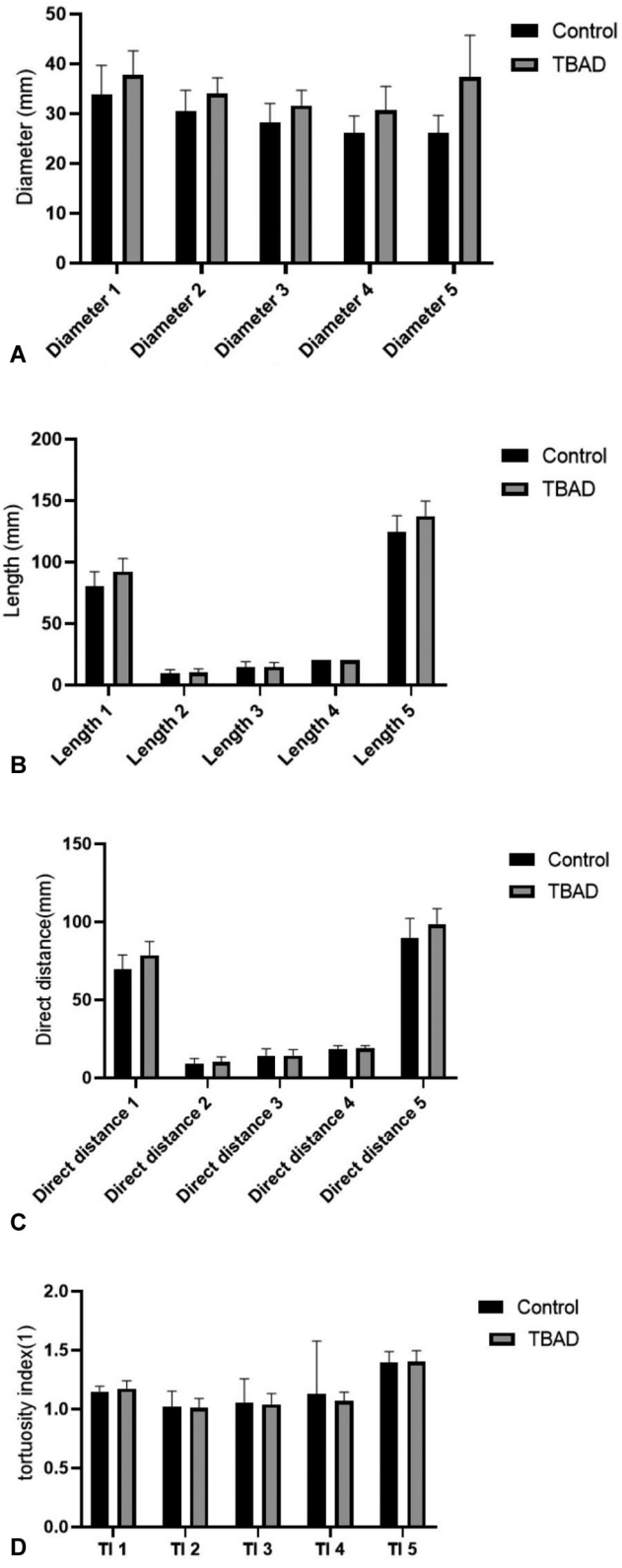

Fig. 3 (A) Comparison of diameter at different location. (B) Comparison of the length in different section. (C) Comparison of the direct distance in different section. (D) Comparison of the tortuosity index (TI) in different section.

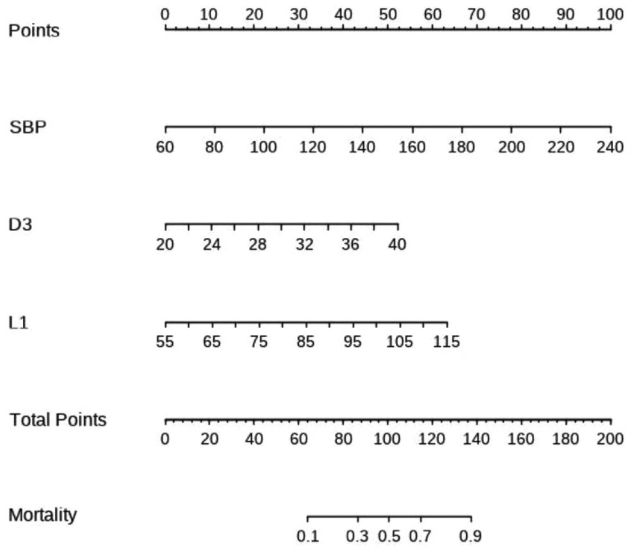

Fig. 4 Systolic blood pressure (SBP); D3, diameter 3; and L1, length 1.

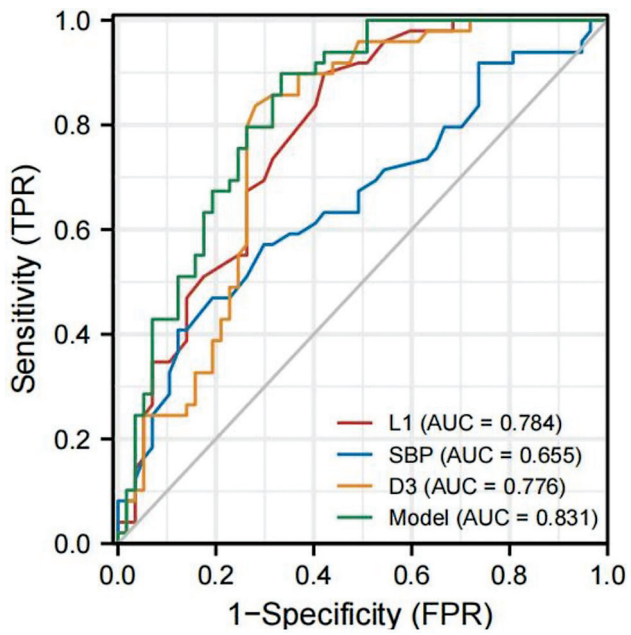

Fig. 5 Receiver operating characteristic (ROC) curve.

et al,[21–23] aortic elongation does exist. Shirali et al's results show that the ascending aorta is obviously prolonged in people with dissection, and they advocate it as a new intervening factor, and advocate more scientists to conduct prospective research. Lescan et al reported a significant increase in aortic arch. Our research results are consistent with Sun et al. After excluding connective tissue patients, we found that the ascending aorta L1 and the whole proximal aorta L5 were lengthened. The length of L1 in the TBAD group was 92.3 ± 10.6 mm, while that in the control group was 80.3 ± 11.7 mm, with significant statistical difference ($p < 0.001$). The average length of L5 in the sandwich population is 137.4 ± 12.4 mm, which is significantly different from that of the normal population (124.5 ± 13.2 mm, $p < 0.001$), and the overall tortuosity index (TI1) is significantly increased (1.17 ± 0.06, $p = 0.022$) (►**Table 2**). The phenomenon of aortic lengthening has been clarified in some studies

and is related to factors, such as age and hypertension. The research of Adriaans et al[24] shows that in the western population, the proximal ascending aorta stretches nearly 2.5 times between the ages of 20 and 80. Moreover, this lengthening mainly occurred in the proximal aorta, and the distal length of the descending aorta changed little. It is mainly because the distal descending aorta has intercostal artery, which fixes it on the side of the spine, resulting in its small range of motion.[25] Sokolis et al[26,27] showed that the increase of shear stress in aortic wall led to the rupture of elastic fibers, which were gradually replaced by extracellular inelastic fibers, and the arch apex gradually shifted from between branches to behind branches.[24] In the future research, the hemodynamic changes caused by morphological changes will enable us to better understand the stress of aorta from the perspective of fluid dynamics, and may help us to improve our understanding.

Limitations

First, this study is a single-center retrospective study. The research results are only applicable to patient in our hospital. Second, the sample size of this study is small, and there is no external verification, so it is difficult to popularize the research results. Third, with regard to the ethnic variation, future studies on different ethnics are necessary to validate the results. Last, CTA before the dissection is difficult to obtain, since patients with TBAD usually do not undergo CTA examination before the onset of the disease. This may affect the results of the study.

Conclusion

According to the results of multivariable logistic regression, we select the length (L1), diameter (D3), and hypertension comprehensively to build a predicting model. A nomogram is shown in ►**Fig. 4** to predict the occurrence of TBAD. The ROC curve as shown in ►**Fig. 5** shows that the AUC of the comprehensive prediction model is 0.831, which is better than using L1, D3, and hypertension alone (0.784, 0.776, and 0.655).

Authors' Contribution

Conception and design of the work, corresponding author: Z.Q.; data collection: L.K., T.T., J.M.; statistical analysis: Z.H.; analysis and interpretation of the data: T.X.; drafting the manuscript: L.H.; critical revision of the manuscript: L.H.

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Conflict of Interest

None declared.

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