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Research Article

Predicting Preoperative Rupture from Imaging Alone in Acute Type A Aortic Dissection

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Objective. To establish risk factors for predicting preoperative ruptures in patients with acute type A aortic dissection (ATAAD) based on computed tomography angiography (CTA) imaging features alone. *Methods*. We retrospectively reviewed patients with ATAAD treated between January 2017 and December 2021 in Fujian Medical University Union Hospital, China. The primary outcome was preoperative rupture after admission. Multivariate logistic regression analysis was performed based on basic characteristics and CTA imaging variables selected by the application of the least absolute shrinkage and selection operator. *Results*. A total of 564 patients were enrolled. The rate of preoperative rupture was 14.2% ($n = 80$). Patients who experienced rupture were significantly older ($P = 0.002$) and had a higher rate of DeBakey II ($P = 0.016$), syncope ($P = 0.003$), ventilator-assisted ventilation ($P = 0.008$), preoperative shock ($P = 0.040$), hypotensive state ($P = 0.009$), hepatic insufficiency ($P = 0.002$), acute kidney injury ($P = 0.045$), and moderate or massive pericardial effusion ($P = 0.007$). Multivariate analysis identified the following independent risk factors for preoperative rupture based on CTA imaging features: DeBakey II (odds ratio (OR) = 1.988, 95% confidence interval (CI) 1.211–3.676, $P = 0.009$), ascending aorta diameter (OR = 2.077, 95% CI 1.335–4.045, $P < 0.001$), ascending aorta false lumen diameter (OR = 2.988, 95% CI 2.055–4.291, $P < 0.001$), ascending aorta false lumen/true lumen diameter ratio >4 : 1 (OR = 3.129, 95% CI 2.031–6.225, $P < 0.001$), and number of branch arteries involved in dissection >6 (OR = 1.154, 95% CI 1.036–2.006, $P = 0.036$). *Conclusions*. CTA imaging features are one of the most convenient indicators for the early prediction of preoperative rupture in patients with ATAAD.

1. Introduction

Acute type A aortic dissection (ATAAD) is a lethal and emergency condition [1]. Timely surgery is effective for most patients. Many patients succumb without an opportunity to receive timely surgical treatment due to acute heart failure, acute myocardial infarction, cerebral infarction, and acute liver and kidney insufficiency; however, preoperative dissection rupture is still one of the main reasons for death [2].

In China, most prefecture-level cities do not have hospitals that are adequately equipped to perform surgery for patients with ATAAD. Therefore, it is necessary to transfer such patients to a centralized hospital that has surgical capabilities. Although there is a risk involved in transporting such patients, this approach is effective in improving the success rate of treatment [3, 4]. Therefore, centralized hospitals often receive several patients with ATAAD at the same time [5].

To address this situation, we performed a risk stratification study for patients with ATAAD to efficiently identify who is more critical and to prioritize their treatment. Some studies have reported the postoperative mortality and preoperative rupture prediction of patients with ATAAD; however, few studies have evaluated the risk factors for preoperative rupture using computed tomography angiography (CTA) imaging alone in these patients. Thus, the current study aims to identify predictors for in-hospital preoperative rupture in ATAAD patients, especially based on imaging features, to aid physicians in optimal arrangement and management.

2. Patients and Methods

This retrospective, single-center research was approved by Fujian Medical University Union Hospital, China (approval number: 2016KY008), and conducted according to the Declaration of Helsinki, with waived informed consent based on the nature of the retrospective study.

2.1. Patients. Patients with ATAAD treated at our center between January 2017 and December 2021 were enrolled. The exclusion criteria were as follows: (1) patients with difficulty distinguishing the true and false lumen, (2) traumatic aortic dissection, and (3) a history of ascending aortic surgery.

2.2. Endpoints. The primary endpoint was preoperative dissection rupture after admission.

2.3. Patient Groups. Patients were divided into a preoperative rupture group or surgical group based on whether or not the dissection ruptured before surgery. Patients who experienced rupture during transport to the operating room or during anesthesia were placed in the preoperative rupture group. The criteria for preoperative rupture were as follows: patients with sudden cardiac arrest, disappearance of blood pressure, and electromechanical separation accompanied by a large amount of new pericardial effusion. The detailed flowchart is shown in Figure 1.

2.4. Data Collection. Demographic characteristics, biochemical indicators, and echocardiography data were recorded at the time of admission and were extracted from the electronic medical records.

2.5. Computed Tomography Angiography (CTA). All CTA data were scanned using a Revolution CT with a scanning thickness of 0.625 mm at our center. Imaging-qualified DICOM data were passed to the center's imaging two experienced senior radiologists for joint interpretation and analysis. The total plane diameter and false lumen diameter of the aortic sinus, the mid-section of the ascending aorta, and the proximal aortic arch were measured. (1) Aortic sinus diameter: the maximum diameter of the parallel sinus section; (2) ascending aortic diameter: the plane of the ascending aorta at the midpoint distance from the sinotubular junction to the proximal end of the arch; (3) proximal aortic arch: the diameter of the proximal plane of the innominate artery (Figure 2).

2.6. Relevant Definitions. Relevant definitions are given as follows:

Shock state: systolic blood pressure was measured to be ≤ 80 mm Hg

Hypotensive state: systolic blood pressure was measured to be <90 mm·Hg but >80 mm·Hg

Renal insufficiency: serum creatinine (Scr) > twice the normal value

Hepatic insufficiency: alanine aminotransferase >4 times the normal value

2.7. Statistical Analysis. Data distribution was determined by Shapiro–Wilk tests. Categorical and continuous variables are presented as n (%) and mean ± standard deviation or median (interquartile range). Categorical data analyses were performed using chi-square or Fisher exact test. Non-normally distributed variables were analyzed using the Mann–Whitney U test, whereas normally distributed variables were analyzed using Student's t-test.

Predictors of preoperative rupture selected based on least absolute shrinkage and selection operator (LASSO) regression analysis were included in the multivariate logistic regression analysis. This approach could avoid issues of multicollinearity and overfitting due to a high number of potential predictors or a small sample size. A graph of statistical OR variable importance was constructed based on the multivariate logistic regression analysis. The OR variable importance was determined based on the percent of model deviance explained in the model. Data analyses were performed using SPSS 26.0 and R version 4.1.3. $P < 0.05$ was considered statistically significant.

3. Results

3.1. Baseline Data. A total of 582 patients with acute Stanford type A aortic dissection (ATAAD) were admitted; 8 patients with missing clinical data, 7 with a history of previous aortic surgery, and 3 with unclear images of the true and false lumens of ascending aortic dissection were excluded from this study. Finally, 564 patients were included in the analysis, of whom 80 (14.2%) died of preoperative rupture and 484 (85.8%) did not experience preoperative rupture. Given the lethal nature of ruptured ATAAD, even after a series of rescue measures, none of these ruptured patients survived and they had no opportunity to be operated on. Patients who experienced preoperative rupture had a larger ascending aorta diameter ($P = 0.001$), ascending aorta false lumen diameter ($P < 0.001$), ascending aorta false lumen/true lumen diameter ratio ($P < 0.001$), a higher rate of ascending aorta false lumen/true lumen diameter ratio >4 : 1 ($P < 0.001$), and the number of branch arteries involved in dissection >6 ($P < 0.001$) (Table 1).

3.2. LASSO Regression Analysis. Potential risk variables of preoperative rupture selected based on LASSO regression analysis are presented in Figure 3.

A total of 36 variables collected from preoperative data were used in LASSO regression analyses, and 11 predictors including age, DeBakey II type, syncope, preoperative shock, hypotensive state, preoperative hepatic insufficiency, moderate or massive pericardial effusion, ascending aorta diameter, ascending aorta false lumen diameter, ascending aorta false lumen/true lumen diameter ratio >4 : 1, and the number of branch arteries involved in dissection >6 were selected for a multivariate logistic regression analysis to predict the independent risk of preoperative rupture.

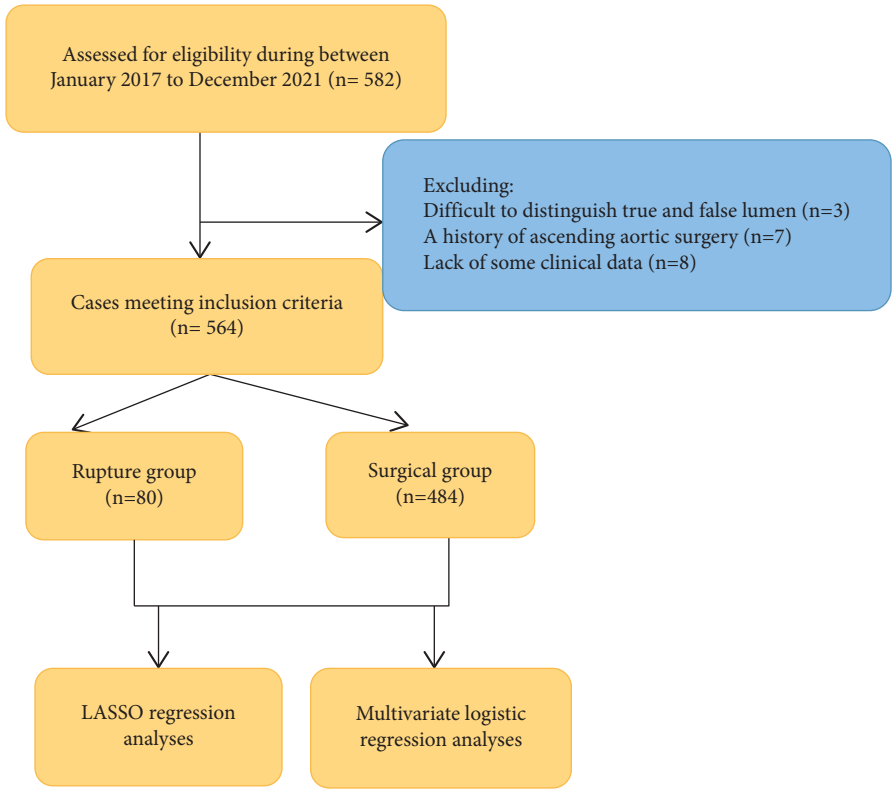

Figure 1: A detailed flowchart of patient selection.

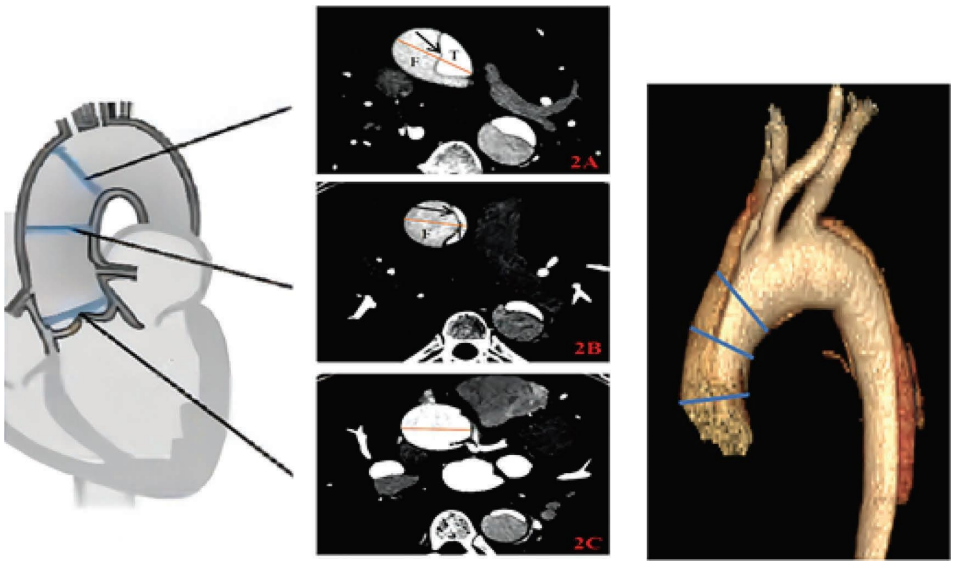

Figure 2: Schematic diagram of the measurement of 2A the proximal aortic arch; 2B the middle part of the ascending aorta; 2C the aortic sinus (black arrows refer to intimal patch; T refers to true lumen; F refers to false lumen).

3.3. Multivariate Logistic Regression Analysis. In the multivariate analysis, DeBakey II type (OR = 1.988, 95% CI 1.211–3.676, $P = 0.009$), ascending aorta diameter (OR = 2.077, 95% CI 1.335–4.045, $P < 0.001$), ascending aorta false lumen diameter (OR = 2.988, 95% CI 2.055–4.291, $P < 0.001$), ascending aorta false lumen/true lumen diameter ratio >4 : 1 (OR = 3.129, 95% CI 2.031–6.225, $P < 0.001$), and the number of branch arteries involved in dissection >6 (OR = 1.154, 95% CI 1.036–2.006, $P = 0.036$) were considered independent risk factors for preoperative rupture. The variable importance was drawn based on the results from multivariate logistic regression. Figure 4 shows the variable importance of each independent risk factor.

Table 1: The clinical characteristics and computed tomography angiography (CTA) features of two groups.

Variables	Rupture group ($n = 80$)	Surgical group ($n = 484$)	t/χ2/Z	P
Demographics				
Age (years)	53.8 ± 11.4	49.6 ± 9.7	3.114	0.002
Male	60 (75.0)	372 (76.9)	0.132	0.716
BMI (kg/m^2)	25.26 ± 3.65	26.03 ± 4.04	−1.600	0.110
DeBakey typing			5.808	0.016
DeBakey II type	11 (13.8)	30 (6.2)		
DeBakey I type	69 (86.3)	454 (93.8)		
Underlying conditions				
CAD	6 (7.5)	35 (7.2)	0.007	0.932
History of hypertension	60 (75.0)	351 (72.5)	0.213	0.644
History of diabetes	8 (10.0)	41 (8.5)	0.202	0.653
History of hyperlipidemia	8 (10.0)	34 (7.0)	0.882	0.348
MFS	6 (7.5)	29 (6.0)	—	0.616
Time range[#], median (Q1; Q3)				
Time 1 (hours)	6 (5; 11)	6 (5; 10)	0.672	0.836
Time 2 (hours)	4 (3; 7)	5 (4; 7)	−1.056	0.608
Time 3 (hours)	9 (7; 12)	9 (8; 13)	−1.635	0.590
Syncope	8 (10.0)	12 (2.5)	—	0.003
Systolic blood pressure (mmHg)	120.1 ± 29.5	122.4 ± 23.6	−0.663	0.509
Mean heart rate (bpm)	98 ± 33	78 ± 40	4.862	<0.001
Ventilator-assisted ventilation	16 (20.0)	48 (9.9)	6.938	0.008
LVEF (%), median (Q1; Q3)	55 (50; 65)	59 (52; 64)	−2.985	0.412
Preoperative shock	6 (7.8)	13 (2.7)	—	0.040
Hypotensive state	8 (10.0)	15 (3.1)	—	0.009
Preoperative hepatic insufficiency	8 (10.0)	10 (2.1)	—	0.002
Preoperative renal insufficiency	8 (10.0)	20 (4.1)	—	0.045
Moderate or severe AR	16 (20.0)	81 (16.7)	0.514	0.474
Moderate or massive pericardial effusion	10 (12.5)	21 (4.3)	—	0.007
Comparison of imaging features				
Aortic sinus diameter (mm)	39.60 ± 7.33	40.66 ± 8.88	−1.160	0.248
Ascending aorta diameter (mm)	50.14 ± 4.68	48.11 ± 6.08	3.431	0.001
Ascending aorta false lumen diameter (mm)	35.09 ± 9.96	25.99 ± 4.34	8.047	<0.001
Ascending aorta false lumen/true lumen diameter ratio (continuous)	3.55 ± 2.05	2.66 ± 2.06	3.582	<0.001
Ascending aorta false lumen/true lumen diameter ratio (categorical)			48.775	<0.001
≧4 : 1*	29[a] (36.3)	54[b] (11.2)		
≧3 : 1	10[a] (12.5)	36[a] (7.4)		
≧2 : 1	16[a] (20.0)	74[a] (15.3)		
≧1 : 1	17[a] (21.3)	145[a] (30.0)		
<1 : 1*	8[a] (10.0)	175[b] (36.2)		
Proximal aortic arch diameter (mm)	36.33 ± 10.40	35.04 ± 10.98	0.981	0.327
Proximal aortic arch false lumen (mm)	20.26 ± 8.98	18.98 ± 9.89	0.238	0.812
Dissection involving the coronary arteries	10 (12.5)	48 (9.9)	0.496	0.481
Dissection involving the brachiocephalic trunk	58 (72.5)	301 (62.2)	3.154	0.076
Dissection involving celiac visceral arteries	48 (60.0)	254 (52.5)	1.561	0.212

Table 1: Continued.

Variables	Rupture group ($n = 80$)	Surgical group ($n = 484$)	t/χ2/Z	P
Dissection involving the renal artery	56 (70.0)	288 (59.5)	3.179	0.075
Dissection involving lower extremity arteries	43 (53.8)	223 (46.1)	1.263	0.203
Number of branch arteries involved in dissection > 6	21 (26.3)	50 (10.3)	15.811	<0.001
Patent thrombosed in the false lumen	48 (60.0)	251 (51.9)	1.826	0.177
Aortic dissection intima sheet without distortion	56 (70.0)	314 (64.9)	0.799	0.371

Data are presented as mean ± SD or median (interquartile range), and categorical variables are presented as number (%). *The proportion of patients was significantly different between the two groups by Bonferroni method. #Time 1: time from onset to admission; Time 2: time from admission to surgery or rupture; Time 3: time from the onset to surgery/rupture. Q1, first quartile; Q3, third quartile; BMI, body mass index; CAD, coronary artery disease; MFS, Marfan syndrome; LVEF, left ventricular ejection fraction; AR, aortic regurgitation.

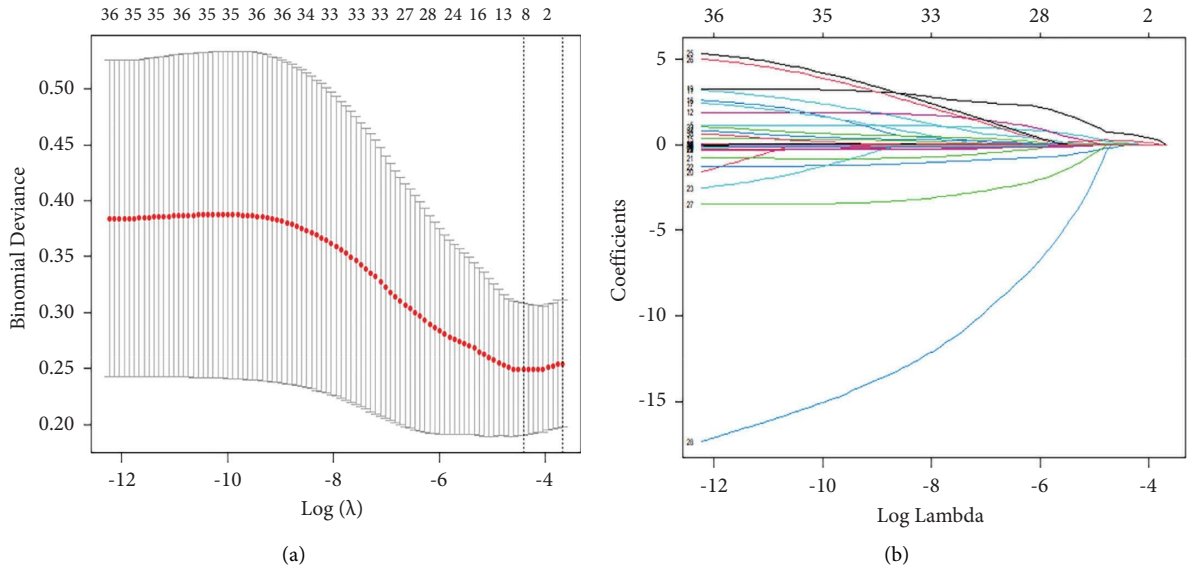

Figure 3: Potential risk variables of preoperative rupture selected based on least absolute shrinkage and selection operator (LASSO) regression analysis.

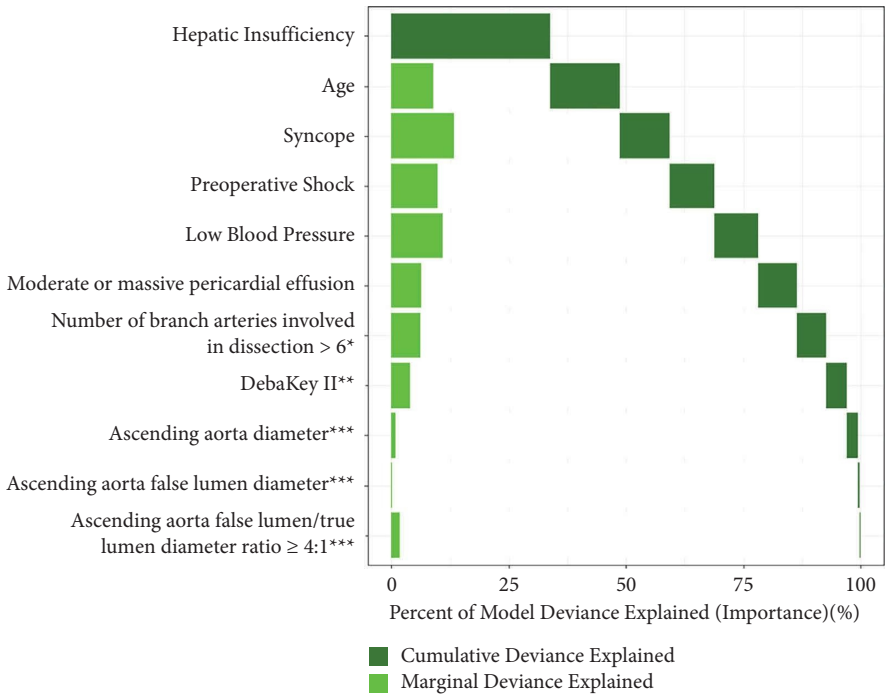

Figure 4: The variable importance plots based on the results from multivariate logistic regression (*: $P < 0.05$, **: $P < 0.01$, ***: $P < 0.001$).

3.4. Characteristics of the Ascending Aorta Diameter. The average diameter of the ascending aorta was 49.70 ± 4.74 mm, and the difference was significant between the rupture and surgical groups ((50.14 ± 3.68) mm vs. (48.11 ± 6.08) mm, $P = 0.001$). The average diameter of the false lumen of the ascending aorta was 29.32 ± 6.95 mm, and there was also a significant difference between the rupture and surgical groups ((35.09 ± 9.96) mm vs. (25.99 ± 4.34) mm, $P < 0.001$). However, there were no significant differences with respect to the diameter of the aortic sinus, the diameter of the proximal end of the arch, and the diameter of the false lumen at the proximal end of the arch (all $P > 0.05$). The average false lumen/true lumen ratio of the ascending aorta was 2.96 ± 2.03, and the differences between the rupture and surgical groups were significant (3.55 ± 2.05 vs. 2.66 ± 2.06, $P < 0.001$). The proportion of false lumen/true lumen ratio greater than 4 : 1 between the rupture group and the surgical group was statistically significantly different (36.3% (29/80) vs. 11.2% (54/484), adjusted $P < 0.001$, adjusted by Bonferroni method) (Table 1).

3.5. Threshold Analysis for Dissection Rupture. The preoperative rupture risk for ATAAD also rose with increasing false lumen diameter. The lower 95% CI of OR tended to be >1 and gradually increased when the false lumen diameter of the ascending aorta was up to 36.3 mm (Figure 5(a)). The smallest false lumen diameter in the rupture group was 13.57 mm, and the largest was 53.07. When the false lumen diameter of the ascending aorta was up to 41.65 mm and above, 90% (72/80) of patients suffer rupture in the rupture group.

Change in the false lumen/true lumen diameter ratio was also associated with preoperative rupture. When it was up to 2.76, the lower 95% CI of OR reached 1 and increased gradually (Figure 5(b)). The smallest false lumen/true lumen diameter ratio in the rupture group was 0.04, and the largest was 8.31. When the false lumen/true lumen diameter ratio was up to 5.38 and above, 90% (72/80) of patients suffer rupture in the rupture group.

3.6. Threshold Analysis for Dissection Rupture in Different States of False Lumen Thrombosed. When patent thrombosed in the false lumen, the lower 95% CI of OR was >1 when the diameter of the false lumen of the ascending aorta was between 22.9 and 26.7 mm (Figure 6(a)). When partial or complete thrombosed in the false lumen, the lower 95% CI of OR was >1 when the diameter of the false lumen of the ascending aorta started greater than 26.7 mm, and the maximum value of OR was attained when the false lumen of the ascending aorta was up to 37.9 mm, which was significantly greater than that when the OR reached its maximum value in the patent thrombosed state (Figure 6(b)).

4. Discussion

Rupture of acute aortic dissection is a catastrophic clinical event, which has become the leading cause of prehospital death in ATAAD patients [6]. Mehta et al. retrospectively analyzed 574 cases of ATAAD and found that during conservative medical treatment, 33.3% died of dissection rupture [7]. Hagan et al. also reported that up to 41.6% of ATAAD patients died of dissection rupture [8]. ATAAD often involves the aortic sinus, ascending aorta, and proximal arch in the pericardial cavity. However, Rylski et al. [9] found that all of the aortic morphology did not change significantly after the occurrence of dissection, especially changes in the aortic sinus, which was possibly related to the fixation effect of the valve annulus. In contrast, the ascending aortic morphology changed significantly [9]. In this study, although the geometry of the aorta changed in both the preoperative rupture group and the surgical group, there were no significant differences in the mean diameter of the aortic sinus, the proximal end of the aortic arch, and the false lumen at the proximal end of the aortic arch between both groups. The geometric difference was mainly found in the ascending aorta (50.14 ± 4.68 mm vs. 48.11 ± 6.08 mm, $P = 0.001$); thus, the ascending aorta may be the target location of dissection lesions causing disastrous preoperative rupture. Based on autopsies, Mészáros et al. [10] confirmed that the rupture site in preoperative rupture ATAAD patients was mainly in the ascending aorta. Newly increased blood of the pericardial cavity confirmed by bedside ultrasound also proved that the ascending aorta was the location of preoperative aortic dissection rupture [10, 11]. Rupture of ascending aortic dissection also obeyed the law that the rupture rate increased with an increase in diameter; however, it was not completely equivalent to the pattern that with the gradual expansion of true aneurysms beyond the critical line, rupture events increased suddenly. Recent studies [12, 13] reported that the diameter of the ascending aorta was <50 mm after dissection in approximately half of the patients. Therefore, different from aneurysm, the pathological characteristics of the false lumen are an important reason for the preoperative rupture of aortic dissection in addition to the influence of the total diameter [7].

The false lumen of aortic dissection is only covered by the adventitia and a part of the media, which is prone to rupture [14, 15]. We found that the diameter of the false lumen in the rupture group was significantly larger than that in the surgical group (35.09 ± 9.96 mm vs. 25.99 ± 4.34 mm, $P < 0.001$); moreover, the preoperative rupture risk rose with an increase in false lumen diameter. The lower 95% CI of OR tended to be >1 and gradually increased when the false lumen diameter of the ascending aorta was up to 36.3 mm. The false lumen/true lumen ratio was also used to describe the relationship between the two groups in our study. The ratio of false lumen/true lumen in the rupture group was significantly different from that in the operation group (3.55 ± 2.05 vs. 2.66 ± 2.06, $P < 0.001$), and patients with severe true lumen compression (false lumen/true lumen $\geq 4:1$) accounted for 36.3% vs. 11.2%, also with different rupture rates among different ratios of false lumen/true lumen (36.3% ($\geq 4:1$) vs. 12.5% ($\geq 3:1$) vs. 20.0% ($\geq 2:1$) vs. 21.3% ($\geq 1:1$) vs. 10.0% ($<1:1$)). An increase in the false lumen/true lumen ratio is a manifestation of increased false lumen pressure, which may be due to a limited extent of aortic dissection (such as DeBakey II type), the lack of a distal breach, or a reverse tear of ATAAD. As the diameter of the false lumen increases, the external tension in the false lumen increases according to Laplace's law and rupture is more likely to occur. Meanwhile, the high pressure in the larger false lumen can cause compression or even a complete block of the true lumen, leading to organ tissue ischemia called "malperfusion syndrome," which is defined as compromised blood flow in 1 or more organs resulting in ischemia and organ dysfunction, such as myocardial infarction, cerebral infarction, lower extremity ischemia, or renal insufficiency, and it remains a severe condition associated with adverse outcomes in patients with ATAAD [16].

Many studies have shown that age is a risk factor for in-hospital mortality in ATAAD [17, 18]. Increased vascular wall stiffness in elderly patients is a cause of susceptibility to rupture [19, 20]. However, multivariate analysis indicated that age was not an independent risk factor for preoperative rupture. This may be because the average age of patients in this study (53.8 ± 11.4 vs. 49.6 ± 9.7) was lower than that in Western countries. We found that an increase in the diameter of the ascending aorta and the diameter of the false lumen was the main reason for rupture. Lesion on the aortic wall itself is the main reason determining whether dissection is prone to

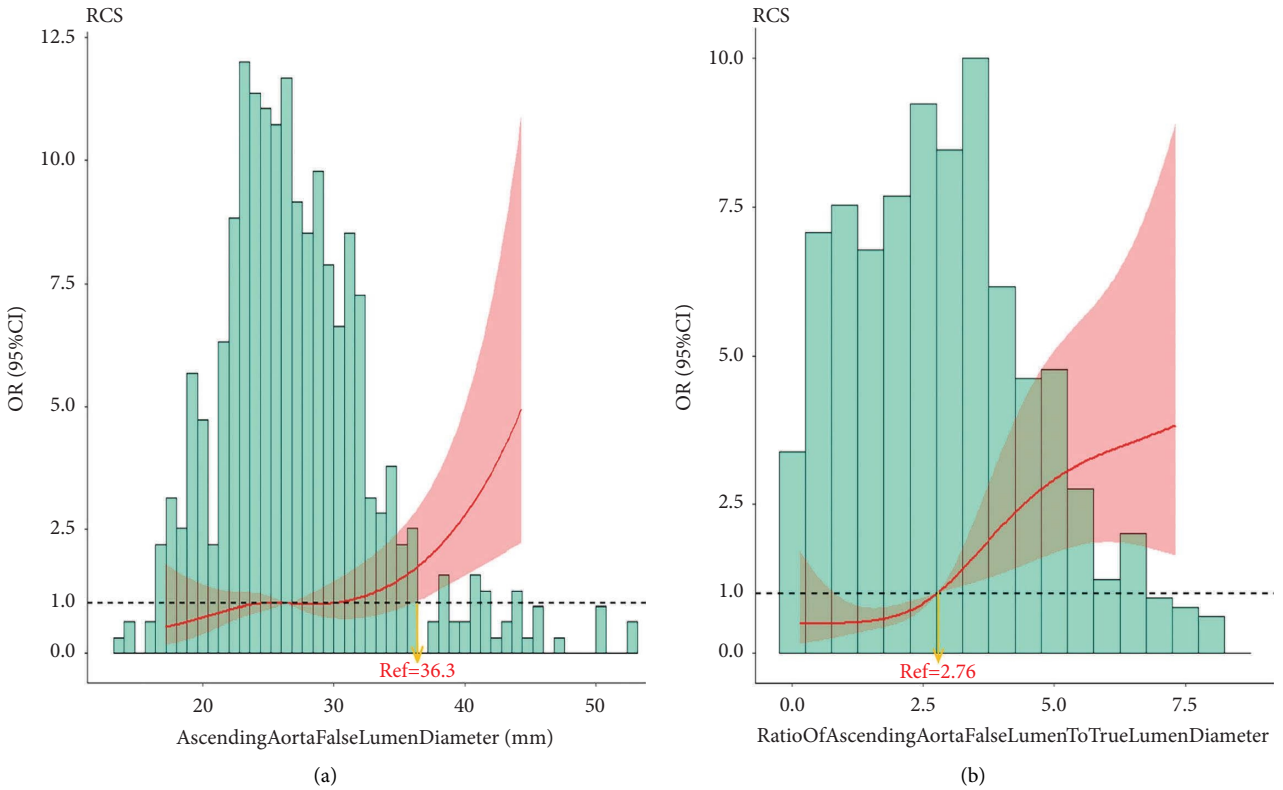

Figure 5: Threshold analysis of ascending aortic false lumen diameter (a) and false lumen/true lumen diameter ratio (b) for aortic dissection rupture.

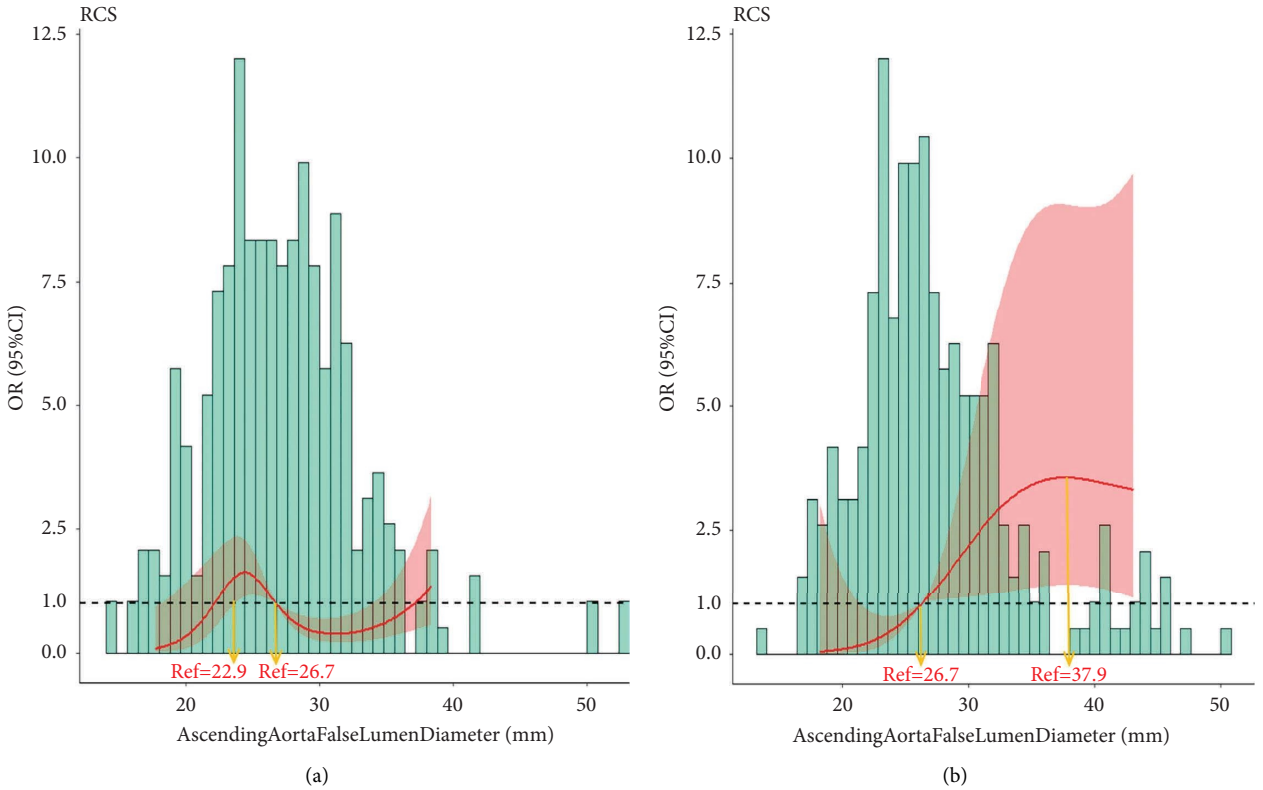

Figure 6: Threshold analysis of ascending aortic false lumen diameter for aortic dissection rupture patent (a) or partial or complete (b) thrombosed in false lumen.

occur, which is different from whether it is prone to rupture after the occurrence of dissection. Similarly, lesions of the middle tissue in patients with Marfan syndrome are prone to aortic dissection [11] but do not increase the preoperative rupture risk itself in patients with acute dissection. Patients with Marfan syndrome mainly present with dilation of the aortic sinus. In our study, the diameter of the aortic sinus was not directly an independent risk factor for aortic dissection rupture. Moreover, some studies show that Marfan syndrome may even slow down the rupture in patients with aortic dissection due to increased tissue compliance caused by the medial lesion, which is more resistant to vasodilation and reduces vascular wall tension [21].

Aortic dissection rupture is affected by a combination of factors such as emergency window, uncontrolled blood pressure, persistent pain, and disease of the media aortic layer [22, 23]. Anagnopoulos et al. [24] found that treatment time and clinical events were closely related. In this study, most patients were transported to our center in a timely manner after onset. The average time interval from onset to the clinical event (including Time 1: time from onset to admission; Time 2: time from admission to surgery or rupture; Time 3: time from the onset to surgery or rupture) between both groups did not differ significantly (with all $P > 0.05$, Table 1), reducing the interference with the study results due to discrepancies in the emergency window. Although with the improvement of medical equipment nowadays, diagnosis of acute aortic syndromes (AAS) may be challenging particularly in physicians who are inexperienced with AAS, misdiagnosis of AAS does not uncommonly occur in patients transferred to by non-experienced referring facilities. The nearly 10% frequency of misdiagnosis was observed in two separate geographic institutional aortic center, and all of the misdiagnosis was due to imaging misinterpretation including imaging artifacts and expected postsurgical changes [25]. However, we reduce the impact of misdiagnosis on this study through the following ways: in the case of suboptimal outside imaging, most of the referred patients in this study experienced repeat imaging, and the diagnosis was reviewed by two experienced board-certified imaging specialists. In addition, we also excluded the patients with difficulty to distinguish the true and false lumen and with a history of ascending aortic surgery. By doing this, none received unnecessary surgery as the misdiagnosis was detected beforehand. Even so, the high rupture rate of 14.2% still surprised us. We analyzed that it was possibly due to the uneven levels of care during transport and less early experience of the operative facility despite expedited care of the aortic dissection patients. In particular, there are quite a few patients with an aura rupture occurred rupture shortly after admission to our hospital. Besides, 3 patients suffered rupture during anesthesia and 5 patients ruptured in route to the operating room who were characterized by sudden confusion, a sharp drop in blood pressure, or a slowdown in heart rate and pulse, without the opportunity to be operated on, indicating again the lethality and urgency of ATAAD [1]. What is more, some patients were first referred to the relevant department for suspected acute coronary syndrome or acute pulmonary embolism, leading to delays in diagnosis and treatment. The above reasons may have resulted in a higher preoperative rupture rate. Blood pressure control is also a key factor in the occurrence of preoperative rupture. In this study, the blood pressure of patients in both groups was well controlled after admission. There were no significant differences in the systolic blood pressure between both groups of patients (120.1 ± 29.5 mm·Hg vs. 122.4 ± 23.6 mm·Hg, $P = 0.509$), but more patients in the preoperative rupture group experienced preoperative shock (7.8% vs. 2.7%, $P = 0.040$) and had hypotensive state (10.0% vs. 3.1%, $P = 0.009$) mainly due to moderate and large pericardial effusions. Therefore, a new or sudden increase in pericardial effusion is usually a warning sign for dissection rupture.

After thrombus formation in the false lumen, blood flow in the false lumen gradually decreases, weakening the shear force on the vascular wall. Thus, the false lumen gradually shrinks and the risk of rupture decreases. We found no discrepancy in false lumen thrombosed between the groups ($P = 0.177$), but when the false lumen was partial or complete thrombosed, the diameter threshold for maximum risk of preoperative rupture (37.9 mm) was significantly greater than that when the false lumen was patent (22.9 to 26.7 mm). It is likely because thrombosis greatly increases the diameter threshold of the false lumen to cause dissection rupture.

Moreover, we found DeBakey II type aortic dissection is an independent risk factor for ATAAD. This type of aortic dissection is characterized by a location of origin break and the limited extent of dissection in the ascending aorta. As the initial site of dissection is limited at the junction of the heart and aorta and there is no reentry tear in the descending aorta, the false lumen remains pressurized due to lack of decompression resulting from reentry tear and is at higher risk for rupture.

Thus, our center is applying the results of this study to current practice: the cardiopulmonary circulation team and the anesthesia team of our unit have specially scheduled emergency classes for off-shift time. When the independent risk factors analyzed above are met, we will immediately perform emergency surgery; otherwise, we will delay the operation to the shift time under the premise of drug treatment.

4.1. Limitations. Our study has several limitations. First, a single-center study may have introduced bias. Second, information on some important clinical risk factors for aortic dissection rupture was not prospectively collected, which may have under- or overestimated the effects of certain risk factors. Third, previous comorbidity data and biochemical data were incomplete in some patients who ruptured shortly after admission. Last, there was a lack of analysis of in-transit rupture patients due to the absence of their medical information.

5. Conclusions

Collectively, CTA is one of the most convenient tools for the early prediction of preoperative rupture in patients with ATAAD.

Abbreviations

ATAAD: Acute type A aortic dissection
CTA: Computed tomography angiography
OR: Odds ratio
LASSO: Least absolute shrinkage and selection operator
Q1: First quartile
Q3: Third quartile
BMI: Body mass index
CAD: Coronary artery disease
MFS: Marfan syndrome
LVEF: Left ventricular ejection fraction
AR: Aortic regurgitation.

Data Availability

The data that support the findings of this study are available from Fujian Cardiac Medical Center but restrictions apply to the availability of these data, which were used under license for the current study, and so are not publicly available. Data are, however, available from the authors upon reasonable request and with permission of Fujian Cardiac Medical Center.

Ethical Approval

The Ethics Committee of Fujian Medical University Union Hospital approved the present study and waived the need for informed consent based on the nature of the retrospective study.

Disclosure

Yi-Dong, Zai-Rong Lin, and Zeng-Rong Luo shared the first authorship.

Conflicts of Interest

All authors declare that they have no conflicts of interest.

Authors' Contributions

Yi-Dong wrote the main manuscript text. Zeng-Rong Luo, Zai-Rong Lin, and Liang-Wan Chen prepared Figures 1–6 and Table 1. All authors reviewed the manuscript. Yi-Dong, Zai-Rong Lin, and Zeng-Rong Luo contributed equally to the study.

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