Original Article

Prognostic value of left ventricular hypertrophy in postoperative outcomes in type A acute aortic dissection

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Background: Left ventricular hypertrophy (LVH) is common in hypertension patients. Hypertension is a recognized risk factor of acute aortic dissection. This study aimed to explore the prognostic value of LVH in predicting postoperative outcomes in acute type A aortic dissection (ATAAD) patients.

Methods: This was a single-central retrospectively designed study. One hundred and ninety-three ATAAD patients who underwent surgical repair at Renmin Hospital of Wuhan University from January 2018 to November 2021 were enrolled. Patients were divided based on their left ventricular mass index (LVMI). We compared their baseline characteristics, perioperative data, and in-hospital outcome. Then nomogram models were developed based on logistic regression to predict the postoperative outcomes.

Results: LVH presented in 28.5% (55 in 193) patients. LVH group had a higher proportion of female patients compared with the non-LVH group (32.7% *vs.* 17.4%, P=0.03). Decreased left ventricular ejection fraction and cardiac tamponade were more prevalent in patients with LVH. LVH group had a higher risk of postoperative composite major outcomes (CMO) and operative mortality. Based on multivariable logistic regression, LVH/LVMI, Penn classification, hyperlipidemia, emergency surgery and cardiopulmonary bypass duration were applied to develop nomogram models for predicting postoperative CMO. The area under curve was 0.825 (95% CI: 0.749–0.900) for Model LVH and 0.841 (95% CI: 0.776–0.905) for Model LVMI. Nomogram models for predicting postoperative cardiac were developed based on LVH/LVMI and cardiopulmonary bypass duration. The area under curves for the models involving LVH or LVMI were 0.782 (95% CI: 0.640–0.923) and 0.795 (95% CI: 0.643–0.947), respectively.

Conclusions: LVH and increased LVMI was associated with increased risk of postoperative CMO and cardiac events in ATAAD patients. The nomogram models based on LVH or LVMI might help predict postoperative CMO. Future research would be necessary to investigate prognostic value of LVH for long-term outcomes in ATAAD patients.

Keywords: Left ventricular hypertrophy (LVH); acute aortic dissection; surgery; risk factor; outcome

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Introduction

Acute aortic dissection (AD) is an urgent, life-threatening medical condition with rapid chest pain as the most common symptom at onset, that has an extremely high mortality (1,2). Acute aortic dissection is classified as acute type A AD (ATAAD) and acute type B AD based on the involvement of the ascending aorta, that differs in symptom, management, and outcome (3). Usually, ATAAD, in which the ascending aorta was involved, needs swift open surgical repair after initial diagnosis, including classic Bentall procedure, wheat procedure and frozen elephant trunk technique. Despite the improvement of clinical outcomes after surgical repair over time, the mortality of ATAAD is still high, about 1 in 5 patients died after surgery (1,2,4-7).

Hypertension is a common condition in AD, with a prevalence of 75–80% among patients with AD (8). Hypertension can be triggered by many factors, such as obesity, genetic background and salt intake (9-12). Heart is one of the major target organs in hypertension-related organs damage (13,14). Left ventricular hypertrophy (LVH), which presents in approximately two-fifth of hypertension patients, is reported to be associated with increased cardiovascular morbidity and mortality, including sudden cardiac death, heart failure, arrhythmias, etc. (14-16). Also, research indicates that LVH is a risk factor of enlarged aorta and dissection (17).

A previous study demonstrates LVH as a biomarker to predict increased mortality in type B AD patients (18). However, the association between LVH and ATAAD remains unknown. Herein, we investigated the prognostic value of LVH in AD patients after surgical repair, and developed nomogram models to predict postoperative outcomes in ATAAD patients. We present the following article in accordance with the TRIPOD reporting checklist (available at https://jtd.amegroups.com/article/view/10.21037/jtd-22-193/rc).

Methods

Study population and data collection

From 1 January 2018 to 31 November 2021, all adult patients (≥18 years) diagnosed with ATAAD in Renmin Hospital of Wuhan University were included. Imaging data (computed tomography angiography and transthoracic/transesophageal echocardiogram) was checked for confirmation. The predefined exclusion criteria were as follows: (I) simple intramural hematoma; (II) traumatic/iatrogenic AD, AD with pregnancy, or patients who had previous cardiac surgery; (III) patients with congenital aortic abnormalities; (IV) patients without complete medical records available.

Demographic and clinical data were extracted individually from original medical records, including gender, age, weight, height, symptoms, medical background (diabetes mellitus, hypertension, coronary artery disease), smoking, alcohol consumption, laboratory biomarkers, electrocardiogram, ultrasound imaging, operation data, and in-hospital outcome. Hypertension was defined as follows, (I) patients with a history of previously diagnosed hypertension, regardless of blood pressure (BP) status. (II) patients with increased BP on admission (systolic BP >140 mmHg or diastolic BP >90 mmHg), or patients who were taking antihypertensive agents with normal BP level on admission.

Left ventricular mass index (LVMI) was calculated based on echocardiogram data, as reported previously (19). LVH was defined as LVMI ≥115 $g{\cdot}m^{-2}$ for males, or LVMI ≥95 $g{\cdot}m^{-2}$ for females. The malperfusion was presented with Penn Classification as reported (20). The patients were divided into two groups, LVH and non-LVH (nLVH) group, based on their LVMI.

Study endpoints and operation procedure

The primary endpoints were postoperative complications within 30 days as follows: operative mortality, strokes, paraplegia, continuous renal replacement therapy (CRRT), and cardiac events. Cardiac events were defined as low cardiac output syndrome or ventricular arrhythmias. To evaluate the in-hospital outcomes, a parameter named composite major outcomes (CMO) was utilized for patients with at least one primary endpoint event. The secondary endpoints were re-exploration for postoperative bleeding, tracheotomy, and new-onset atrial fibrillation after surgery.

The operation plan was decided by experienced surgeons. Moderate hypothermic circulatory arrest was applied for patients required arch replacement. Cold antegrade custodial-histidine-trypthophan-ketoglutarate solution (Custodial-HTK) was applied for myocardial preservation.

Statistical analysis

Statistical analysis was performed utilizing the R 4.0.5 (R Foundation for Statistical Computing, Vienna, Austria). A two-tailed P value of <0.05 was considered statistically significant.

Normally distributed continuous variables were expressed as mean ± standard deviation (SD) and compared with student's *t*-test. Skewed continuous variables were expressed as the median and interquartile range (IQR) and compared with Mann-Whitney U-test. Categorical variables are described as frequencies with percentages, and analyzed by Fisher's exact test. Shapiro-Wilk-test was used to evaluate the normality of continuous data. Logistic regression analysis was performed to evaluate the correlation and select predictors for the nomogram model. The bootstrap method was applied for internal validation. Calibration curve and decision curve analysis were applied to assess model performance. Propensity score matching was applied for confounding control.

Nomogram models were developed based on multivariable logistic regression. Variables with a P value <0.05 were selected for model development. LVH and LVMI were used separately for model development. Calibration curve and decision curve analysis were used to assess model performance.

Patient and public involvement statement

The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). This study was approved by the ethics commission of Renmin Hospital of Wuhan University (WDRM2020-K230). Informed consent was not required due to its retrospective nature.

Results

Demographic and clinical data

One hundred and ninety-three patients were included in the final analysis (Figure S1). Demographic and clinical data were summarized in *Table 1*. The two groups did not differ in age. However, there were more females in patients with LVH (32.7% *vs.* 17.4%, P=0.03). Patients without LVH had a higher median BMI of 25.0 $kg{\cdot}m^{-2}$. However, the proportion of overweight/obesity was similar among the two groups. Sudden anterior chest pain was a common symptom in both groups. No significant difference in the prevalence of hypertension (94.5% in LVH group *vs.* 89.1% in nLVH group, P=0.29). Patients with LVH had higher presenting diastolic BP (82.2±17.2 *vs.* 76.0±19.2 mmHg, P=0.04) and higher presenting pulse pressure (79.8±17.5 *vs.* 67.9±20.1 mmHg, P<0.01), but similar systolic BP (138.3±24.1 *vs.* 141.0±29.6 mmHg, P=0.51). Renal dysfunction was more common in the LVH group (25.5% in the LVH group *vs.* 12.3% in the nLVH group, P=0.03). No other apparent differences were found among the 2 groups in terms of hyperlipidemia, coronary artery disease, diabetes mellitus, alcohol consumption, smoking, liver lesions, or hypoxemia at admission.

Perioperative data and laboratory examination

Laboratory examination results were presented in *Table 2*. Patients with LVH had slightly decreased hemoglobin and alanine aminotransferase concentrations. There were no significant differences among the two groups in terms of white blood cell counts, neutrophil counts, platelet counts, total bilirubin urea, creatine, uric acid, blood glucose, fibrinogen, or d-dimer concentration.

Table 3 presented the perioperative and postoperative information data of the two groups. Patients with LVH were more likely to experience cardiac tamponade and decreased left ventricular ejection function. Ultra-sound detected aortic valve insufficiency was common in both two groups, that 58.2% of the LVH group and 50.7% of the nLVH group had aortic insufficiency. About 43.6% of patients with LVH underwent surgical repair within the first 24 hours of admission, while 37.7% of patients without LVH underwent emergency surgery. The overall median (IQR) of cardiopulmonary bypass (CPB) duration, aortic cross-clamping duration, and circulatory arrest duration were 267.0 (241.0–297.0), 140.0 (124.0–165.0), and 31.0 (20.0–38.0) min, respectively. In terms of surgical procedures, there was no apparent difference among the two groups.

As showed in *Table 3*, CMO occurred in 17 (30.9%) of 55 LVH patients and 21 (15.2%) of 138 nLVH patients. LVH group had higher operative mortality of 18.2%, while the nLVH group had operative mortality of 7.2% (P=0.04). LVH patients had a higher prevalence of postoperative stroke (4 in 55 patients) and cardiac events (7 in 55 patients). In the nLVH group, the prevalence of stroke and cardiac events were 1.4% (2 in 138) and 4.3% (6 in 138), respectively. However, they narrowly missed the significant point. New-onset atrial fibrillation after surgery was present in 6 (10.9%) patients with LVH, while in the nLVH group only 4 (2.9%) patients had new-onset atrial fibrillation (10.9% *vs.* 2.9%, P=0.03). Two groups had no significant differences in paraplegia, CRRT, re-exploration, or tracheotomy after surgical repair.

Table 1 Demographic and clinical characteristics

Characteristics	Overall (N=193)	Left ventricular hypertrophy		P value
		Presence (N=55)	Absence (N=138)	
Age (y)	52.9±10.8	53.2±10.5	52.8±10.9	0.817
Gender				
Male	151 (78.2)	37 (67.3)	114 (82.6)	0.032*
Female	42 (21.8)	18 (32.7)	24 (17.4)	
LVMI ($g·m^{-2}$)	99.4 (87.2–111.9)	123.7 (115.7–142.8)	91.1 (82.8–102.4)	<0.001*
Body mass index ($kg·m^{-2}$)	24.8 (22.7–27.2)	23.7 (21.9–26.0)	25.0 (23.0–27.7)	0.016*
Overweight	70 (36.3)	15 (27.3)	55 (39.9)	0.086
Obesity	42 (21.8)	10 (18.2)	32 (23.2)	
Blood type				
A	60 (31.1)	12 (21.8)	48 (34.8)	0.091
B	42 (21.8)	18 (32.7)	24 (17.4)	
O	83 (43.0)	23 (41.8)	60 (43.5)	
AB	8 (4.1)	2 (3.6)	6 (4.3)	
Presenting symptoms				
Chest pain (anterior)	162 (83.9)	43 (78.2)	119 (86.2)	0.194
Back pain	113 (58.5)	33 (60.0)	80 (58.0)	0.872
Syncope	12 (6.2)	3 (5.5)	9 (6.5)	1.000
Medical background				
Hyperlipidemia	71 (36.8)	21 (38.2)	50 (36.2)	0.869
CAD	17 (8.8)	6 (10.9)	11 (8.0)	0.576
Diabetes mellitus	11 (5.7)	3 (5.5)	8 (5.8)	1.000
Hypertension	175 (90.7)	52 (94.5)	123 (89.1)	0.287
Presenting blood pressure				
Systolic BP (mmHg)	140.2±28.2	138.3±24.1	141.0±29.6	0.514
Diastolic BP (mmHg)	77.8±18.8	82.2±17.2	76.0±19.2	0.038*
PP (mmHg)	71.3±20.1	79.8±17.5	67.9±20.1	<0.001*
Drinking history	43 (22.3)	9 (16.4)	34 (24.6)	0.253
Smoking history	71 (36.8)	18 (32.7)	53 (38.4)	0.511
Ultrasound-detected liver lesions	47 (24.4)	11 (20.0)	36 (26.1)	0.459
Hypoxemia[†]	68 (35.2)	18 (32.7)	50 (36.2)	0.739
Renal function				
Cr >140 mmol/L	31 (16.1)	14 (25.5)	17 (12.3)	0.031*

Data are expressed as mean ± standard deviation or medians and interquartile ranges or numbers (percentages). *, P value <0.05; [†], hypoxemia was defined as an artery oxygen partial pressure <60 mmHg on admission. LVMI, left ventricular mass index; CAD, coronary artery disease; BP, blood pressure; PP, pulse pressure; Cr, creatinine.

Table 2 Laboratory examination data

Biomarkers	Overall (N=193)	Left ventricular hypertrophy		P value
		Presence (N=55)	Absence (N=138)	
WBC (10^9/L)	12.32 (10.30–14.29)	11.71 (8.74–15.12)	12.51 (10.48–14.23)	0.396
Neu (10^9/L)	10.26 (8.29–12.49)	9.76 (7.50–12.62)	10.46 (8.65–12.47)	0.423
Hb (g/L)	130.1±18.8	125.8±21.6	132.0±17.5	0.040*
Plt (10^9/L)	162.0 (134.0–191.0)	150.5 (135.0–191.0)	163.0 (134.0–191.0)	0.340
ALT (U/L)	23.0 (15.0–34.5)	19.5 (13.0–25.0)	24.0 (17.0–36.0)	0.006*
TBil (μmol/L)	16.03 (11.62–22.62)	15.80 (10.75–22.19)	16.80 (11.86–22.75)	0.287
Urea (mmol/L)	6.85 (5.70–8.48)	6.91 (5.97–9.86)	6.77 (5.57–8.20)	0.070
Cr (μmol/L)	84.0 (66.5–118.5)	89.0 (65.0–140.0)	83.0 (67.0–118.0)	0.456
UA (μmol/L)	399.0 (320.5–489.0)	409.0 (339.0–490.0)	398.0 (313.0–488.0)	0.270
Glucose (mmol/L)	7.14 (6.18–8.40)	7.36 (6.55–8.60)	7.01 (6.05–8.40)	0.226
FIB (g/L)	2.16 (1.68–3.14)	2.13 (1.62–2.78)	2.18 (0.75–3.32)	0.555
D-dimer (mg/L)	6.44 (3.38–13.92)	7.96 (4.42–15.01)	5.73 (3.32–12.80)	0.080

*, P value <0.05. WBC, white blood cell counts; Neu, neutrophil; Hb, hemoglobin; Plt, platelets; ALT, alanine aminotransferase; TBil, total bilirubin; Cr, creatinine; UA, uric acid; FIB, fibrinogen.

Risk factors for CMO and cardiac events

To investigate risk factors for postoperative CMO, a univariate logistic regression was performed. Perioperative data, including baseline characteristics and operative information, was included in univariate logistic regression as summarized in *Table 4*. Results indicated that, LVH (OR: 2.5, 95% CI: 1.2–5.2, P=0.02), LVMI (per 10 g·m^{-2}) (OR: 1.2, 95% CI: 1.0–1.3, P<0.01), ischemia (Penn Classification Ac, or Ab&c) (OR: 15.4, 95% CI: 4.0–59.9, P<0.01), hyperlipidemia (OR: 3.4, 95% CI: 1.6–7.1, P<0.01), renal dysfunction (OR: 3.3, 95% CI: 1.4–7.6, P<0.01) and emergency surgery (OR: 3.4, 95% CI: 1.6–7.1, P<0.01) were risk factors for postoperative CMO in ATAAD patients. Moreover, the increased durations of operation, including CPB duration (per 10 minutes) (OR: 1.1, 95% CI: 1.0–1.2, P<0.01), aortic cross-clamping duration (per 10 minutes) (OR: 1.1, 95% CI: 1.0–1.2, P=0.05) and circulatory arrest duration (per 5 minutes) (OR: 1.0, 95% CI: 1.0–1.4, P≤0.01), were associated with CMO.

Another univariate analysis (cardiac events as endpoint) was performed and presented in *Table 4*. Consistent with previous results, LVH (OR: 3.2, 95% CI: 1.0–10.0, P=0.04), LVMI (per 10 g·m^{-2}) (OR: 1.2, 95% CI: 1.1–1.3, P<0.01) and CPB duration (per 10 minutes) (OR: 1.1, 95% CI: 1.0–1.2, P<0.01) were predictors for postoperative cardiac events. However, hyperlipidemia narrowly missed the significant point (OR: 3.0, 95% CI: 0.9–9.5, P=0.06).

Multivariate logistic regression model for CMO

Multivariate logistic regression was performed to identify independent predictors for CMO. Indicators with a P value of less than 0.05 in univariate analysis were included in the multivariate model. Aortic cross-clamping duration and circulatory arrest duration were excluded from the model, as shown in Table S1. Variables included in the final multivariate analysis for CMO were LVH/LVMI, Penn Classification, hyperlipidemia, smoking, renal dysfunction, coronary artery disease, emergency surgery, and CPB duration. *Table 5* presented result of the multivariate analysis.

The results indicated that, LVH (OR: 2.6, 95% CI: 1.1–6.2, P=0.04), ischemia (Penn Classification Ac, or Ab&c) (OR: 13.5, 95% CI: 2.8–64.4, P<0.01), hyperlipidemia (OR: 3.0, 95% CI: 1.2–7.0, P=0.01), emergency surgery (OR: 2.8, 95% CI: 1.2–6.8, P=0.02) and increased CPB duration (per 10 minutes) (OR: 1.1, 95% CI: 1.0–1.2, P<0.01) were independent risk factors for postoperative CMO in ATAAD patients. Consistent with previous results, increased LVMI was independent risk factor for CMO when LVH was

Table 3 Perioperative data and postoperative outcomes

Characteristics	Left ventricular hypertrophy		P value
	Presence (N=55)	Absence (N=138)	
Penn classification			
Penn Aa	26 (47.3)	70 (50.7)	0.874
Penn Ab	26 (47.3)	59 (42.8)	
Penn Ac/Ab&c	3 (5.5)	9 (6.5)	
Myocardial infarction	4 (7.3)	11 (8.0)	1.000
Maximum AAoD (mm)	41.9±7.6	40.9±8.0	0.448
Echocardiogram			
Decreased LVEF[†]	8 (14.5)	2 (1.4)	0.001*
Pericardial effusion			
Absence	28 (50.9)	63 (45.7)	0.030*
Presence	20 (36.4)	70 (50.7)	
Cardiac tamponade	7 (12.7)	5 (3.6) [‡]	
Aortic insufficiency			
Mild	16 (29.1)	37 (26.8)	0.162
Middle	10 (18.2)	29 (21.0)	
Severe	6 (10.9)	4 (2.9)	
Surgical repair			
Within 24 h	24 (43.6)	52 (37.7)	0.514
After 24 h	31 (56.4)	86 (62.3)	
Cannulation strategy			
Femoral artery	28 (50.9)	75 (54.3)	0.889
Axillary artery	1 (1.8)	2 (1.4)	
Femoral artery & axillary artery	26 (47.3)	61 (44.2)	
Operation durations			
CPB durations (min)	269.0 (238.5–308.0)	265.0 (243.0–297.0)	0.710
ACx durations (min)	139.0 (123.0–161.0)	142.5 (125.0–165.0)	0.526
CA durations (min)	33.0 (20.0–37.5)	31.0 (20.0–38.0)	0.736
CABG	3 (5.5)	10 (7.2)	0.761
Proximal reconstruction			
Modified Bentall	9 (16.4)	20 (14.5)	0.933
Aortic valve replacement/repair	6 (10.9)	17 (12.3)	
Valve conservative surgery	40 (72.7)	101 (73.2)	
Arch replacement	41 (74.5)	117 (84.8)	0.102
Distal aortic operation			
Frozen elephant trunk	30 (54.5)	67 (48.6)	0.683

Table 3 *(continued)*

Table 3 *(continued)*

Characteristics	Left ventricular hypertrophy		P value
	Presence (N=55)	Absence (N=138)	
Hybrid	24 (43.6)	65 (47.1)	
Automatic heart resuscitation	12 (21.8)	38 (27.5)	0.470
In-hospital outcome			
Composite major outcomes	17 (30.9)	21 (15.2)	0.017*
Operative mortality	10 (18.2)	10 (7.2)	0.035*
Stroke	4 (7.3)	2 (1.4)	0.056
Paraplegia	2 (3.6)	3 (2.2)	0.624
CRRT	9 (16.4)	12 (8.7)	0.131
Cardiac events	7 (12.7)	6 (4.3)	0.053
Re-exploration	1 (1.8)	4 (2.9)	1.000
Tracheotomy	5 (9.1)	6 (4.3)	0.299
Atrial fibrillation	6 (10.9)	4 (2.9)	0.033*

Data are expressed as mean ± standard deviation or medians and interquartile ranges or numbers (percentages). *, P value <0.05. †, decreased LVEF was defined as a LVEF <50%; ‡, compared with LVH group. Post hoc test was adjusted with Bonferroni method. AAoD, ascending aortic diameter; LVEF, left ventricular ejection fraction; CPB, cardiopulmonary bypass; ACx, aortic cross-clamping; CA, circulatory arrest; CABG, coronary artery bypass graft; CRRT, continuous renal replacement therapy.

replaced with LVMI, with an OR of 1.2 (95% CI: 1.0–1.3, P=0.02) for every 10 $g \cdot m^{-2}$ increase in LVMI.

Clinical features and in-hospital outcomes after propensity score matching

Propensity score matching was applied to reduce potential baseline confounding. Cardiac tamponade, hyperlipidemia, Penn classification, emergency surgery and renal dysfunction were included as covariates in the model Based on logistic regression results showed in *Table 4* and *Table 5*. Cases were matched in a 1:2 ratio to cases without LVH based on the propensity score with a standard caliper width of 0.2. Jitter plot and line plot for matching were presented in Figure S2.

Clinical features and in-hospital outcomes after matching were summarized in *Table 6*. After matching, ATAAD patients with LVH had higher rates of postoperative CMO (16/52 *vs.* 13/94, P=0.02). Despite the relatively higher rates of decreased LVEF in LVH patients, no association between decreased LVEF and postoperative CMO was found by logistic regression analysis (OR: 2.6, 95% CI: 0.6–11.5, P=0.21).

Univariable logistic regression analyses were applied to evaluate the prognostic value of LVH, as showed in *Table 7*. Two main variables, LVH and LVMI, were analyzed respectively. The results indicated that LVH was the risk factor for postoperative CMO (OR: 2.8, 95% CI: 1.2–6.4, P=0.02), while increasing LVMI was associated with higher risks of postoperative CMO (OR: 1.2, 95% CI: 1.0–1.3, P<0.01) and cardiac events (OR: 1.2, 95% CI: 1.0–1.3, P<0.01). In addition, increasing LVMI was associated with increased risk of postoperative CRRT (OR: 1.2, 95% CI: 1.0–1.3, P<0.01), tracheotomy (OR: 1.2, 95% CI: 1.0–1.3, P=0.02) and atrial fibrillation (OR: 1.2, 95% CI: 1.0–1.3, P=0.01).

Prognostic nomogram models for postoperative outcomes in ATAAD patients

Based on data from 193 enrolled patients, nomograms of postoperative CMO and cardiac events in ATAAD patients were developed and established, as shown in *Figure 1* and *Figure 2*. Two models for postoperative CMO, model LVH and model LVMI, were developed (details were present in Table S2). Nomograms can be interpreted by adding up the points assigned to each variable, as indicated at the top of the point scale. The total point projected on the bottom scale represents the probability of postoperative CMO or

Table 4 Univariable logistic regression for indicators of CMO and cardiac events

Characteristics	CMO			Cardiac events		
	OR	95% CI	P value	OR	95% CI	P value
Left ventricular hypertrophy	2.492	1.193–5.207	0.015*	3.208	1.027–10.02	0.045*
LVMI						
Linear, per 10g·m^{-2}	1.169	1.051–1.301	0.004*	1.194	1.061–1.343	0.003*
Age (y)						
Linear, per 10 y	0.865	0.630–1.186	0.367	1.252	0.733–2.140	0.410
≥60 y	0.588	0.241–1.436	0.244	1.875	0.584–6.024	0.291
Male gender	2.070	0.753–5.685	0.158	1.571	0.335–7.382	0.567
BMI (kg·m^{-2})						
Linear	1.018	0.928–1.116	0.701	1.035	0.899–1.193	0.630
Overweight	0.779	0.343–1.768	0.550	0.311	0.062–1.549	0.154
Obesity	1.027	0.413–2.552	0.955	1.113	0.307–4.039	0.871
Penn classification						
Penn Aa	–	–	Reference	–	–	Reference
Penn Ab	2.224	0.990–4.996	0.053	1.382	0.406–4.703	0.604
Penn Ac/Ab&c	15.45	3.988–59.89	<0.001*	3.640	0.623–21.26	0.151
Medical background						
Hyperlipidemia	3.418	1.640–7.122	0.001*	2.971	0.933–9.465	0.065
CAD	2.454	0.845–7.127	0.099	2.000	0.405–9.873	0.395
Diabetes mellitus	0.392	0.049–3.159	0.379	–	–	0.999
Drinking history	0.914	0.384–2.174	0.839	1.050	0.276–3.999	0.943
Smoking history	1.981	0.966–4.061	0.062	2.115	0.681–6.561	0.195
Decreased LVEF[†]	1.812	0.446–7.362	0.406	3.909	0.740–20.66	0.108
Cardiac tamponade	3.203	0.957–10.72	0.059	3.091	0.602–15.87	0.176
Ultrasound-detected liver lesions	1.139	0.506–2.563	0.753	1.416	0.415–4.828	0.578
Elevated total bilirubin	1.061	0.484–2.325	0.882	1.156	0.340–3.922	0.817
Hypoxemia	0.700	0.323–1.518	0.367	0.806	0.239–2.720	0.728
Renal dysfunction	3.304	1.432–7.619	0.005*	0.417	0.052–3.326	0.409
Myocardial infarction	0.272	0.035–2.137	0.216	–	–	0.999
Surgical timing						
After 24 h	–	–	Reference	–	–	Reference
Within 24 h	3.396	1.622–7.107	0.001*	1.877	0.606–5.816	0.275
Operation duration						
CPB duration (per 10 min)	1.133	1.057–1.214	<0.001*	1.131	1.036–1.233	0.006*
ACx duration (per 10 min)	1.098	1.000–1.206	0.049*	1.047	0.907–1.209	0.528
CA duration (per 5 min)	1.025	1.055–1.376	0.006*	1.111	0.917–1.348	0.283

Table 4 *(continued)*

Table 4 *(continued)*

Characteristics	CMO			Cardiac events		
	OR	95% CI	P value	OR	95% CI	P value
CABG	1.908	0.555-6.565	0.305	2.793	0.550–14.19	0.215
Proximal reconstruction						
Valve conservative root surgery	–	–	Reference	–	–	Reference
Modified Bentall procedure	1.101	0.409–2.969	0.848	2.347	0.670–8.215	0.182
Aortic valve replacement/repair	1.173	0.400–3.440	0.772	–	–	0.998
Arch replacement	1.228	0.470–3.209	0.676	2.795	0.351–22.23	0.331

*, P value <0.05; †, decreased LVEF was defined as a LVEF <50%. CMO, composite major outcomes; LVMI, left ventricular mass index; BMI, body mass index; CAD, coronary artery disease; LVEF, left ventricular ejection fraction; CPB, cardiopulmonary bypass; ACx, aortic cross-clamping; CA, circulatory arrest; CABG, coronary artery bypass graft.

Table 5 Multivariable logistic regression for indicators of CMO

Characteristics	β	S.E.	Wald	OR	95% CI	P value
LVH						
Left ventricular hypertrophy	0.942	0.448	4.409	2.564	1.065–6.175	0.036*
Penn Ab	0.558	0.488	1.306	1.748	0.671–4.552	0.253
Penn Ac/Ab&c	2.601	0.798	10.62	13.48	2.819–64.42	0.001*
Hyperlipidemia	1.085	0.441	6.066	2.960	1.248–7.020	0.014*
Renal dysfunction	0.768	0.559	1.890	2.156	0.721–6.448	0.169
Emergency surgical repair†	1.045	0.441	5.624	2.845	1.199–6.750	0.018*
CPB durations (per 10 min)	0.115	0.040	8.329	1.122	1.038–1.213	0.004*
Intercept	-6.638	1.274	27.16	–	–	–
LVMI						
LVMI (per 10 $g \cdot m^{-2}$)	0.149	0.063	5.686	1.161	1.027–1.312	0.017*
Penn Ab	0.574	0.491	1.366	1.776	0.678–4.653	0.242
Penn Ac/Ab&c	2.381	0.826	8.302	10.81	2.141–54.59	0.004*
Hyperlipidemia	1.084	0.442	6.013	2.958	1.243–7.036	0.014*
Renal dysfunction	0.797	0.552	2.082	2.219	0.752–6.554	0.149
Emergency surgical repair†	1.137	0.448	6.423	3.116	1.294–7.504	0.011*
CPB durations (per 10 min)	0.119	0.040	8.807	1.126	1.041–1.219	0.003*
Intercept	–8.087	1.488	29.55	–	–	–

†, emergency surgical repair was defined as surgery within first 24 h of admission; *, P value <0.05. CMO, composite major outcomes; S.E., standard error; OR, odds ratio; CI, confidence interval; LVH, left ventricular hypertrophy; CPB, cardiopulmonary bypass; LVMI, left ventricular mass index.

cardiac events. Collinearity analyses of model LVH and model LVMI were performed, as showed in Table S3. Collinearity was not found in both models. *Figure 3* showed the results of the calibration curve and decision curve analysis. Model LVH and model LVMI for postoperative CMO contained different indicators used in the nomogram.

Table 6 Clinical features and in-hospital outcomes after propensity score matching

Characteristics	Left ventricular hypertrophy		P value
	Presence (N=52)	Absence (N=94)	
Age (years)	53.0±10.5	52.7±11.0	0.899
Gender			
Male	36	77	0.099
Female	16	17	
LVMI ($g \cdot m^{-2}$)	125.2 (115.7–145.2)	90.8 (82.8–102.4)	<0.001*
Body mass index ($kg \cdot m^{-2}$)	23.7 (22.0–26.0)	25.0 (23.0–26.6)	0.150
Medical background			
Hyperlipidemia	19	37	0.859
Diabetes mellitus	3	8	0.747
Hypertension	49	83	0.380
Ultrasound-detected liver lesions	11	18	0.830
Renal function			
Cr >140 mmol/L	13	17	0.393
Penn classification			
Penn Aa	26	44	0.802
Penn Ab	23	46	
Penn Ac/Ab&c	3	4	
Myocardial infarction	3	6	0.700
Echocardiogram			
Decreased LVEF†	7	1	0.003*
Cardiac tamponade	4	4	0.456
Surgical repair			
Within 24 h	23	38	0.727
After 24 h	29	56	
Cannulation strategy			
Femoral artery	27	51	0.936
Axillary artery	1	1	
Femoral artery & axillary artery	24	42	
Operation durations			
CPB durations (min)	268.5 (236.0–308.0)	267.5 (245.0–296.0)	0.933
ACx durations (min)	138.0 (121.5–161.0)	142.5 (126.0–166.0)	0.313
CA durations (min)	33.0 (20.0–37.5)	31.0 (23.0–38.0)	0.871
Automatic heart resuscitation	10	24	0.421
In-hospital outcome			
Composite major outcomes	16	13	0.018*

Table 6 *(continued)*

Table 6 *(continued)*

Characteristics	Left ventricular hypertrophy		P value
	Presence (N=52)	Absence (N=94)	
Operative mortality	8	7	0.158
Stroke	4	2	0.187
Paraplegia	2	2	0.616
CRRT	8	7	0.158
Cardiac events	6	4	0.167
Re-exploration	1	3	1.000
Tracheotomy	5	3	0.133
Atrial fibrillation	6	3	0.069

*, P value <0.05. †, decreased LVEF was defined as a LVEF <50%. LVMI, left ventricular mass index; Cr, creatinine; LVEF, left ventricular ejection fraction; CPB, cardiopulmonary bypass; ACx, aortic cross-clamping; CA, circulatory arrest; CRRT, continuous renal replacement therapy.

Table 7 Univariable logistic regression for postoperative outcomes after propensity score matching

Characteristics	β	S.E.	Wald	OR	95% CI	P value
LVH						
CMO	1.019	0.424	5.778	2.769	1.207–6.354	0.016*
Operative mortality	0.815	0.550	2.200	2.260	0.770–6.636	0.138
Stroke	1.344	0.884	2.310	3.833	0.678–21.68	0.129
Paraplegia	0.610	1.015	0.361	1.840	0.252–13.46	0.548
CRRT	0.815	0.550	2.200	2.260	0.770–6.636	0.138
Cardiac events	1.077	0.670	2.579	2.935	0.789–10.92	0.108
Re-exploration	-0.520	1.168	0.198	0.595	0.060–5.867	0.656
Tracheotomy	1.172	0.752	2.427	3.227	0.739–14.09	0.119
Atrial fibrillation	1.375	0.730	3.551	3.957	0.946–16.54	0.060
LVMI (per 10 g·m^{-2})						
CMO	0.174	0.060	8.490	1.190	1.059–1.337	0.004*
Operative mortality	0.088	0.058	2.286	1.092	0.974–1.223	0.131
Stroke	0.096	0.076	1.603	1.101	0.949–1.278	0.205
Paraplegia	–0.039	0.166	0.056	0.962	0.695–1.331	0.813
CRRT	0.153	0.059	6.821	1.165	1.039–1.307	0.009*
Cardiac events	0.180	0.064	7.986	1.197	1.057–1.356	0.005*
Re-exploration	0.017	0.130	0.017	1.017	0.789–1.311	0.898
Tracheotomy	0.145	0.064	5.109	1.156	1.019–1.310	0.024*
Atrial fibrillation	0.154	0.063	5.994	1.166	1.031–1.318	0.014*

*, P<0.05. S.E., standard error; OR, odds ratio; CI, confidence interval; LVH, left ventricular hypertrophy; CMO, composite major outcomes; CRRT, continuous renal replacement therapy; LVMI, left ventricular mass index.

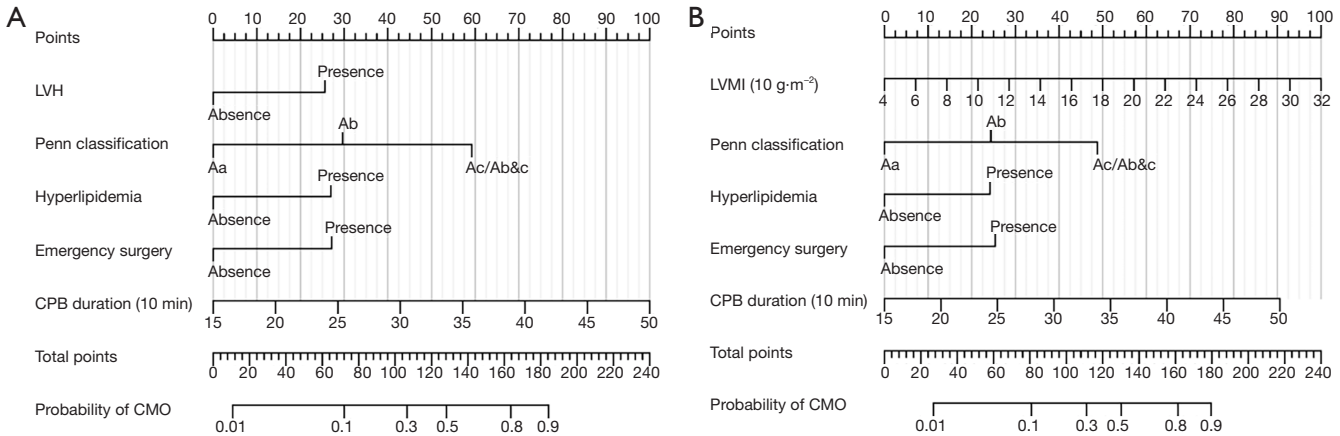

Figure 1 Nomograms for postoperative CMO. (A) nomogram for model LVH. (B) nomogram for model LVMI. Nomograms can be interpreted by adding up the points assigned to each variable, as indicated at the top of the point scale. The total point projected on the bottom scale represents the probability of postoperative CMO. CMO, composite major outcomes; LVH, left ventricular hypertrophy; LVMI, left ventricular mass index; CPB, cardiopulmonary bypass.

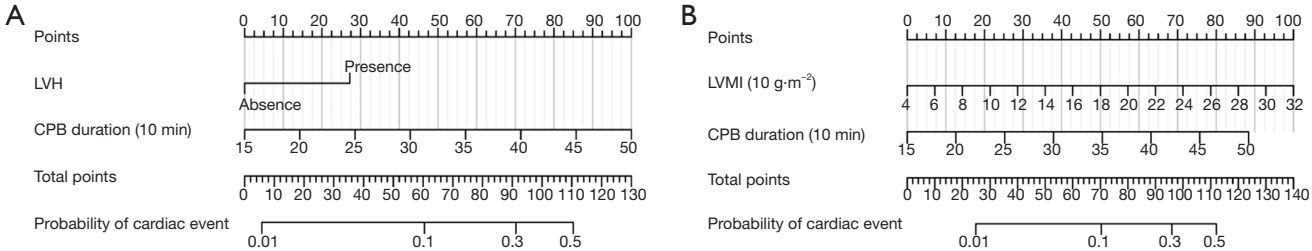

Figure 2 Nomograms for postoperative cardiac events. (A) Nomogram for model LVH. (B) Nomogram for model LVMI. Nomograms can be interpreted by adding up the points assigned to each variable, as indicated at the top of the point scale. The total point projected on the bottom scale represents the probability of postoperative cardiac events. LVH, left ventricular hypertrophy; LVMI, left ventricular mass index; CPB, cardiopulmonary bypass.

The area under curve was 0.825 (95% CI: 0.749–0.900) for Model LVH and 0.841 (95% CI: 0.776–0.905) for Model LVMI. The calibration curve and decision curve analysis indicated good clinical utility and consistency in both models in predicting postoperative CMO.

In addition, two models for postoperative cardiac events, model LVH and model LVMI, were developed and presented in *Figure 2* (details were present in Table S4). Collinearity analyses of model LVH and model LVMI for cardiac events were performed, as showed in Table S5. *Figure 4* showed the results of the calibration curve and decision curve analysis. The area under curve was 0.782 (95% CI: 0.640–0.923) for Model LVH and 0.795 (95% CI: 0.643–0.947) for Model LVMI. The calibration curve and decision curve analysis indicated good clinical utility and consistency in both models for postoperative cardiac events.

Discussion

Our main results were: (I) LVH was more prevalent in female patients with ATAAD. (II) Decreased left ventricular ejection fraction and cardiac tamponade were more prevalent in patients with LVH. (III) Increasing LVMI was associated with a higher risk of postoperative CMO and cardiac events. (IV) Nomogram models based on LVH/LVMI were developed for predicting postoperative CMO and cardiac events in ATAAD patients.

As a disastrous medical condition, acute aortic dissection has a high mortality rate, despite the 30-day mortality rate having decreased to 12.6% from 18.1% in recent two decades (5). Hypertension is diagnosed in approximately 80% of aortic dissection patients, which promotes aortic degeneration and weakens the aortic wall (21). Previous

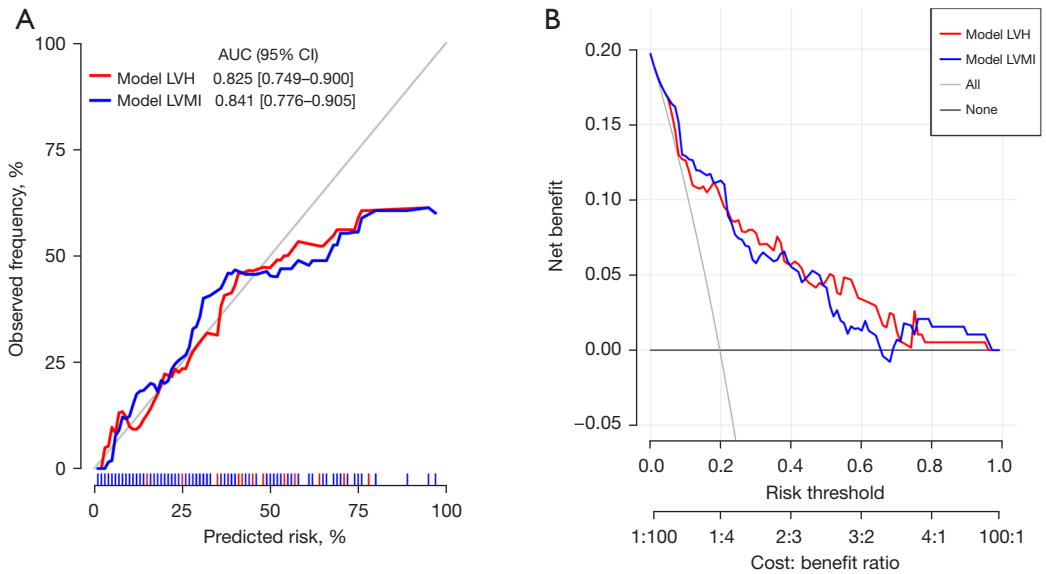

Figure 3 Validity test of the models for postoperative composite major outcomes. Both two models had an appropriate fit and a good predictive ability. (A) Calibration curve with area under curve (95% CI). (B) Decision curve analysis. AUC, area under the curve; CI, confidence interval; LVH, left ventricular hypertrophy; LVMI, left ventricular mass index.

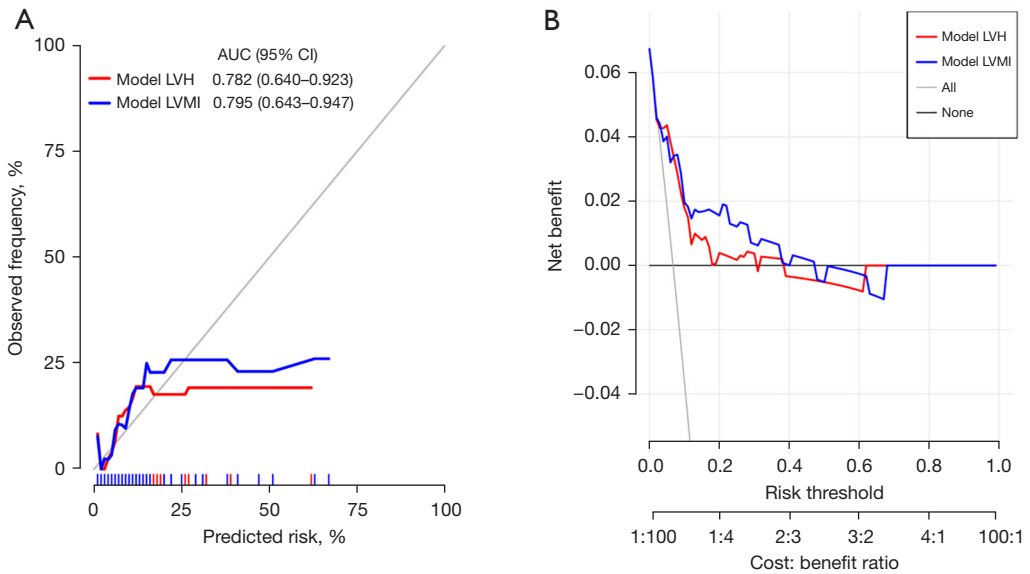

Figure 4 Validity test of the models for postoperative cardiac events. Both two models had an appropriate fit and a good predictive ability. (A) Calibration curve with area under curve (95% CI). (B) Decision curve analysis. AUC, area under the curve; CI, confidence interval; LVH, left ventricular hypertrophy; LVMI, left ventricular mass index.

study has highlighted that established hypertension is associated with target organ damage, in particular, the heart, kidney, brain, etc. (22). Ventricular hypertrophy is regarded as a result of uncontrolled hypertension. Increased BP leads to left ventricular remodeling, including concentric or eccentric LVH, which results in an increased risk of adverse cardiovascular diseases (15).

Our results confirmed that LVH, which was diagnosed with an increased LVMI, was the independent risk factor for both postoperative CMO and cardiac events (showed

in *Table 5* and Table S2). The major concerns about LVH are adverse cardiovascular events (including sudden death, ischemic heart disease, heart failure, arrhythmias, and stroke), and impaired left ventricular diastolic/systolic function that associated with geometric changes (15,23,24). Our results indicated that LVH was related to higher risk of CMO (30.9% *vs.* 15.2%, P=0.02) and new-onset postoperative atrial fibrillation (10.9% *vs.* 2.9%, P=0.03), especially in operative mortality (18.2% *vs.* 7.2%, P=0.04) before matching. These results differ from previous reports by Rocha *et al.* (25). Rocha *et al.* reported that left ventricular concentricity, instead of hypertrophy, was related to a higher risk of mortality (25). However, one-fourth of involved type A aortic dissection patients were subacute/chronic. The previous study has demonstrated the significant difference in early and late outcomes among acute and subacute/chronic aortic dissection (26). The different compositions of subjects involved might contribute to the different results.

Since LVH was a binary variable with predefined diagnostic criteria, we then assessed the prognostic value of LVMI as a continuous variable, as showed in *Table 7*. After propensity score matching, the increasing LVMI was associated with worse outcomes, including postoperative CMO, CRRT, cardiac events, tracheotomy and atrial fibrillation. Our results suggested a better predictive value of LVMI as a continuous variable compared with binary defined LVH. Previously study demonstrated LVMI as a strong independent predictor of perioperative mortality after adult cardiac surgery, including coronary artery bypass grafting and transcatheter aortic valve replacement (27-29). Increased LVMI indicated poor controlled hypertension or unaware hypertension, which was associated with other hypertensive mediated organ damage, including renal damage and vascular dysfunction (30). In fact, decreased left ventricular ejection fraction and renal dysfunction were more prevalent in patients with LVH, as showed in *Table 1* and *Table 3*. The hypertensive mediated organ damage, such as ventricular hypertrophy and renal dysfunction, might lead to poor prognosis for patients underwent cardiovascular surgery performed with CPB (31).

The results also indicated that the risk of CMO and cardiac events rapidly increased with prolonged CPB duration. Despite contemporary cardioprotective strategies having been well developed, ischemia-reperfusion injury and systemic inflammation that occurs during cardiopulmonary bypass may cause inevitable damage to the body (32). However, several studies reported that hypertrophic hearts are more vulnerable to ischemic–reperfusion injury, resulting in a larger infract area, higher peak cardiac troponin concentration and decreased LVEF (33-36). In addition, coronary microvascular dysfunction, which might present in some LVH patients, could also have an adverse effect on cardiomyocytes (37). Wever *et al.* reported that cardiac grafts with LVH from older donors contributed to a 6-fold increase in the risk of mortality after heart transplantation (38). Therefore, patients with LVH may be more susceptible to CPB-related injury due to their present cardiac abnormalities.

In conclusion, we conducted a retrospectively study with a relatively large sample to evaluate the impact of LVH in ATAAD patients who received surgical repair. We found that LVH was more prevalent in female patients. In addition, we confirmed the prognostic value of LVH/LVMI in predicting postoperative CMO and cardiac events for ATAAD patients. We also developed nomogram models for predicting postoperative CMO and cardiac events in ATAAD patients based on LVH or LVMI, that may help clinicians estimate prognosis in the early period after surgery. Future studies are required to investigate LVH's effects on long-term prognosis in ATAAD patients.

This study has several limitations. First, our conclusion may not be generalizable to other populations and regions due to its single-central retrospective nature. Second, the study was based on data from acute type A aortic dissection patients who underwent surgical repairs. Therefore, results may be different in other aortic dissection patients. Third, genetic evidence is required for the diagnosis of hypertrophic cardiomyopathy. Therefore, subgroup analysis was not applied for hypertrophic cardiomyopathy. Patients with hypertrophic cardiomyopathy may differ in outcomes. In addition, LVH contributes to an increased risk of heart failure, which might result in poorer prognosis. Lastly, our model lacked external validation, therefore it should be regarded as a preliminary tool.

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Footnote

Reporting Checklist: The authors have completed the

TRIPOD reporting checklist. Available at https://jtd.amegroups.com/article/view/10.21037/jtd-22-193/rc

Data Sharing Statement: Available at https://jtd.amegroups.com/article/view/10.21037/jtd-22-193/dss

Peer Review File: Available at https://jtd.amegroups.com/article/view/10.21037/jtd-22-193/prf

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://jtd.amegroups.com/article/view/10.21037/jtd-22-193/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). This study was approved by the ethics commission of Renmin Hospital of Wuhan University (WDRM2020-K230). Informed consent was not required due to its retrospective nature.

References

1. Hagan PG, Nienaber CA, Isselbacher EM, et al. The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. JAMA 2000;283:897-903.
2. Hiratzka LF, Bakris GL, Beckman JA, et al. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM Guidelines for the diagnosis and management of patients with thoracic aortic disease. A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, American Association for Thoracic Surgery, American College of Radiology,American Stroke Association, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of Thoracic Surgeons,and Society for Vascular Medicine. J Am Coll Cardiol 2010;55:e27-e129.
3. Nienaber CA, Clough RE. Management of acute aortic dissection. Lancet 2015;385:800-11.
4. Andersen ND, Ganapathi AM, Hanna JM, et al. Outcomes of acute type a dissection repair before and after implementation of a multidisciplinary thoracic aortic surgery program. J Am Coll Cardiol 2014;63:1796-803.
5. Zhu Y, Lingala B, Baiocchi M, et al. Type A Aortic Dissection-Experience Over 5 Decades: JACC Historical Breakthroughs in Perspective. J Am Coll Cardiol 2020;76:1703-13.
6. Malvindi PG, Modi A, Miskolczi S, et al. Acute type A aortic dissection repair in elderly patients. Eur J Cardiothorac Surg 2015;48:664-70; discussion 671.
7. Pape LA, Awais M, Woznicki EM, et al. Presentation, Diagnosis, and Outcomes of Acute Aortic Dissection: 17-Year Trends From the International Registry of Acute Aortic Dissection. J Am Coll Cardiol 2015;66:350-8.
8. Bossone E, Eagle KA. Epidemiology and management of aortic disease: aortic aneurysms and acute aortic syndromes. Nat Rev Cardiol 2021;18:331-48.
9. Seravalle G, Grassi G. Obesity and hypertension. Pharmacol Res 2017;122:1-7.
10. Arnett DK, Claas SA. Omics of Blood Pressure and Hypertension. Circ Res 2018;122:1409-19.
11. Manosroi W, Williams GH. Genetics of Human Primary Hypertension: Focus on Hormonal Mechanisms. Endocr Rev 2019;40:825-56.
12. He FJ, Tan M, Ma Y, et al. Salt Reduction to Prevent Hypertension and Cardiovascular Disease: JACC State-of-the-Art Review. J Am Coll Cardiol 2020;75:632-47.
13. Eirin A, Lerman A, Lerman LO. Mitochondrial injury and dysfunction in hypertension-induced cardiac damage. Eur Heart J 2014;35:3258-66.
14. Shlomai G, Grassi G, Grossman E, et al. Assessment of target organ damage in the evaluation and follow-up of hypertensive patients: where do we stand? J Clin Hypertens (Greenwich) 2013;15:742-7.
15. Yildiz M, Oktay AA, Stewart MH, et al. Left ventricular hypertrophy and hypertension. Prog Cardiovasc Dis 2020;63:10-21.
16. Wang SX, Xue H, Zou YB, et al. Prevalence and risk factors for left ventricular hypertrophy and left ventricular geometric abnormality in the patients with hypertension among Han Chinese. Chin Med J (Engl) 2012;125:21-6.

17. Iarussi D, Caruso A, Galderisi M, et al. Association of left ventricular hypertrophy and aortic dilation in patients with acute thoracic aortic dissection. Angiology 2001;52:447-55.
18. Taylor AP, Freeman RV, Bartek MA, et al. Left ventricular hypertrophy is a possible biomarker for early mortality after type B aortic dissection. J Vasc Surg 2019;69:1710-8.
19. Marwick TH, Gillebert TC, Aurigemma G, et al. Recommendations on the use of echocardiography in adult hypertension: a report from the European Association of Cardiovascular Imaging (EACVI) and the American Society of Echocardiography (ASE)†. Eur Heart J Cardiovasc Imaging 2015;16:577-605.
20. Augoustides JG, Geirsson A, Szeto WY, et al. Observational study of mortality risk stratification by ischemic presentation in patients with acute type A aortic dissection: the Penn classification. Nat Clin Pract Cardiovasc Med 2009;6:140-6.
21. Nienaber CA, Clough RE, Sakalihasan N, et al. Aortic dissection. Nat Rev Dis Primers 2016;2:16053.
22. Messerli FH, Williams B, Ritz E. Essential hypertension. Lancet 2007;370:591-603.
23. Oktay AA, Lavie CJ, Milani RV, et al. Current Perspectives on Left Ventricular Geometry in Systemic Hypertension. Prog Cardiovasc Dis 2016;59:235-46.
24. Haider AW, Larson MG, Benjamin EJ, et al. Increased left ventricular mass and hypertrophy are associated with increased risk for sudden death. J Am Coll Cardiol 1998;32:1454-9.
25. Rocha WEM, Oliveira MFRA, Soares JD, et al. Left Ventricular Concentric Geometric Patterns Are Associated With Worse Prognosis Among Patients With Type-A Aortic Dissection. J Am Heart Assoc 2021;10:e018273.
26. Wu J, Xie E, Qiu J, et al. Subacute/chronic type A aortic dissection: a retrospective cohort study. Eur J Cardiothorac Surg 2020;57:388-96.
27. Weiner MM, Reich DL, Lin HM, et al. Influence of increased left ventricular myocardial mass on early and late mortality after cardiac surgery. Br J Anaesth 2013;110:41-6.
28. Zhu P, Dai Y, Qiu J, et al. Prognostic implications of left ventricular geometry in coronary artery bypass grafting patients. Quant Imaging Med Surg 2020;10:2274-84.
29. Rozenbaum Z, Finkelstein A, Zhitomirsky S, et al. Impact of preprocedural left ventricle hypertrophy and geometrical patterns on mortality following TAVR. Am Heart J 2020;220:184-91.
30. Korhonen PE, Kautiainen H, Järvenpää S, et al. Target organ damage and cardiovascular risk factors among subjects with previously undiagnosed hypertension. Eur J Prev Cardiol 2014;21:980-8.
31. Apostolakis EE, Baikoussis NG, Parissis H, et al. Left ventricular diastolic dysfunction of the cardiac surgery patient; a point of view for the cardiac surgeon and cardio-anesthesiologist. J Cardiothorac Surg 2009;4:67.
32. De Hert S, Moerman A. Myocardial injury and protection related to cardiopulmonary bypass. Best Pract Res Clin Anaesthesiol 2015;29:137-49.
33. Stiermaier T, Pöss J, Eitel C, et al. Impact of left ventricular hypertrophy on myocardial injury in patients with ST-segment elevation myocardial infarction. Clin Res Cardiol 2018;107:1013-20.
34. Fernández-Jiménez R, Silva J, Martínez-Martínez S, et al. Impact of left ventricular hypertrophy on troponin release during acute myocardial infarction: new insights from a comprehensive translational study. J Am Heart Assoc 2015;4:e001218.
35. Mølgaard S, Faricelli B, Salomonsson M, et al. Increased myocardial vulnerability to ischemia-reperfusion injury in the presence of left ventricular hypertrophy. J Hypertens 2016;34:513-23; discussion 523.
36. Nepper-Christensen L, Lønborg J, Ahtarovski KA, et al. Left Ventricular Hypertrophy Is Associated With Increased Infarct Size and Decreased Myocardial Salvage in Patients With ST-Segment Elevation Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention. J Am Heart Assoc 2017;6:004823.
37. Camici PG, Tschöpe C, Di Carli MF, et al. Coronary microvascular dysfunction in hypertrophy and heart failure. Cardiovasc Res 2020;116:806-16.
38. Wever Pinzon O, Stoddard G, Drakos SG, et al. Impact of donor left ventricular hypertrophy on survival after heart transplant. Am J Transplant 2011;11:2755-61.

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Supplementary

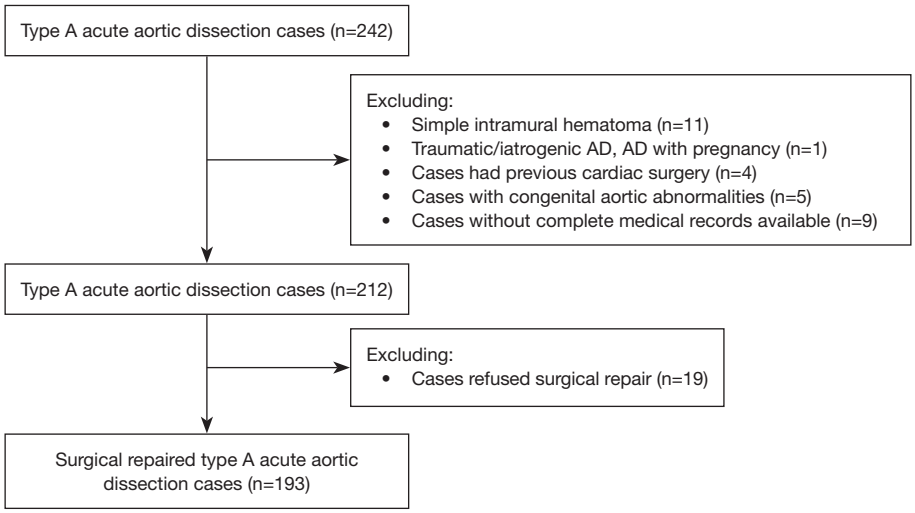

Figure S1 Flowchart of this study.

Table S1 Multivariable logistic regression for operation duration in CMO

Operation duration	β	S.E.	Wald	OR	95% CI	P value
Circulatory arrest duration (per 5 min)	0.081	0.103	0.619	1.085	0.886–1.328	0.431
Aortic cross-clamping duration (per 10 min)	–0.133	0.100	1.778	0.875	0.719–1.065	0.182
Cardiopulmonary bypass duration (per 10 min)	0.168	0.062	7.372	1.183	1.048–1.336	0.007*
Intercept	–5.951	1.550	14.75	–	–	–

*, P value <0.05. CMO, composite major outcomes; S.E., standard error; OR, odds ratio; CI, confidence interval.

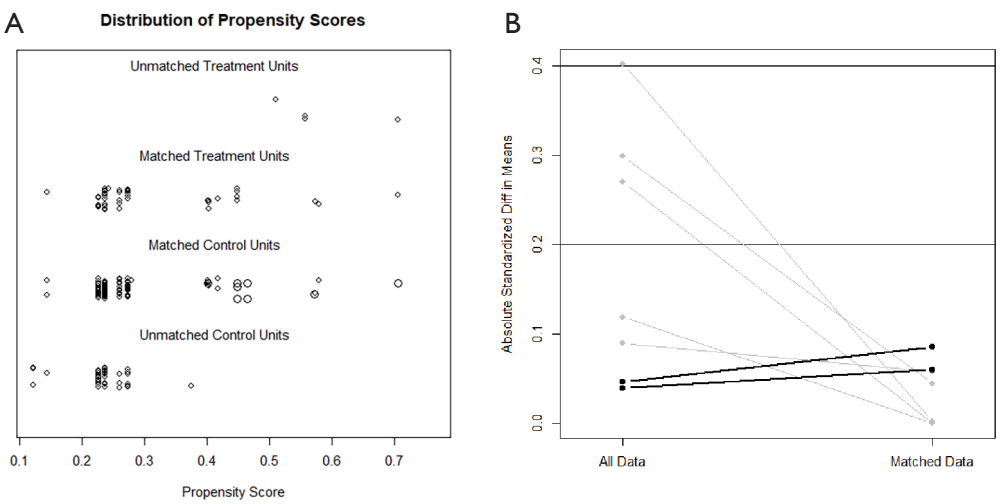

Figure S2 Jitter plot and line plot of individual cases before and after propensity score matching. (A) Jitter plot of individual cases. (B) line plot of individual cases.

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Table S2 Two models for CMO

Model information	β	S.E.	Wald	OR	95% CI	P value
Model LVH						
Left ventricular hypertrophy	1.045	0.439	5.680	2.844	1.204–6.719	0.017*
Penn Ab	0.779	0.458	2.900	2.180	0.889–5.346	0.089
Penn Ac/Ab&c	2.742	0.776	12.49	15.51	3.391–70.94	<0.001*
Hyperlipidemia	1.102	0.436	6.375	3.010	1.280–7.081	0.012*
Emergency surgical repair	1.005	0.435	5.334	2.732	1.164–6.409	0.021*
CPB durations (per 10 min)	0.113	0.040	8.158	1.120	1.036–1.211	0.004*
Intercept	–6.592	1.270	26.94	–	–	–
Model LVMI						
LVMI (per 10 $g \cdot m^{-2}$)	0.161	0.064	6.440	1.175	1.037–1.331	0.011*
Penn Ab	0.806	0.460	3.069	2.240	0.909–5.522	0.080
Penn Ac/Ab&c	2.524	0.804	9.857	12.48	2.581–60.32	0.002*
Hyperlipidemia	1.103	0.439	6.308	3.013	1.274–7.124	0.012*
Emergency surgical repair	1.096	0.442	6.138	2.992	1.257–7.120	0.013*
CPB durations (per 10 min)	0.116	0.040	8.465	1.123	1.039–1.215	0.004*
Intercept	–8.091	1.484	29.74	–	–	–

*, P value <0.05. CMO, composite major outcomes; S.E., standard error; OR, odds ratio; CI, confidence interval; LVH, left ventricular hypertrophy; LVMI, left ventricular mass index; CPB, cardiopulmonary bypass.

Table S3 Collinearity analysis of model LVH and model LVMI for postoperative CMO

Model information	Tolerance	VIF
Model LVH		
LVH	0.996	1.004
Penn classification	0.988	1.013
Hyperlipidemia	0.955	1.047
Emergency surgical repair	0.962	1.040
CPB durations (per 10 min)	0.970	1.031
Model LVMI		
LVMI (per 10 $g \cdot m^{-2}$)	0.981	1.019
Penn classification	0.971	1.030
Hyperlipidemia	0.955	1.048
Emergency surgical repair	0.964	1.038
CPB durations (per 10 min)	0.971	1.030

LVH, left ventricular hypertrophy; LVMI, left ventricular mass index; CMO, composite major outcomes; VIF, variance inflation factor; CPB, cardiopulmonary bypass.

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Table S4 Two models for postoperative cardiac event

Model information	β	S.E.	Wald	OR	95% CI	P value
Model LVH						
Left ventricular hypertrophy	1.193	0.603	3.920	3.297	1.012–10.74	0.048*
CPB durations (per 10min)	0.125	0.046	7.499	1.134	1.036–1.240	0.006*
Intercept	–6.732	1.485	20.56	–	–	–
Model LVMI						
LVMI (per 10 $g \cdot m^{-2}$)	0.188	0.061	9.559	1.207	1.071–1.360	0.002*
CPB durations (per 10min)	0.133	0.047	8.169	1.143	1.043–1.252	0.004*
Intercept	–8.660	1.745	24.63	–	–	–

*, P value <0.05. S.E., standard error; OR, odds ratio; CI, confidence interval; LVH, left ventricular hypertrophy; LVMI, left ventricular mass index; CPB, cardiopulmonary bypass.

Table S5 Collinearity analysis of model LVH and model LVMI for postoperative cardiac event.

Model information	Tolerance	VIF
Model LVH		
LVH	0.999	1.001
CPB durations (per 10min)	0.999	1.001
Model LVMI		
LVMI (per 10 $g \cdot m^{-2}$)	0.999	1.001
CPB durations (per 10min)	0.999	1.001

VIF, variance inflation factor; LVH, left ventricular hypertrophy; CPB, cardiopulmonary bypass; LVMI, left ventricular mass index.

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