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Original Article

The risk factors for postoperative acute respiratory distress syndrome in Stanford type a acute aortic dissection patients

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Abstract: Objective: To explore the risk factors for postoperative acute respiratory distress syndrome (ARDS) in Stanford type A acute aortic dissection (AAD) patients. Methods: This study included 64 patients with Stanford type A AAD who underwent Sun's procedure. The patients were divided into an ARDS group (PaO_2/FiO_2 < 200 mmHg) and a non-ARDS group (PaO_2/FiO_2 ≥ 200 mmHg). We compared the patients' perioperative clinical features in the two groups. A multivariate binary logistic regression was used to analyze the risk factors for ARDS in the Stanford type A AAD patients. Results: The incidence of postoperative ARDS was 56.25%. There were 13 deaths in the 6-month follow-up, including 8 in the ARDS group and 5 in the non-ARDS group. There were differences in the body mass index (BMI) levels, the times from onset to operation, the preoperative white blood cell counts, the preoperative hemoglobin levels, the preoperative alanine aminotransferase levels, the preoperative blood creatinine levels, the preoperative PaO_2/FiO_2, intraoperative blood transfusion volumes, the assisted mechanical ventilation times, and the durations of the intensive care unit stays between the two groups (all $P < 0.05$). The logistic regression analysis revealed that a BMI ≥ 25 kg/m^2, a time from onset to operation ≥ 24 hours, a preoperative white blood cell count ≥ 10×10^9/L, and a preoperative PaO_2/FiO_2 < 300 mmHg were the independent risk factors for postoperative ARDS in patients with Stanford type A AAD. Conclusions: ARDS occurs often in Stanford type A AAD patients. A BMI ≥ 25 kg/m^2, a time from onset to operation ≥ 24 hours, a preoperative white blood cell count ≥ 10×10^9/L, and a preoperative PaO_2/FiO_2 < 300 mmHg are the independent risk factors for postoperative ARDS in these patients.

Keywords: Stanford A acute aortic dissection, Sun's procedure, postoperative acute respiratory distress syndrome

Introduction

Acute aortic dissection (AAD) is an extremely dangerous aortic disease, and it has an extremely high early mortality. Getting surgery as soon as possible is the only effective treatment for AAD [1]. The Stanford system is popular for the classification of aortic dissection, of which Stanford A type accounts for more than 60% [2, 3]. Early surgical repair helps improve the prognosis and reduce the mortality [4]. With the continuous improvement of perioperative treatment surgical techniques, especially the promotion and application of Sun's procedure, the aortic dissection-related surgery success rate has been greatly improved, but the postoperative death rate still remains at a high level [5]. It has been reported that the mortality rate following Stanford type A AAD reaches 9%-30% [6]. Acute respiratory distress syndrome (ARDS) is a common complication after AAD, especially after Sun's procedure [7]. ARDS significantly increases the postoperative mortality, which accounts for 30%-55% of the overall mortality [8]. At the same time, AAD also seriously affects the prognosis, prolongs the ventilator use and postoperative ICU retention times, and may induce multiple organ failure [9].

At present, the causes and mechanisms of ARDS in Stanford type A AAD patients after Sun's procedure are unclear, the risk factors and related mechanisms that may lead to the development of ARDS are also unclear, and there are few theoretical studies on this topic [10].

Therefore, it is necessary to explore the related risk factors of ARDS in Stanford type A AAD patients. This study aimed to examine the risk factors for ARDS in Stanford A type AAD patients after surgery, and to provide further guidance for their clinical treatment.

Materials and methods

Clinical data

The ethics committees of our hospital approved this retrospective study. Sixty-four patients who underwent Sun's procedure for Stanford type A acute aortic dissection and who were admitted to our hospital from January 2017 to January 2019 were included in this study. The general patient data included age, gender, body mass index, hypertension, diabetes, hyperlipidemia, smoking, alcohol drinking, and aortic-related surgery. Their preoperative data consisted of the routine preoperative laboratory examination data (blood routine, liver function indexes, renal function indexes), combined with other operations or not, operation times, extracorporeal circulation times, deep hypothermia circulatory arrest times, minimum rectal temperatures, and intraoperative blood transfusions. The postoperative data included the 1 hour postoperative hemoglobin, the 1 hour postoperative albumin, the assisted mechanical ventilation times, the durations of the intensive care unit stays, the use of continuous renal replacement therapy, the use of extra-corporeal membrane oxygenation, and the total lengths of the hospital stays. The postoperative mortality included the 30-day postoperative deaths and the total deaths after 6 months of follow-up.

Inclusion criteria: (1) Patients with complete medical records. (2) After admission, patients who received a chest imaging examination, echocardiography examination, aortic enhanced CT, and aortic CT angiography to confirm their diagnosis and to classify their aortic dissection. (3) Patients who underwent the surgery. (4) Patients whose time from onset to surgery was less than 14 days.

Exclusion criteria: (1) Patients with preoperative hemodynamic instability, patients with left ventricular ejection fractions < 40%, and patients with a left ventricular end-diastolic diameter > 701 mm. (2) Patients also suffering from underlying chronic respiratory diseases, severe infections, severe liver or kidney insufficiencies, heart failure, or massive pleural or pericardial effusion. (3) Pregnant women.

According to the Berlin Conference for the diagnostic criteria of acute respiratory distress syndrome in 2010 and existing research, the PaO_2/FiO_2 < 200 mmHg level was used as the diagnostic criteria for ARDS in this study [11, 12]. At the same time, to avoid the interference of other factors such as transportation, the PaO_2/FiO_2 value from 6 h to 12 h after transferring the patient to the intensive care unit after operation was measured. Using this level as a reference value, the 64 patients were divided into the ARDS group (n=36) and the non-ARDS group (n=28).

Methods

The axillary vein, the axillary artery, and other related blood vessels were separated and prepared for backup. Median sternotomy was carried out. The left innominate artery, left cervical artery, and right subclavian artery were separated. Heparinization and cooling was carried out, and a cardiopulmonary bypass was established. The ascending aorta was blocked; aortic root surgery was performed. The cardiopulmonary bypass was ceased during the cooling. The three branches of the aortic arch blood vessels were blocked. The left subclavian artery and aortic arch were transected. A stent that can open up blood vessels with plaque blockages was installed to restore the thoracic aortic blood flow perfusion, which matched the total artery of the left neck and the branch of artificial blood vessels. The perfusion of the brain was resumed. The aorta proximal and artificial blood vessels proximal to the heart were sewed after the normal temperature returned. Exhaustion, perfusion, defibrillation, and cardiac resuscitation were performed. Finally, the left subclavian artery, left innominate artery, and artificial vascular branches were sewed up. The classic Sun's procedure was completed [9]. After the surgery, according to each patient's condition, platelets and fresh plasma were infused and a temporary pacemaker was placed. The chest was sewed up, and then the patient was transferred to the intensive care unit. Vital sign changes were closely monitored. Routine fasting, rehydration, maintenance of the electrolyte balance, double

lower limb braking at 24 hours after the surgery, prevention of the arterial puncture seepage, the use of anti-inflammatory analgesic drugs and small doses of glucocorticoids for the postoperative fever patients were administered. The blood pressure was closely monitored. The intravenous micro-pumping of the vasoactive drugs (e.g. sodium nitrate) were adjusted according to the blood pressure. The blood pressure control target value was 120-140/60-80 mmHg. For patients who had ARDS, a pulmonary protective ventilation treatment strategy was used as soon as they were diagnosed. The target tidal volume was 6.4±0.5 mL/kg, and the SIMV pressure control (PB840, Puritan Bennett, USA) mode was used. If ventilation with the inspiratory pressure > 20 cmH_2O still failed to achieve the target tidal volume, the ventilation mode was switched to the SIMV volume control mode. The ventilation frequency was 12 times/min, the inhalation time was set at 1.4 s, the FiO_2 was 60%-80%, and the peak inspiratory pressure was 35 cmH_2O. PEEP was set to (8±2) cmH_2O. A lung maneuver was performed once every 6 h. Before the sputum suction, pure oxygen was given for 2 min. After the sputum suction, lung inflation was performed for 5-10 min, with a frequency of 20 times/min, a flow of 15 L/min, and a PEEP of 10 cmH_2O. The static lung compliance was checked using the gas inhalation/breath-holding method once the lung maneuver was completed. Other comprehensive therapies were adopted, including cooling, maintaining the rectal temperature around 36.5°C, maintaining the hemoglobin above 90 g/L, and pumping dopamine to increase the myocardial contractility and increase the cardiac output.

Outcome measures

The independent risk factors for ARDS in Stanford type A AAD patients after Sun's procedure were the main outcome measures. The total incidence, postoperative complications, 30-day postoperative deaths and total deaths after 6 months of follow-up were the secondary outcome measures.

Statistical analysis

SPSS 26.0 software was used for the statistical analyses. The measurement data were expressed as the mean ± standard deviation ($\bar{x}$ ± sd) and were compared using t-tests. Chi-square tests were used to analyze the count data, which was expressed as n (%). The occurrence of postoperative ARDS in the Stanford type A AAD patients was regarded as a dependent variable (0= ARDS, 1= non-ARDS). The indicators of the differences between the two groups (BMI, times from onset to operation, preoperative white blood cell counts, preoperative hemoglobin levels, preoperative alanine aminotransferase levels, preoperative blood creatinine levels, preoperative PaO_2/FiO_2 levels, intraoperative blood transfusion volumes, assisted mechanical ventilation times, and the durations of the intensive care unit stays) were considered to be the independent variables, which were assigned values to examine independent risk factors for postoperative ARDS using the binary Logistics regression analysis model. Statistical significance was defined as $P < 0.05$.

Results

General clinical data

There were no significant differences among the patients in terms of age, gender, smoking, alcohol drinking, hypertension, diabetes, hypercholesterolemia, cardiovascular disease, or history of aortic surgery (all P>0.05). The ARDS group had a higher body mass index ($P < 0.05$), and significantly lower PaO_2/FiO_2 and SaO_2 at 6-12 hours after surgery (both $P < 0.05$) than the non-ARDS group. See **Table 1**.

Preoperative data

No differences were found between the two groups regarding their rates of time from onset to operation more than 24 hours, the rates of time from onset to operation more than 7 days, their preoperative platelet counts, or their preoperative D-dimer concentrations (all P>0.05). The ARDS group had longer times from onset to operation, higher preoperative white blood cell counts and glutamic-pyruvic transaminase levels, and higher serum creatinine concentrations (all $P < 0.05$), but lower preoperative hemoglobin and PaO_2/FiO_2 levels than the non-ARDS group (all $P < 0.05$). See **Table 2**.

Intraoperative data

The ARDS group had higher intraoperative blood transfusion volumes than the non-ARDS

Table 1. General patient clinical data

Category of general clinical data	ARDS group (n=36)	Non-ARDS group (n=28)	χ^2/t	P
PaO_2/FiO_2 at 6-12 hours after surgery (mmHg)	181.54±17.18	242.56±21.74	12.551	< 0.001
SaO_2 at 6-12 hours after surgery (%)	89.43±2.84	97.28±2.78	2.148	0.036
Age (years)	52.7±8.3	50.6±10.2	0.901	0.371
Gender (Male/Female)	25/11	16/12	1.035	0.309
Body mass index (kg/m^2)	25.67±3.86	22.93±4.01	1.079	0.007
Smoking	9	6	0.112	0.738
Alcohol drinking	10	11	0.946	0.331
Hypertension	28	26	2.717	0.099
Diabetes	18	15	0.082	0.777
Hypercholesterolemia	20	19	1.001	0.317
Cardiovascular disease	10	6	0.339	0.561
History of aortic surgery	3	4	0.573	0.449

Note: ARDS: acute respiratory distress syndrome.

Table 2. Preoperative data

Category of preoperative data	ARDS group (n=36)	Non-ARDS group (n=28)	χ^2/t	P
Time from onset to operation (h)	68.56±10.56	48.56±10.38	7.572	< 0.001
Time from onset to operation >24 h	29	25	0.911	0.340
Time from onset to operation >7 d	4	5	0.593	0.441
White blood cell count (10^9/L)	12.12±3.43	9.84±3.27	1.129	< 0.001
Hemoglobin (g/L)	128.22±21.38	138.78±18.54	2.075	0.042
Blood platelet count (10^{12}/L)	164.28±55.74	170.45±50.13	0.459	0.648
Alanine aminotransferase (U/L)	38.21±10.85	32.65±9.77	2.123	0.038
Blood creatinine (μmol/L)	85.38±14.23	77.62±12.78	2.264	0.027
D-dimer (mg/L)	2.65±1.08	2.35±1.12	1.085	0.282
PaO_2/FiO_2 (mmHg)	157.98±98.33	239.42±107.62	3.154	0.003

Note: ARDS: acute respiratory distress syndrome.

group (P < 0.05). There were no significant differences in the combinations with other operations, the extracorporeal circulation times, the deep hypothermic circulatory arrest times, the operation times, or the minimum rectal temperatures between the two groups (all P>0.05). See **Table 3**.

Postoperative data

There were no differences in terms of the 1-hour postoperative hemoglobin level, 1-hour postoperative albumin level, the use of continuous renal replacement therapy, the use of extracorporeal membrane oxygenation, or the total lengths of the hospital stays in the two groups (all P>0.05). The ARDS group had longer assisted mechanical ventilation times and intensive care unit stays than the non-ARDS group (both P < 0.05), See **Table 4**.

Postoperative complications

No differences were found in the postoperative gastrointestinal hemorrhage, acute renal failure, multiple organ dysfunction syndrome, secondary intubation, re-thoracotomy to stop bleeding, 30-day postoperative death, or 6-month death rates (all P>0.05). See **Table 5**.

The postoperative ARDS influencing factors in Stanford type A AAD patients

The occurrence of postoperative ARDS in Stanford type A AAD patients was regarded as a dependent variable (0= ARDS, 1= non-ARDS). The indicators of the differences between the two groups (BMI, times from onset to operation, preoperative white blood cell counts, preoperative hemoglobin levels, preoperative alanine aminotransferase levels, preoperative bl-

Table 3. Intraoperative data

Category of intraoperative data	ARDS group (n=36)	Non-ARDS group (n=28)	χ^2/t	P
Complication			0.350	0.554
Bentall operation	9	6		
Wheat operation	1	1		
David operation	2	1		
Coronary artery bypass grafting	2	2		
Valvuloplasty	1	1		
Extracorporeal circulation time (min)	211.86±65.34	192.55±67.73	1.154	0.253
Deep hypothermia circulatory arrest time (min)	119.89±35.67	114.98±38.66	0.527	0.601
Operation time (h)	8.54±1.52	8.61±1.46	0.186	0.853
Minimum rectal temperature (°C)	25.3±1.3	25.2±1.2	0.316	0.753
Intraoperative blood transfusion (mL)	3654.75±1269.54	2998.57±1064.98	2.198	0.032

Note: ARDS: acute respiratory distress syndrome.

Table 4. Postoperative data

Category of postoperative data	ARDS group (n=36)	Non-ARDS group (n=28)	χ^2/t	P
1-hour postoperative hemoglobin (g/L)	89.12±27.58	101.54±28.98	1.748	0.085
1-hour postoperative albumin (g/L)	27.77±2.73	28.24±3.12	0.642	0.523
Assisted mechanical ventilation time (min)	2135.45±1988.64	1355.69±987.23	6.443	< 0.001
Intensive care unit stay (d)	9.6±4.6	5.2±1.9	12.613	< 0.001
Use of continuous renal replacement therapy	4	2	0.292	0.589
Use of extracorporeal membrane oxygenation	5	3	0.201	0.654
Total length of hospital stays (d)	24.6±9.9	23.6±9.6	0.386	0.701

Note: ARDS: acute respiratory distress syndrome.

Table 5. Postoperative complications

Category of postoperative complications	ARDS group (n=36)	Non-ARDS group (n=28)	χ^2/t	P
Gastrointestinal hemorrhage	4	4	0.145	0.703
Acute renal failure	7	8	0.731	0.393
Multiple organ dysfunction syndrome	2	1	0.139	0.709
Secondary intubation	4	2	0.292	0.589
Re-thoracotomy to stop bleeding	3	4	0.573	0.449
30-day postoperative death	4	2	0.292	0.589
Total deaths after 6 months of follow-up	8	5	0.185	0.667

Note: ARDS: acute respiratory distress syndrome.

ood creatinine levels, preoperative PaO_2/FiO_2 levels, intraoperative blood transfusions, assisted mechanical ventilation times, and the durations of the intensive care unit stays) were considered independent variables to investigate the independent risk factors for postoperative ARDS using the binary logistic regression analysis. The outcome showed that a BMI ≥ 25 kg/m^2, a time from onset to operation ≥ 24 hours, a preoperative white blood cell count ≥ 10×10^9/L, and a preoperative PaO_2/FiO_2 < 300 mmHg were the independent risk factors for postoperative ARDS. See **Tables 6** and **7**.

Discussion

Postoperative ARDS is a common complication in cardiopulmonary bypass surgery, with an incidence rate of about 12.2%-27.1% generally. Its incidence is higher in patients after ADD, and the proportion of deaths has reached more than half [8]. In this study, the incidence of postoperative ARDS was 56.25%, and the total mortality at 6 months after surgery was 23.44%, of which the ARDS group accounted for 61.54%, which is consistent with the results published in current research [8]. The current

Table 6. Independent variables and assignment for the influencing factors of postoperative ARDS in Stanford type A AAD patients

Factors	Independent variable	Assignment
Body mass index (kg/m^2)	X1	< 25=0, ≥ 25=1
Time from onset to operation (h)	X2	< 24=0, ≥ 24=1
Preoperative white blood cell count (10^9/L)	X3	< 10=0, ≥ 10=1
Preoperative hemoglobin (g/L)	X4	Male/Female < 120/110=0, ≥ 120/110=1
Preoperative alanine aminotransferase (U/L)	X5	< 40=0, ≥ 40=1
Preoperative blood creatinine (umol/L)	X6	< 90=0, ≥ 90=1
Preoperative PaO_2/FiO_2 (mmHg)	X7	< 300=0, ≥ 300=1
Intraoperative blood transfusion (mL)	X8	< 2500=0, ≥ 2500=1
Assisted mechanical ventilation time (min)	X9	< 2400=0, ≥ 2400=1
Intensive care unit stay (d)	X10	< 6=0, ≥ 6=1

Note: ARDS: acute respiratory distress syndrome; AAD: acute aortic dissection.

Table 7. The outcomes of the postoperative ARDS influencing factors in Stanford type A AAD patients

Factors	B	Wald	OR	95% CI	P
Body mass index ≥ 25	0.243	15.725	1.465	1.145-1.387	< 0.001
Time from onset to operation ≥ 24 h	0.156	1.316	1.185	1.085-1.276	0.027
Preoperative white blood cell count ≥ 10 (10^9/L)	1.674	4.236	5.747	1.135-18.785	0.032
Preoperative hemoglobin < 120/110 (g/L)	0.009	0.003	1.001	0.999-1.005	0.947
Preoperative alanine aminotransferase ≥ 40 (U/L)	0.044	0.268	1.002	0.997-1.008	0.658
Preoperative blood creatinine ≥ 90 (umol/L)	0.082	0.341	1.039	0.899-1.057	0.158
Preoperative PaO_2/FiO_2 (< 300 mmHg)	1.965	6.203	8.765	1.164-27.118	0.014
Intraoperative blood transfusion ≥ 2,500 (mL)	0.494	0.445	1.096	1.027-1.236	0.058
Assisted mechanical ventilation time ≥ 2,400 (min)	0.301	0.557	1.317	1.087-1.174	0.082
Intensive care unit stay ≥ 6 d	0.178	0.874	1.451	1.113-1.194	0.064

Note: ARDS: acute respiratory distress syndrome; AAD: acute aortic dissection.

research on the risk factors for ARDS in Stanford type A AAD patients mainly includes obesity, the times from onset to operation, excessive blood transfusions, infections, etc. [13, 14]. Obesity has been shown to be an independent risk factor for postoperative ARDS, which is not limited to aortic surgery. An increased body mass index can lead to a significant increase in the incidence of ARDS before and after Stanford type A AAD [3, 15]. Obese patients have fat infiltration. With increased airway resistance, greater respiratory resistance occurs, which in turn consumes more energy and causes oxidative stress, the release of chemokines and vasoactive substances, and cell membrane damage [16, 17]. In this study, according to our binary logistic regression, a BMI ≥ 25 kg/m^2 is an independent risk factor for ARDS in patients with Stanford type A AAD, which is consistent with the results reported in recent studies [14]. When Stanford A type AAD occurs, the intima of the artery is torn, and a large amount of the extracellular matrix of the vessel wall is exposed to the blood circulation, which itself causes the release of a large number of inflammatory factors into the blood, causing pulmonary capillary bed destruction and pulmonary interstitial edema, inducing acute respiratory distress [18]. Studies have shown that a decrease in the PaO_2/FiO_2 levels before surgery in patients with aortic dissection may be related to the systemic inflammatory response [19]. Aortic dissection can cause endarteritis, and the cascade of inflammation will increase the permeability of the endothelia and epithelia, which directly affects the pulmonary blood vessel pressure, thereby reducing the function of the alveolar surfactants, leading to increased pulmonary blood pressure, impairing lung oxygen and eventually leading to ARDS. It has been reported that the preoperative white blood cell count of the ARDS group after

surgery was significantly higher than that of the non-ARDS group [7]. In this study, the preoperative white blood cell count ≥ 10×10^9/L was considered to be an independent risk factor for ARDS in patients with Stanford A AAD, which is consistent with the results of current research [7]. Studies have pointed out that as the time after the onset of AAD increases, the length of the aortic tear will continue to increase. With the increasing volume of the false lumen, the blood flow in the true lumen continues to decrease, and the oxygenation index decreases as well. The decrease in blood flow in the involved arteries leads to ischemia and hypoxia in related tissues and organs, which eventually jointly lead to the development of ARDS [20, 21]. In this study, it was observed that a time from onset to operation >24 hours was an independent risk factor for ARDS in Stanford type A AAD patients, and the preoperative times from onset to operation in the non-ARDS group were significantly lower than they were in the ARDS group, suggesting that timely surgery after the onset of disease may reduce the incidence of ARDS, which is in line with the findings of current research [22]. It has been reported that patients with a preoperative $PaO_2/FiO_2 \leq 300$ mmHg are more likely to develop ARDS after surgery [23]. This may due to a pre-operative lung injury, or the patients also may have experienced some surgical trauma, a cardiopulmonary bypass, or a deep hypothermia arrest, so more inflammatory factors were released, and a large number of neutrophils aggregated and were activated in the lung tissue and the accumulation of alveolar fluid increased [24]. In this study, it was confirmed that preoperative $PaO_2/FiO_2 < 300$ mmHg is an independent risk factor for ARDS in Stanford type A AAD patients, which is also consistent with the results reported in current research. Studies have shown that an excessive blood transfusion volume during aortic dissection is harmful to the body and increases the incidence of postoperative ARDS [12]. Because a large number of cell debris and exogenous proteins in the stored blood enter the body, it may directly affect the patient's lung function and induce ARDS. In this study, the intraoperative blood transfusion volumes in the ARDS group were significantly higher than the volumes in the non-ARDS group, which proves that excessive blood transfusion volumes during Stanford type A AAD may be likely to cause ARDS after surgery.

A retrospective analysis found that the preoperative alanine transaminase and blood creatinine levels were different between the groups, but we ultimately did not prove that these two levels were independent risk factors. The reason for this difference may be due to the systemic inflammatory reaction or an internal environment disorder when important organs were damaged [13]. In this study, the preoperative alanine transaminase and blood creatinine levels of the two groups were also different. According to some earlier studies, the prolonged intraoperative extracorporeal circulation time and the extended hypothermia circulatory arrest time may be independent risk factors [21]. In this study, there was no difference between the intraoperative extracorporeal circulation time and the deep hypothermic circulatory arrest time in the two groups. This may be due to the fact that the patients included in this study all underwent Sun's procedure and all the aortic arch lesions were treated instead of just the ascending aorta surgery alone. It has been pointed out that smoking is an independent risk factor [25]. However, there are also studies that have found that age and smoking status are not independent risk factors [26]. In this study, we found no difference in age or smoking status between the two groups, but further research is needed on this controversial topic.

However, this study was limited by the small sample size, which was not accurate enough for the outcome measures (such as the failure to further observe the indicators of the patients with mild, moderate, and severe ARDS), a short follow-up time, etc. The final result may have a certain degree of bias, and the sample size ought to be expanded in future research.

In summary, a BMI ≥ 25 kg/m^2, a time from onset to operation ≥ 24 hours, a preoperative white blood cell count ≥ 10×10^9/L, and a preoperative $PaO_2/FiO_2 < 300$ mmHg are the independent risk factors for postoperative ARDS in Stanford A type AAD patients. These outcomes instruct us to be fully aware that the Stanford A type AAD patients with obesity and infection complications, with a time from onset to the start of surgery more than 24 hours, with preoperative infections, and with decreased oxygenation index levels are more likely to develop postoperative ARDS. These outcomes have a certain guiding significance in clinical practice.

Disclosure of conflict of interest

None.

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