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Risk factor analysis of postoperative acute respiratory distress syndrome after type A aortic dissection repair surgery

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Abstract

To investigate the incidence, outcomes, and risk factors of postoperative acute respiratory distress syndrome (ARDS) in patients undergoing surgical repair for acute type A aortic dissection.

This retrospective study involved 270 patients who underwent surgical repair for acute type A aortic dissection between January 2009 and December 2015. Data on clinical characteristics and outcomes were collected. Patients who immediately died after surgery and with preoperative myocardial dysfunction were excluded. The included patients were divided into the ARDS (ARDS patients who met the Berlin definition) and non-ARDS groups. Primary outcome was postoperative ARDS, according to the 2012 Berlin definition for ARDS and was reviewed by 2 qualified physicians with expertise in critical care and cardiac surgery. Outcomes of interest were the incidence and severity of risk factors for ARDS in this population, and perioperative outcomes and survival rates were compared with patients with or without ARDS.

A total of 233 adult patients were enrolled into this study; of these, 37 patients (15.9%) had ARDS. Three, 20, and 14 patients had mild, moderate, and severe ARDS, respectively, according to the Berlin definition, with no significant difference in age, sex, and underlying disease. The ARDS group had lower mean oxygenation index (OI) than the non-ARDS group in the first 3 days post-surgery and demonstrated an improvement in lung function after the fourth day. Postoperative complication risks were higher in the ARDS group than in the non-ARDS group. However, no significant difference was observed in in-hospital mortality between the 2 groups (10.8% vs 5.6%, $P = .268$). Additionally, there was also no significant difference in the 3-year mortality rate between the 2 groups (P of log-rank test = .274). Postoperative hemoglobin level (odds ratio [OR]: 0.78; 95% confidence interval [CI]: 0.62–0.99) and perioperative blood transfusion volume (OR: 1.07; 95% CI: 1.03–1.12) were associated with ARDS risk.

Postoperative ARDS after type A aortic dissection repair surgery was associated with risks of postoperative complications but not with risk of in-hospital mortality or 3-year mortality. A higher perioperative blood transfusion volume and a lower postoperative hemoglobin level may be risk factors for ARDS.

Abbreviations: AKI = acute kidney injury, ARDS = acute respiratory distress syndrome, BMI = body mass index, BT = blood transfusion, CABG = coronary artery bypass grafting, CI = confidence interval, COPD = chronic obstructive pulmonary disease, CPB = cardiopulmonary bypass, CXR = chest x-ray, ECMO = extracorporeal membrane oxygenation, ESRD = end-stage renal disease, FFP = fresh frozen plasma, FiO_2 = fraction of inspired oxygen, H/D = hemodialysis, IABP = intra-aortic balloon pump, ICU = intensive care unit, LIPS = lung injury prediction score, OI = oxygen index, PaO_2 = partial pressure of oxygen in the arterial blood, PLT = platelet, PRBC = packed red blood cell, SBP = systolic blood pressure, SLIP = surgical lung injury prediction, SLIP-2 = surgical lung injury prediction-2, SOFA = sequential organ failure assessment, TACO = transfusion-associated circulatory overload, WBC = white blood count.

Keywords: aortic dissection, acute respiratory distress syndrome, type A dissection

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1. Introduction

Acute respiratory distress syndrome (ARDS) is a known critical complication that includes increased pulmonary vascular permeability and loss of functional lung tissue. ARDS leads to high patient mortality. In the United States, ARDS affects approximately 200,000 patients per year, causing nearly 75,000 deaths.[1] It is also associated with higher mortality compare with other common acute illness, such as acute kidney injury (AKI) and stroke.[2,3] A recent large observational study enrolling 50 intensive care unit (ICU) patients from 5 continents reported an ARDS prevalence of 10.4%. ICU and in-hospital mortality rates of ARDS were 35.3% and 40.0%, respectively.[4] ARDS is under-recognized and undertreated. The management of ARDS patients can be greatly improved.

Acute type A aortic dissection is an emergent situation with high mortality and morbidity rates and requires prompt surgical repair. Aortic and cardiac surgeries are major risk factors for postoperative lung injury, which occur in as many as 20% of patients undergoing cardiac surgery.[5] Different procedures may lead to different levels of risk-inducing ARDS; valve surgery contributes to 8.1% of the incidence of ARDS,[6] whereas aortic surgery contributes to 16% of the incidence.[7] The risk is higher when the operation is emergent.[8] The mortality rate of patients undergoing emergent surgery who developed ARDS can even be as high as 80%, which is significantly higher than that of the general population with ARDS.[9,10] In the different score-assessing models for lung injury, such as lung injury prediction score (LIPS), surgical lung injury prediction (SLIP), and surgical lung injury prediction-2 (SLIP-2),[5,9,11] aortic surgery contributes greatly to the scoring. However, only a few studies have discussed ARDS after aortic dissection surgery; therefore, we aimed to investigate the incidence, outcomes, and risk factors of postoperative ARDS in patients undergoing emergent acute type A aortic dissection repair surgery.

2. Methods

2.1. Study participants and design

The 2012 Berlin definition defines ARDS as acute-onset hypoxemia associated with bilateral opacities, as observed on chest fluoroscopy, and not induced by pleural effusion, atelectasis, nodules, heart failure, or fluid overload.[12] This prospective data collection and retrospective analysis study were approved by the institutional review board of our hospital (Approval No: 201601407B0). Individual consent was not required. We reviewed the medical records of 270 consecutive patients who were diagnosed with acute Stanford classification type A aortic dissection and who received emergent repair surgery in a tertiary referral hospital between January 2009 and December 2015. The included patients were divided into the ARDS group (n = 37, ARDS patients who met the Berlin definition) and the non-ARDS group (n = 196) (Fig. 1).

2.2. Data collection and definition

Data on clinical characteristics, demographic characteristics, and preoperative ejection fraction were collected. ARDS was defined according to the 2012 Berlin definition. The inclusion criteria

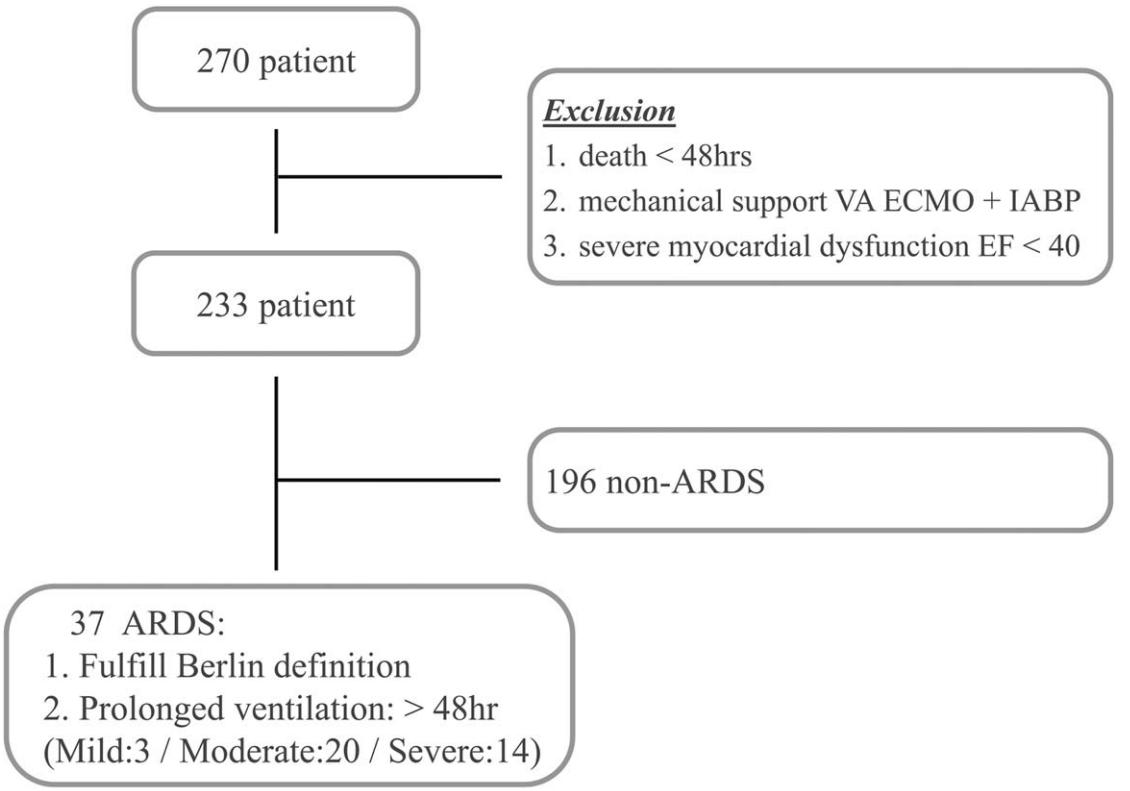

Figure 1. Flowchart of patient screening and enrolment. ARDS = acute respiratory distress syndrome, ECMO = extracorporeal membrane oxygenation, EF = ejection fraction, IABP = intra-aortic balloon pump, VA = venoarterial.

Table 1

Surgical data.

Variable	All patients (N = 233)	Non-ARDS (n = 196)	ARDS (n = 37)	*P*-value
Surgical method, n (%)				
Aortic root replacement (Bentall)	29 (12.5)	23 (11.8)	6 (16.2)	.425
Aortic valve replacement	3 (1.3)	3 (1.5)	0 (0.0)	1.000
Aortic arch	59 (25.3)	49 (25.0)	10 (27.0)	.837
Additional surgery, n (%)				
CABG	14 (6.0)	11 (5.6)	3 (8.1)	.473
Redo sternotomy	10 (4.3)	6 (3.1)	4 (10.8)	.057
Intraoperative data, min				
Bypass time	257.5 ± 65.4	251.4 ± 61.8	289.9 ± 74.6	.001
Clamp time	168.9 ± 50.5	165.9 ± 49.7	185.1 ± 52.5	.034
Arrest time	51.7 ± 24.3	50.7 ± 24.0	57.1 ± 25.7	.143
Lowest BT during operation	19.1 ± 1.7	19.1 ± 1.8	19.0 ± 1.6	.798
Brain protection (antegrade), n (%)	218 (94.4)	184 (94.8)	34 (91.9)	.443

Data are presented as mean ± standard deviation or frequency (%).

ARDS = acute respiratory distress syndrome, BT = blood transfusion, CABG = coronary artery bypass grafting.

for the ARDS patients were as follows: (1) PaO_2/FIO_2 levels of 200 mm Hg $< PaO_2/FIO_2 \leq$ 300 mm Hg, 100 mm Hg $< PaO_2/FIO_2 \leq$ 200 mm Hg, and $PaO_2/FIO_2 \leq$ 100 mm Hg. Based on these levels, ARDS patients were classified into 3 subgroups: mild, moderate, and severe subgroups, respectively; acute onset within 7 days after surgery with ventilation setting for positive end-expiratory pressure (PEEP) of $\geq$5 cmH_2O; and bilateral lung infiltration, detected through chest x-ray, cannot be fully explained by effusion, lobar, lung collapse, or nodules. Fluoroscopic findings were reviewed by 2 qualified doctors, and in cases of disagreement of the diagnosis, a third physician was consulted.

We excluded patients who immediately died after surgery (<48 hours after surgery) because we could not monitor the primary outcomes. Moreover, to correctly appraise ARDS according to the Berlin definition, we also excluded patients with hydrostatic lung edema (e.g., cardiogenic pulmonary edema or fluid overload), perioperative heart failure patients with transesophageal echocardiography ejection fraction of <40%, and those on perioperative mechanical support (intraaortic balloon pump [IABP] and extracorporeal membrane oxygenation [ECMO]).

2.3. Surgical technique and postoperative care

Once dissection was confirmed through computed tomography and by a cardiac surgeon, emergent surgery was arranged within 30 minutes. Intraoperative transesophageal echocardiography was conducted to monitor cardiac function. Cardiopulmonary bypass (CPB) was established in the usual manner, and most of the patients received antegrade brain protection. We maintained the core temperature of patients at approximately 24 to 26 °C. Myocardial protection was achieved using histidine-tryptophan-ketoglutarate solution (Custodiol; Essential Pharmaceuticals, LLC, Newtown, PA) to maintain cardiac arrest, and this process was repeated every 90 to 120 minutes. Operative details are described in Table 1.

All patients were transferred to the ICU for postoperative care. Once a patient presented with acute lung injury, the current management for ADRS was performed, such as total sedation, low tidal volume (4–8 mL/kg predicted body weight), and lower inspiratory pressure (plateau pressure <30 cm H_2O). For each patient, chest x-ray (CXR) was conducted every day for routine follow-up. Arterial blood gas data were obtained regularly to monitor oxygenation in patients. We did not adopt the prone position for our postoperative patients during the study period.

2.4. Statistical analysis

We compared the patient data (demographics, surgical details, and perioperative blood transfusion volume) between the ARDS and non-ARDS groups using Student *t* test for continuous variables or Fisher exact test for categorical variables. Variables with *P* values <.2 in the univariate analyses were entered into a multivariable logistic model with backward elimination. The 3-year survival curves of the ARDS and non-ARDS groups were depicted using Kaplan–Meier estimates, and a log-rank test was used to compare between-group differences. All tests were 2-tailed, and $P < .05$ was considered statistically significant. Data were analyzed using SPSS 22.0 software (IBM SPSS, Armonk, NY: IBM Corp).

3. Results

3.1. Study population characteristics and surgical details

A total of 233 adult patients were enrolled into this study; of these, 37 patients (15.9%) had ARDS. Baseline patients' characteristics are shown in Table 2. No significant differences were observed in age, sex, underlying diseases, and essential laboratory data, such as serum creatinine level, preoperative hemoglobin level, white blood cell count, and preoperative ejection fraction, between the ARDS and non-ARDS groups (Table 2).

3.2. Surgical data: ARDS versus non-ARDS groups

No significant difference was observed in the distribution of surgical methods and additional surgeries between the ARDS and non-ARDS groups. The ARDS group had longer CPB time ($P = .001$) and clamp time ($P = .034$) than the non-ARDS group. However, no group difference was observed in the arrest time (Table 1).

Table 2

Preoperative demographic data.

Variable	All patients (N = 233)	Non-ARDS (n = 196)	ARDS (n = 37)	*P*-value
Age, y	54.9 ± 13.7	54.3 ± 14.0	57.9 ± 11.8	.140
Sex, female (%)	68 (29.2)	58 (29.6)	10 (27.0)	.845
Body weight, kg	73.3 ± 16.2	72.9 ± 16.2	75.2 ± 16.3	.422
Body height, cm	167.3 ± 9.8	167.2 ± 10.0	168.2 ± 8.7	.611
Body mass index, kg/m^2	26.0 ± 5.0	25.7 ± 4.8	27.5 ± 5.5	.080
BMI group, n (%)				.376
BMI >30 kg/m^2	61 (26.2)	52 (26.5)	9 (24.3)	
BMI ≤30 kg/m^2	144 (61.8)	123 (62.8)	21 (56.8)	
Unknown	28 (12.0)	21 (10.7)	7 (18.9)	
Diabetes mellitus, n (%)	12 (5.2)	10 (5.1)	2 (5.4)	1.000
Hypertension, n (%)	166 (71.6)	140 (71.8)	26 (70.3)	.844
Old stroke, n (%)	16 (6.9)	12 (6.2)	4 (10.8)	.295
Ejective fraction (%)	60.6 ± 10.8	60.8 ± 10.7	59.9 ± 11.3	.681
Smoking, n (%)	103 (44.4)	87 (44.6)	16 (43.2)	1.000
COPD, n (%)	4 (1.7)	4 (2.1)	0 (0.0)	1.000
ESRD under H/D, n (%)	6 (2.6)	5 (2.6)	1 (2.7)	1.000
Liver cirrhosis, n (%)	3 (1.3)	2 (1.0)	1 (2.7)	.408
Hemopericardium, n (%)	71 (30.6)	59 (30.3)	12 (32.4)	.846
Shock (SBP <90 mmHg)	22 (9.4)	16 (8.2)	6 (16.2)	.131
Laboratory data				
Creatinine, mg/dL	1.4 ± 1.3	1.4 ± 1.1	1.8 ± 2.1	.086
WBC (×10^3/μL)	13.1 ± 4.7	12.9 ± 4.5	14.0 ± 5.5	.179
Hemoglobin, g/dL	13.5 ± 2.1	13.5 ± 2.1	13.6 ± 2.3	.814
PLT (×10^3/μL)	178.4 ± 56.8	181.4 ± 56.4	162.2 ± 57.2	.059

Data are presented as mean ± standard deviation or frequency (%).

ARDS = acute respiratory distress syndrome, BMI = body mass index, COPD = chronic obstructive pulmonary disease, ESRD = end-stage renal disease, H/D = hemodialysis, PLT = platelet, SBP = systolic blood pressure, WBC = white blood count.

3.3. Postoperative tube drainage volume, perioperative blood transfusion volume, and outcomes: ARDS versus non-ARDS groups

The ARDS group had a larger postoperative drainage volume ($P < .001$) than the non-ARDS group, and a difference was observed in the perioperative blood transfusion volume between the 2 groups. The ARDS group received a larger volume of packed red blood cells and frozen fresh plasma ($P < .001$) than the non-ARDS group (Table 3).

A total of 15 in-hospital deaths were recorded (6.4%), and a nonsignificant difference was observed between the ARDS and non-ARDS groups (10.8% vs 5.6%, $P = .268$). Patients in the ARDS group were more likely to stay in the ICU for >7 days and to be hospitalized for >28 days ($P < .01$). The incidence of postoperative shock and transient ischemic attack was higher in the ARDS group ($P < .05$) than in the non-ARDS group. By contrast, a nonsignificant difference was observed in postoperative ventilation duration, new-onset hemodialysis, tracheostomy, and wound infection between the 2 groups (Table 4).

3.4. Oxygenation index (OI; PaO_2/FiO_2)

The ARDS group had a lower mean OI in the first 3 days after surgery than the non-ARDS group. Moreover, the ARDS group demonstrated an improvement in lung function after the fourth day and even outperformed the non-ARDS group on the sixth and seventh days. By contrast, the non-ARDS group showed stable OI levels, which were between 220 and 260 (Fig. 2).

Table 3

Perioperative drainage volume, hemogram, and blood transfusion volume.

Variable	All patients (N = 233)	Non-ARDS (n = 196)	ARDS (n = 37)	*P*-value
Drainage volume (24 h)	1148 ± 1266	1002 ± 1125	1912 ± 1654	<.001
Laboratory data				
Hemoglobin, g/dL	10.7 ± 1.6	10.9 ± 1.5	9.9 ± 1.9	.001
PLT (×10^3/μL)	136.2 ± 40.2	138.8 ± 41.4	122.3 ± 30.0	.022
Transfusion 0–48 h				
PRBC (U)	6.7 ± 4.5	6.1 ± 4.1	9.6 ± 5.0	<.001
FFP (U)	6.0 ± 4.2	5.4 ± 3.8	9.1 ± 5.0	<.001
PLT (U)	13.1 ± 8.8	12.0 ± 8.2	18.5 ± 9.6	<.001
PRBC + FFP (U)	12.7 ± 8.5	11.5 ± 7.8	18.7 ± 9.7	<.001

Data are presented as mean ± standard deviation or frequency (%).

ARDS = acute respiratory distress syndrome, FFP = fresh frozen plasma, PLT = platelet, PRBC = packed red blood cell.

Table 4

Postoperative data.

Variable	All patients (N = 233)	Non-ARDS (n = 196)	ARDS (n = 37)	*P*-value
In-hospital mortality, n (%)	15 (6.4)	11 (5.6)	4 (10.8)	.268
ICU stay, d	7.9 ± 16.4	7.3 ± 17.1	11.2 ± 11.9	.176
ICU >7 days, n (%)	61 (26.2)	42 (21.4)	19 (51.4)	<.001
Hospital stay, d	29.2 ± 39.9	27.8 ± 41.6	36.9 ± 28.6	.203
Hospital stay >28 days, n (%)	67 (28.8)	48 (24.5)	19 (51.4)	.002
Ventilator time, h	131 ± 413	109 ± 391	245 ± 506	.068
Postoperative shock (SBP <90 mm Hg)	19 (8.2)	11 (5.6)	8 (21.6)	.004
Unexpected check bleeding, n (%)	6 (2.6)	4 (2.0)	2 (5.4)	.243
Transient ischemic attack, n (%)	40 (17.2)	29 (14.8)	11 (29.7)	.034
New H/D (%)	20 (8.6)	14 (7.1)	6 (16.2)	.102
Tracheostomy, n (%)	6 (2.6)	5 (2.6)	1 (2.7)	1.000
Wound infection, n (%)	5 (2.2)	4 (2.1)	1 (2.7)	.584
SOFA score	6.3 ± 2.9	6.1 ± 2.9	7.5 ± 2.6	.009

Data are presented as mean ± standard deviation or frequency (%).

ARDS = acute respiratory distress syndrome, H/D = hemodialysis, ICU = intensive care unit, SBP = systolic blood pressure, SOFA = Sequential Organ Failure Assessment.

3.5. Logistic regression of postoperative ARDS incidence according to perioperative variables

Univariate analyses (Tables 1, 3, 4) revealed that the following variables were significantly correlated with ARDS risk: CPB time, clamp time, postoperative drainage volume, perioperative blood transfusion volume, and postoperative hemoglobin and platelet levels. After including these significant variables as well as variables with *P* values of <.1 in the multivariable logistic model, higher postoperative hemoglobin level (odds ratio [OR]: 0.78, 95% confidence interval [CI]: 0.62–0.99, *P* = .041) was associated with lower ARDS risk. In contrast, greater perioperative blood transfusion volume (OR: 1.07, 95% CI: 1.03–1.12, *P* < .001) was associated with higher ARDS risk (Table 5).

3.6. Survival curve analysis during follow-up

In addition, our data revealed a nonsignificant difference in 3-year mortality between the ARDS and non-ARDS groups (*P* of log-rank test = .274) (Fig. 3).

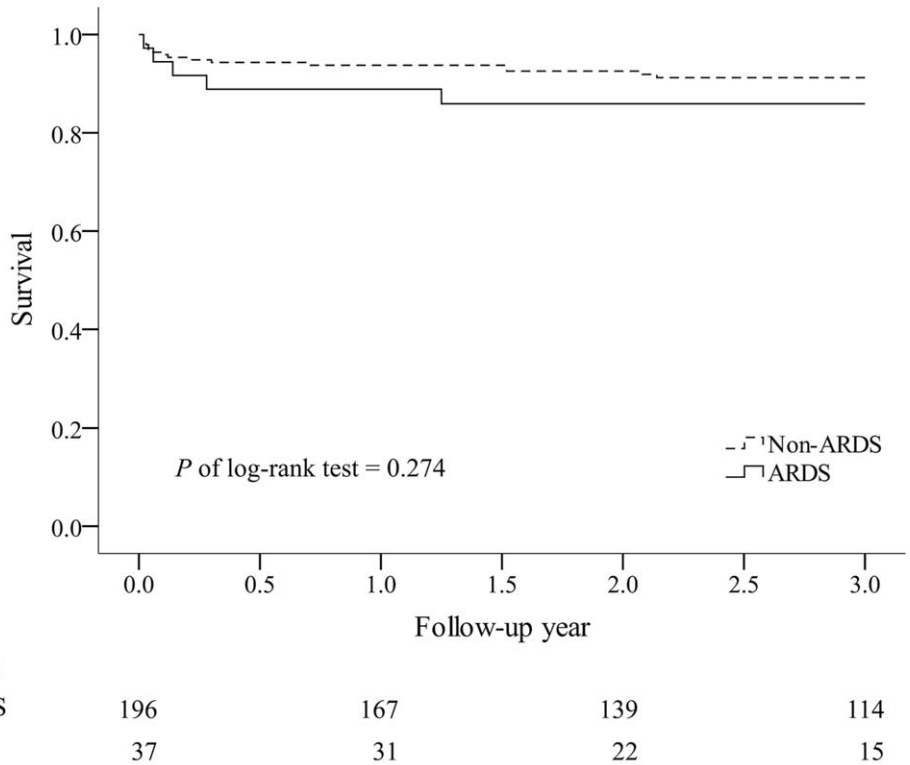

Figure 2. Oxygenation index after surgery of the ARDS and non-ARDS groups. ARDS = acute respiratory distress syndrome, FiO_2 = fraction of inspired oxygen, OP = operation, PaO_2 = partial pressure of oxygen in the arterial blood.

Table 5

Multivariable analysis for associated factors of ARDS.

Variable	Odds ratio	95% CI	*P*-value
Postoperative hemoglobin level, g/dL	0.78	0.62–0.99	.041
Perioperative blood transfusion volume 0–48 h: PRBC + FFP(U)	1.07	1.03–1.12	<.001

ARDS = acute respiratory distress syndrome, CI = confidence interval, FFP = fresh frozen plasma, PRBC = packed red blood cell.

4. Discussion

In this study, we compared the data of ARDS (37/233) and non-ARDS (196/233) patients undergoing acute type A dissection repair surgery. This is the first study to evaluate postoperative ARDS according to the Berlin definition in this patient cohort. The principal findings of the current study are as follows: the incidence rate of ARDS in this population was 15.8%, with a mortality rate approaching 10.8%, and the recovery of lung function usually occurred 4 days after surgery; the ARDS group had a higher postoperative complication rate and longer ICU and hospital stay, but the longer ICU and hospital stay had no significant effect on survival; multivariate analysis identified higher perioperative blood transfusion volume and lower postoperative hemoglobin level as independent risk factors for postoperative ARDS; and ARDS was not associated with postoperative survival.

ARDS is a leading cause of respiratory failure after surgical intervention, with a mortality rate of approximately 40% in the general population; mortality even reaches up to 80% in patients undergoing cardiac surgery.[13] Cardiac surgery is considered a risk factor for ARDS, and different types of cardiac surgeries contribute to different levels of risk. Gajic et al[7] have demonstrated an ARDS risk of 10% after cardiac surgery. However, our study revealed a mortality rate of only 10.8%. In our study, we selected ARDS patients according to the Berlin definition; therefore, some patients were excluded, such as those with immediate mortality, myocardial dysfunction, and signs of malperfusion who represent most type A dissection patients with a high mortality risk. This might explain why the mortality rate in this study is lower than that of other studies.

The Berlin definition of ARDS is an updated version of the 1994 American–European Consensus Conference definition.[12] The new definition has improved predictive validity for mortality and corresponding severity.[11] In addition, using the Berlin definition, cases can be detected earlier, and more suitable treatment options based on severity can be provided.[5] However, only a few studies have applied the Berlin definition to patients undergoing surgery and have investigated the risk factors for ARDS after aortic surgery. In our study, the ARDS group had higher postoperative complications as well as longer ICU and hospital stay, without any significant effect on survival, than the non-ARDS group. Moreover, the recovery of lung function usually occurred 4 days after surgery in the ARDS group.

A previous study has reported multiple possible factors causing the onset of ARDS in patients undergoing cardiac surgery. Exposure of blood to abnormal surfaces and conditions during CPB may induce a systemic inflammatory response that contributes to ARDS.[14] The increased release of C3a and C5a[15] and the combined administration of heparin and protamine at the end of CPB also contribute to inflammatory reactions after the procedure.[16] However, our study revealed no significant association between CPB time and arrest time and ARDS, which requires further investigations for clarification.

During type A aortic dissection repair surgery, blood transfusion is essential due to profound coagulopathy and the nature of aortic surgery. The development of transfusion-related acute lung injury (TRALI) may be not only a challenging circumstance but also a major complication during clinical practice. In TRALI, hypoxia and bilateral lung infiltration occur within 6 hours after transfusion, with a mortality rate of 5% to 25%.[5] The cause of TRALI is multifactorial.[17–19] Plasma-containing blood products full of leukocyte antibodies and

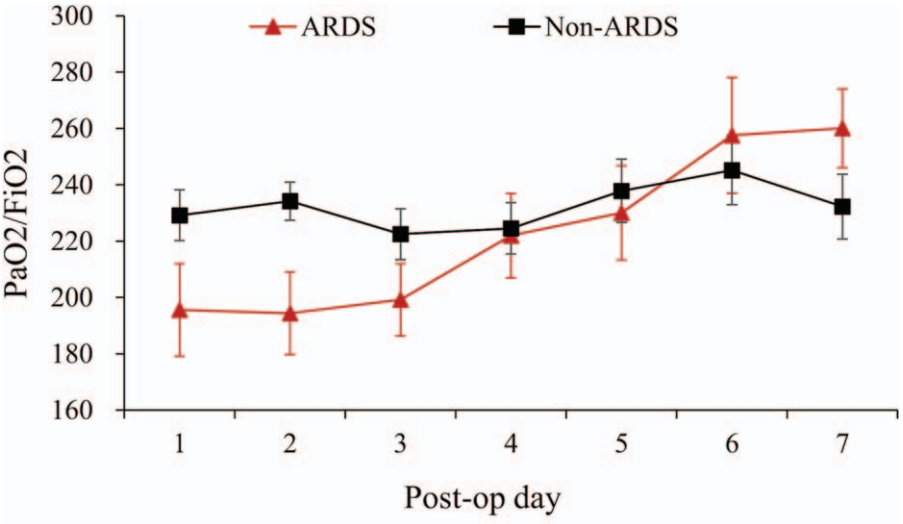

Figure 3. Three-year mortality rates of the ARDS and non-ARDS groups. ARDS = acute respiratory distress syndrome.

biologically active substances, such as lipids and cytokines, will increase pulmonary microvascular permeability, which may lead to lung damage and capillary leakage.[20] Chen et al's study involving 527 patients who had undergonetype A dissection repair surgery showed that perioperative transfusion volume was an independent risk factor for postoperative ARDS.[21] A targeted preventative strategy may decrease the development of ARDS, if risk factors can be identified early. Meticulous hemostasis and improvement of surgical technique for decreasing perioperative blood product transfusion may reduce the incidence of postoperative ARDS and improve postoperative outcomes in this population.

Transfusion-associated circulatory overload (TACO) is another situation that causes pulmonary edema. TACO induces hydrostatic pulmonary edema, unlike TRALI, which increases pulmonary vascular permeability. Apart from symptoms noted in lung edema, such as dyspnea and jugular vein engorgement, elevated systolic blood pressure is also usually present. Patients with TACO usually show improvement after body fluid reduction with diuresis. In this study, we tried to distinguish TACO from TRALI by monitoring the central venous pressure, performing 2D echo examination, and maintaining a balanced fluid status. However, it is still difficult to distinguish one from another.[22,23]

4.1. Limitation

This study has some limitations. First, our study is limited by its retrospective nature and inherent limitations. Second, similar to other clinical studies of ARDS, the reproducibility of the diagnosis is limited in this study. Reproducibility may be biased when studying postoperative ARDS after CPB and cardiac arrest, because the occurrence of both cardiogenic pulmonary edema and ARDS in these patients is not uncommon. Furthermore, it is difficult to clearly differentiate TRALI and TACO; hence, a well-designed prospective study investigating this issue is warranted. Finally, this is a single-center study, which limits the external generalizability of the current findings to the entire population. Therefore, a multicenter study involving a larger patient number is required to provide more definite results.

5. Conclusion

Postoperative ARDS, according to the Berlin definition, in patients undergoing surgical repair of type A aortic dissection was associated with higher postoperative complication rate, without any effect on mid-term survival. Lung function usually improved at 4 days post-surgery. Higher perioperative blood transfusion volume and lower postoperative hemoglobin level were identified as independent risk factors for postoperative ARDS. Meticulous hemostasis and improvement of surgical technique for decreasing perioperative blood product transfusion may reduce the incidence of postoperative ARDS and improve postoperative outcomes in this population.

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