Impact of Presurgical Mild Acute Respiratory Distress Syndrome on Surgical Mortality After Surgical Repair of Acute Type A Aortic Dissection

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Summary

To evaluate the impact of presurgical mild acute respiratory distress syndrome (ARDS) on surgical mortality in patients undergoing surgical repair of acute type A aortic dissection by means of deep hypothermic circulatory arrest (DHCA) in a single-center, retrospective study.

From January 2011 to December 2015, 333 eligible patients were divided into either a mild-ARDS group (n = 136) or a no-ARDS group (n = 197). The definition of mild ARDS referred to the recent revision of ARDS definition (Berlin criteria). The surgical mortality and major postoperative morbidity were investigated and analyzed.

A total of 136 patients developed presurgical mild ARDS, with an incidence of 40.8%. No significant difference emerged between the 2 groups in major postoperative morbidity except for pulmonary complications. Multivariate logistic regression displayed that the risk of postoperative pulmonary complications in patients with presurgical mild ARDS was 4.25 times that in patients without presurgical ARDS (OR = 4.25, 95% CI 2.05-7.72). Twenty-four patients died after surgery, with significantly higher surgical mortality in the mild-ARDS group compared with the no-ARDS group (12.5% versus 3.6%, P = 0.002). Kaplan-Meier curves showed a poor surgical survival in the mild-ARDS group (χ2=12.958, Log-Rank P < 0.001). And Cox regression revealed the hazard ratio for surgical mortality in the mild-ARDS group compared with the no-ARDS group was 2.52 (95%CI 1.41-5.32, P = 0.016).

Presurgical mild ARDS increased postoperative respiratory morbidity, and then increased surgical mortality after surgical repair of acute type A aortic dissection by means of DHCA (Int Heart J Advance Publication)

Key words: Mild acute respiratory distress syndrome, Acute type A aortic dissection, Deep hypothermic circulatory arrest, Surgical mortality, Morbidity

Acute lung injury, which was used to describe mild acute respiratory distress syndrome (ARDS) previously, is one of most common presurgical complications following acute aortic dissection, with a prevalence of 30-45%.

Although it is recognized that activation of the inflammatory system caused by aortic injury due to acute aortic dissection may play a crucial role in the occurrence and development of acute lung injury, the specific mechanism is still unclear. Acute lung injury or even severe ARDS is considered to be systemic complications of acute aortic dissection rather than a simple pulmonary disease.

An overwhelming majority of previous studies focused on evaluating risk factors for presurgical mild ARDS following acute aortic dissection, identifying the predictors for hypoxemia following surgical repair of acute type A aortic dissection by means of deep hypothermic circulatory arrest (DHCA), and exploring the pathogenesis of postoperative hypoxemia. However, few reports focused on evaluating the impacts of presurgical mild ARDS on surgical mortality and major postoperative morbidity in patients undergoing surgical repair of acute type A aortic dissection by means of DHCA. We hypothesized that patients with presurgical mild ARDS, in a state of stronger systemic inflammatory response, undergoing surgical repair of acute type A aortic dissection by means of DHCA, may aggravate lung injury and then cause more negative impacts on postoperative morbidity and mortality.

Based on the above analysis, through reviewing 333 eligible patients undergoing surgical repair of acute type A aortic dissection by means of DHCA within two weeks after the onset of symptoms, this single-center study...
aimed to evaluate the impacts of presurgical mild ARDS on surgical mortality and major postoperative morbidity.

Methods

Inclusion criteria: Patients undergoing surgical repair of acute type A aortic dissection by means of DHCA within two weeks after the onset of symptoms without meeting exclusion criteria were eligible for this study. Surgical repair of acute type A aortic dissection included extended hemiarch or total arch replacement, which was defined as the procedure requiring circumferential anastomosis at the level of aortic arch or the descending aorta with the use of techniques of brain protection (including DHCA or selective antegrade cerebral perfusion-SACP), and/or debranching of at least one supra-aortic vessel, with concomitant procedures.\(^6\) Exclusion criteria included an echocardiographically estimated left ventricular ejection fraction of 35% or less, preoperative concurrent congestive heart failure, prior cardiac surgery, a positive history of chronic obstructive pulmonary disease, chronic renal failure, advanced liver disease, malignant disease, infectious disease, and other inflammatory diseases that required therapy with steroids or nonsteroidal anti-inflammatory drugs. Patients with presurgical moderate or severe ARDS were also excluded from this study.

Research protocols: This is a single-center retrospective study performed in accordance with the Declaration of Helsinki on medical research in human patients. Data collection was performed by trained staff (two people), and they did not know the purpose of this study.

The diagnosis of acute aortic dissection was made on the basis of the detection of two aortic lumens with blood flow or thrombus by contrast-enhanced computed tomography. Computed tomography scans were performed using slices that were 5 mm in thickness from the apex of the lung to the common femoral artery. The extent of dissection was determined according to Stanford classification: the dissecting process of type A involved the ascending aorta and/or aortic arch, and possibly the descending aorta. The tear can originate in the ascending aorta, the aortic arch, or, more rarely, in the descending aorta.\(^6\)

No term of acute lung injury is used in the recent revision of ARDS definition (Berlin criteria). In the Berlin definition, ARDS was classified as mild, moderate, and severe according to the oxygenation index.\(^9\) In this study, mild ARDS was used instead of acute lung injury. According to the Berlin definition, patients meeting all of the following criteria were considered as patients suffering from mild ARDS: acute onset within one week; 200 mmHg < PpaO2/FiO2 ≤ 300 mmHg with positive end-expiratory pressure or continuous positive airway pressure ≥ 5 cmH2O; chest radiograph or computed tomography scan showing bilateral opacities (not fully explained by effusions, lobar/lung collapse, or nodules); and respiratory failure not fully explained by cardiac failure or fluid overload.\(^10\) Oxygenation index of moderate and severe ARDS was 101 to 200, and less than or equal to 100 mmHg, respectively. The value of oxygenation index was calculated as the ratio of arterial partial pressure of oxygen (PaO2) to the fraction of inspired oxygen (FiO2), PaO2/FiO2. In patients who were mechanically ventilated, the FiO2 setting of the respirator was adopted as the FiO2 value. In patients who were breathing spontaneously with nasal prongs, a face mask or a non-rebreathing mask, the estimated FiO2 was adopted as the FiO2 value.\(^11\) Accordingly, all included patients were divided into either a mild-ARDS group (patients suffered from presurgical mild ARDS) or a no-ARDS group (patients did not suffer from presurgical ARDS).

The primary end point was surgical mortality, which was defined as death occurring during the hospitalization in which the operation was performed, even if after 30 days; or death occurring after discharge from the hospital, but within 30 days of the procedure, unless the cause of death was clearly unrelated to the operation. The secondary endpoint was the occurrence of major postoperative morbidity (occurred during the same hospitalization or within 30 days of surgery), including low cardiac output syndrome (LCOS), pulmonary complications, stroke, hepatic failure, renal failure, re-operation for bleeding and deep sternal wound infection. Postoperative LCOS were considered with those who met the following criteria before discharge from first hospitalization in the intensive care unit immediately after surgery: 1. need for mechanical circulatory support with intra-aortic balloon pump to maintain systolic blood pressure greater than 90 mmHg after correction of electrolytes and blood gas abnormalities while adjusting preload volume to its optimal values; 2. signs of impairment of body perfusion after correction of electrolytes and blood gas abnormalities while adjusting preload volume to its optimal values.\(^12\) Postoperative pulmonary complications included pneumonia (a positive result in a sputum culture requiring anti-infective treatment, or chest X-ray diagnosis of pneumonia following cardiac surgery) and respiratory failure, which was defined as the duration of mechanical ventilation more than 48 hours or re-intubation following surgery.\(^13\) Postoperative stroke was defined as a new local or global cerebral dysfunction lasting over 24 hours. Hepatic failure was defined as a total bilirubin level of more than 3.0 mg/dL and a 3-fold rise in glutamate-pyruvate transaminase in the absence of myocardial or muscular damage. Renal failure was defined as a creatinine level > 3.0 mg/dL or anuria requiring renal replacement therapy. In addition, the following data were also recorded: demographic data, preoperative peak C-reactive protein, intraoperative parameters (including duration of cardiopulmonary bypass, aortic cross-clamping, DHCA and SACP, lowest nasopharyngeal temperature, and the number of blood units transfused), and other postoperative variables (including oxygenation index at postsurgical 6 hours, duration of mechanical ventilation, and length of ICU stay).

There were standard protocols for extubation and leaving ICU. Criteria for extubation\(^16\) included an alert and hemodynamically stable patient with no excessive bleeding, ability of the patient to breathe through a T tube for at least 30 minutes with a fraction of inspired oxygen of less than 0.40 and a respiratory rate less than 25 breaths/minute, an arterial blood PO2 greater than 70 mmHg, a PCO2 less than 40 mmHg and a pH greater than 7.35, with no metabolic acidosis. Other criteria for extuba-
tation were a tidal volume of 6 mL/kg, a peak negative inspiratory pressure of less than -20 cmH₂O and a mandatory chest radiograph before extubation to rule out pneumothorax, pleural effusion, and atelectasis. Criteria for leaving ICU included alert, hemodynamically stable patients with an inotropic score less than 5, good pulmonary function with no metabolic acidosis, urine output over 0.5 mL/kg/hour and a mandatory chest radiograph before leaving ICU to rule out pneumothorax, pleural effusion, and atelectasis.

**Surgical procedure:** Anesthesia was induced with moderate doses of fentanyl, midazolam, and a long-acting muscle relaxant and was maintained with isoflurane or sevoflurane in 100% oxygen and supplemental intravenous opioids. The nasopharyngeal temperature was monitored. The pressure in both radial artery and dorsalis pedis artery was monitored.

Cardiopulmonary bypass was conducted with femoral and axillary arteries cannulation and the right atrium cannulation, initially using the flow index within the range of 2.4-2.8 L/minute/m² (maintaining mean arterial pressure on the level of approximately 60-70 mmHg), decreasing it gradually with the reduction of body temperature according to the index 0.1 L/minute/m²/1°C. Myocardial protection was obtained with antegrade and retrograde infusion of cold cardioplegic solution. Depending on the operator's decision regarding the method of brain protection, the patient was cooled down to 18-20°C if only DHCA was applied, while with the use of SACP, the patient was cooled down to 23-25°C. After the distal anastomosis was completed, a cardiopulmonary bypass was restarted. Rewarming was conducted with the 10°C gradient to 34°C. Cerebral protection was achieved by DHCA or SACP with perfusion of innominate artery and, if necessary, the left common carotid artery and was monitored with the use of visualization of cerebral tissue regional oxygenation based on the module of Near-Infrared Spectroscopy. The volumetric flow rate of cerebral perfusate was maintained at the level of 10 mL/kg/minute. Head ice packing was performed to further improve cerebral protection. Pharmacological neuroprotection (methylprednisolonum, MgSO₄, mannitol, etc.) was administered during cardiopulmonary bypass.

All of the patients were operated on through a median sternotomy. The surgical techniques included extended to hemiarch and total arch replacement with concomitant procedures. Hemiarch involved resection of the smaller curvature of the aortic arch and open anastomosis by means of DHCA/SACP. The total arch procedure consisted of a transverse aortic arch replacement with vascular prosthesis either with island technique or debranching of at least one supra-aortic vessel, or a combination of these methods. Depending on the condition of the descending aorta, the distal anastomosis was performed in an “end-to-end” fashion, or as an elephant trunk procedure, classic or frozen. Concomitant procedures concerning aortic pathology included: implantation of supra-coronary prosthesis, Bentall’s technique, and David’s procedure.

**Statistical analysis:** Normally distributed continuous variables were expressed as the mean ± standard deviation and were compared between the groups using the Student’s t-test. Non-normally distributed continuous variables were expressed as median and quartiles and were compared between the groups with Wilcoxon rank sum test. Categorical variables were expressed as frequency distributions and single percentages, and were compared between the groups using χ² test and Fisher’s exact test, where appropriate. Surgical survival analysis was conducted by Kaplan-Meier method with log-rank test for group comparisons. Estimations of risk for surgical mortality were calculated using Cox regression analysis. Stepwise multivariate logistic regression was performed in order to identify independent predictors for statistically significant morbidity assessed by univariate analysis. A value of two-sided P less than 0.05 was considered statistically significant. Statistical analysis was performed with SPSS statistical package version 17.0 (SPSS Inc, Chicago, IL, USA).

**Results**

**Study population:** From January 2011 to December 2015, after obtaining approval by the ethic committee, a total of 420 consecutive patients underwent extended hemiarch or total arch replacement with concomitant procedures in this center. Among them, 362 patients suffering from acute type A aortic dissection who met the inclusion criteria were entered into this study. Twenty-four patients were excluded: an echocardiographic estimated left ventricular ejection fraction of 35% or less in 3 patients, prior cardiac surgery in 3 patients, a positive history of chronic obstructive pulmonary disease in 8 patients, a positive history of chronic renal failure in 2 patients, and preoperative concurrent congestive heart failure in 8 patients. Another 5 patients with presurgical moderate or severe ARDS were also excluded. And finally, 333 patients of both sexes (265 men and 68 women) between 22 and 76 years of age (mean, 50 ± 12 years) were analyzed. In this cohort, 136 patients suffered from presurgical mild ARDS, with an incidence of 40.8%. Based on preoperative information, 136 patients with presurgical mild ARDS were entered into the mild-ARDS group, and the remaining 197 patients without presurgical ARDS were entered into the no-ARDS group.

Baseline characteristics are shown in Table I. Both of the two groups had similar baseline characteristics, including age, body mass index, left ventricular ejection fraction evaluated by echocardiography, and the proportion of older age, gender, recent smoking, hypertension, Marfan syndrome, coronary artery disease, diabetes, hypercholesterolemia, prior cerebrovascular accident, left ventricular dysfunction, and emergency operation. No significant difference emerged between the 2 groups in the proportion of obesity. Of note was that patients in the mild-ARDS group had significantly higher peak serum CRP levels as compared to the no-ARDS group (15.9 ± 7.8 mg/L versus 12.0 ± 6.7 mg/L, P < 0.001).

Intraoperative parameters are shown in Table I. Both of the 2 groups had similar intraoperative characteristics, including duration of cardiopulmonary bypass, aortic cross-clamping, deep hypothermic circulatory arrest and...
selective antegrade cerebral perfusion, lowest nasopharyngeal temperature, and the amount of red cells and plasma transfusion. The mild-ARDS group compared with the no-ARDS group had a slightly high proportion of prolonged cardiopulmonary bypass (more than 240 minutes) and prolonged aortic cross-clamping (more than 120 minutes) without significant differences (8.8% versus 6.1%, \( P = 0.391 \); 17.6% versus 15.2%, \( P = 0.550 \), respectively).

**Major postoperative morbidity:** Major postoperative morbidity is summarized in Table II. No significant differences emerged between the 2 groups in the occurrence of major postoperative morbidity, including low cardiac output, stroke, hepatic failure, renal failure, re-operation for bleeding and deep sternal wound infection, except for postoperative pulmonary complications. Notably, there was no phrenic nerve or recurrent laryngeal nerve dysfunction. In addition, patients in the mild-ARDS group had significantly longer length of ICU stay as compared to the no-ARDS group (median, 5 days versus 4 days, \( P = 0.031 \)).

Detailed data of postoperative respiratory system are shown in Table II. Besides lower oxygenation index at postsurgical 6 hours (253 ± 41 mmHg versus 276 ± 55 mmHg, \( P < 0.001 \)), patients in the mild-ARDS group compared with the no-ARDS group had a significantly longer duration of mechanical ventilation and a higher incidence of postoperative pulmonary complications. Postoperative pulmonary complications included pneumonia, prolonged mechanical ventilation (more than 48 hours), and re-intubation. Except higher ratio of prolonged mechanical ventilation (34.6% for the mild-ARDS group versus 20.8% for the no-ARDS group, \( P = 0.008 \)), no significant difference emerged between the two groups in the incidence of pneumonia and the ratio of re-intubation. No significant difference was found between the two groups in the ratio of tracheotomy.

Multivariate logistic regression analysis displayed that grouping (the mild-ARDS group versus the no-ARDS group) and recent smoking were two independent risk factors for pulmonary complications following surgical repair of acute type A aortic dissection by means of DHCA. The risk of postoperative pulmonary complications in patients with presurgical mild ARDS was 4.25 times that in patients without presurgical ARDS (OR = 4.25, 95% CI 2.05-7.72, \( P = 0.004 \)).

**Surgical mortality:** A total of 24 patients died after surgery, with a surgical mortality of 7.2%. The surgical mortality was more than three-fold higher in the mild-ARDS group compared with the no-ARDS group (12.5% versus 3.6%, \( P = 0.002 \)). Deaths were due to multiple organ failure in 11 patients, severe sepsis in 8 patients, gastrointestinal bleeding in 4 patients, and malignant arrhythmia in 1 patient.
### Table II. Major Postoperative Morbidity and Surgical Mortality

<table>
<thead>
<tr>
<th></th>
<th>mild-ARDS group</th>
<th>no-ARDS group</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>LCOS</td>
<td>18 (13.2%)</td>
<td>21 (10.7%)</td>
<td>0.492</td>
</tr>
<tr>
<td>Respiratory morbidity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oxygenation index at postsurgical 6 hours</td>
<td>253 ± 41</td>
<td>276 ± 55</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>Median duration of MV (hours)</td>
<td>60 (37, 113)</td>
<td>35 (26, 83)</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>Tracheotomy</td>
<td>16 (11.8%)</td>
<td>14 (7.1%)</td>
<td>0.174</td>
</tr>
<tr>
<td>Pulmonary complications</td>
<td>79 (58.1%)</td>
<td>70 (35.5%)</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>26 (19.1%)</td>
<td>25 (12.7%)</td>
<td>0.123</td>
</tr>
<tr>
<td>Prolonged MV (&gt; 48 hours)</td>
<td>47 (34.6%)</td>
<td>41 (20.8%)</td>
<td>0.008</td>
</tr>
<tr>
<td>Re-intubation</td>
<td>6 (4.4%)</td>
<td>4 (2.0%)</td>
<td>0.327</td>
</tr>
<tr>
<td>Stroke</td>
<td>16 (14.7%)</td>
<td>12 (6.1%)</td>
<td>0.073</td>
</tr>
<tr>
<td>Hepatic failure</td>
<td>19 (14.0%)</td>
<td>24 (12.2%)</td>
<td>0.623</td>
</tr>
<tr>
<td>Renal failure</td>
<td>22 (16.2%)</td>
<td>23 (11.7%)</td>
<td>0.253</td>
</tr>
<tr>
<td>Re-operation for bleeding</td>
<td>14 (10.3%)</td>
<td>18 (9.1%)</td>
<td>0.711</td>
</tr>
<tr>
<td>Deep sternal wound infection</td>
<td>12 (8.8%)</td>
<td>9 (4.6%)</td>
<td>0.167</td>
</tr>
<tr>
<td>Median length of ICU stay (days)</td>
<td>5 (3.9)</td>
<td>4 (3.6)</td>
<td>0.031</td>
</tr>
<tr>
<td>Surgical mortality</td>
<td>17 (12.5%)</td>
<td>7 (3.6%)</td>
<td>0.002</td>
</tr>
</tbody>
</table>

ARDS indicates acute respiratory distress syndrome; LCOS, low cardiac output syndrome; MV, mechanical ventilation; and ICU, intensive care unit.

![Figure](image)

**Figure.** Actuarial curves of surgical survival after aortic arch surgery.

As shown in the figure, Kaplan-Meier curves displayed a poor surgical survival in the mild-ARDS group ($\chi^2 = 12.958$, Log-rank $P < 0.001$). And Cox regression revealed that grouping (the mild-ARDS group versus the no-ARDS group) was a significant variable related to surgical mortality. After the Cox proportional model was used, the hazard ratio for surgical mortality in patients with presurgical mild ARDS was 2.52 (95% CI 1.41-5.32, $P = 0.016$).

**Discussion**

According to published data, acute lung injury (or mild ARDS) is one of most common presurgical compli-
cations caused by systemic inflammatory response resulted from aortic injury due to acute type A aortic dissection. In this series of 333 patients undergoing surgical repair of acute type A aortic dissection by means of DHCA, spanning a 5-year time period, 136 patients were diagnosed as presurgical mild ARDS, with an incidence of 40.8%. The incidence of presurgical mild ARDS in this series was similar to other series which reported the incidence of 30-45%. This study showed that patients in the mild-ARDS group, compared with the no-ARDS group, had significantly higher plasma levels of inflammatory marker, C-reactive protein, which was consistent with the findings of previous studies. So, this study again suggested that systemic inflammation played an important role in the occurrence and development of mild ARDS.

An important finding of this study was that presurgical mild ARDS increased postoperative respiratory morbidity. In patients with acute type A aortic dissection, nonpulmonary factors can be contributing to the poor oxygenation, including congestive heart failure. So, 8 patients (2.4%) with congestive heart failure in this cohort were excluded to rule out the contribution of heart failure to our results. And to rule out the contribution of obstructive pulmonary disease to our results, another 8 patients (2.4%) with a positive history of chronic obstructive pulmonary disease were excluded from this study. In this series, univariate analysis showed that patients in the mild-ARDS group compared with the no-ARDS group had lower oxygenation index at postsurgical 6 hours, longer duration of mechanical ventilation, higher ratio of prolonged mechanical ventilation and higher incidence of postoperative pulmonary complications, and multivariate logistic regression analysis displayed that the risk of postoperative pulmonary complications in patients with presurgical mild ARDS was 4.25 times that in patients without presurgical ARDS. Furthermore, results mentioned above were obtained under condition of similar baseline and intraoperative characteristics between the two groups. So, it may be inferred that presurgical mild ARDS, together with surgical trauma and cardiopulmonary bypass as well as DHCA, further aggravated lung injury postoperatively and increased postoperative respiratory morbidity. The reason may be that in the process of strong systemic inflammation, more pro-inflammatory cytokines were released, causing more neutrophil accumulation and activation in the lungs. The activated neutrophils released toxic mediators that destroyed the pulmonary capillary endothelium and increased their vascular permeability, leading to the alveolar fluid accumulation, aggravating lung injury, and finally reducing postoperative pulmonary function and increasing postoperative respiratory morbidity. So, this study suggested that presurgical mild ARDS aggravated lung injury and more severely affected postoperative respiratory system in patients undergoing surgical repair of acute type A aortic dissection by means of DHCA. Additionally, the mild-ARDS group had significantly longer length of ICU stay as compared to the no-ARDS group (median, 5 days versus 4 days, \( P = 0.031 \)), which may result from increased postoperative respiratory morbidity.

Another important finding of this study was that pre-surgical mild ARDS increased surgical mortality after surgical repair of acute type A aortic dissection by means of DHCA. In this series, surgical mortality was 7.2%, which was lower than that in previous reports. Reason of this difference can be the age (50 ± 12 years old for this series versus 63 ± 14 years old for a cohort of International Registry Of Acute Aortic Dissection) and predisposing factors for acute aortic dissection (hypertension and Marfan syndrome for this series versus hypertension and aortic degeneration for a cohort of International Registry Of Acute Aortic Dissection). Through univariate analysis, Kaplan-Meier curves and Cox regression, this study revealed the mild-ARDS group compared with the no-ARDS group increased surgical mortality after aortic arch surgery, which may contribute from increased postoperative respiratory morbidity and a prolonged ICU stay.

This study revealed that presurgical mild ARDS increased postoperative respiratory morbidity, and consequently increased surgical mortality after aortic arch surgery. Concurrent presurgical mild ARDS may predict a poor prognosis for patients undergoing surgical repair of acute type A aortic dissection by means of DHCA. It is important to attach great importance to concurrent presurgical mild ARDS, and to take measures to reduce its adverse effect. Perioperative anti-inflammatory therapy with neutrophil elastase inhibitors, improving operative technique to shorten duration of cardiopulmonary bypass and aortic cross-clamping, meticulous intraoperative hemostasis to reduce the amount of blood products, reasonable application of ultrafiltration during cardiopulmonary bypass, and careful management of respiratory disorder may contribute toward relieving perioperative systemic inflammation, reducing respiratory morbidity, shortening the length of ICU stay, and finally, improving the prognosis.

The present study has some potential limitations. Firstly, it was a single-center, retrospective study, which may influence the generalizability. The number of patients in each group is relatively small, implying some weakness of the statistical results. A final determination would need a multi-center study involving a larger sample size. Second, although the inclusion criteria were clearly aimed at selecting a homogenous study population (freedom from chronic obstructive pulmonary disease, etc.), the role of some confounding factors (duration of cardiopulmonary bypass, blood transfusion, postoperative cardiac function, etc.) was difficult to assess due to the unpredictability of the surgical procedure and, above all, due to the small sample size. Inferences from the results of this study to the routine clinical environment might need careful consideration. Third, this study did not explore the precise mechanisms of presurgical mild ARDS negatively affecting postoperative clinical outcomes. Finally, this study only focused on early clinical outcomes after surgical repair of acute type A aortic dissection by means of DHCA between the mild-ARDS group and the no-ARDS group. Further follow-up is necessary to evaluate the medium- and long-term outcomes.

Conclusion

This single-center retrospective study reviewed 333
eligible patients undergoing surgical repair of acute type A aortic dissection by means of DHCA, spanning a 5-year time period, and found that presurgical mild ARDS increased postoperative respiratory morbidity, and then increased surgical mortality after surgical repair of acute type A aortic dissection by means of DHCA.

Disclosures

Conflict of interest: None.

References