Severe Bronchospasm During Aortic Surgery for Type A Aortic Dissection

Zairong Lin[1], Kun'an Huang[1], Dongdong Chen[2], and Qianzhen Li[1]

[1]Fujian Medical University Union Hospital
[2]Anxi County Hospital

July 2, 2020

Abstract

Severe bronchospasm during cardiopulmonary bypass is an unusual but potentially fatal event. No literature has previously reported such an event observed during surgery for type A aortic dissection. Herein, we report on a case of severe bronchospasm following cardiopulmonary bypass during aortic surgery for type A aortic dissection. Bronchospasm did not respond to any conventional therapy, necessitating extracorporeal membrane oxygenation. Extracorporeal membrane oxygenation thus serves as an alternative and effective therapy for refractory bronchospasm.

Introduction

Severe bronchospasm during cardiopulmonary bypass (CPB) is an unusual event (1-12), with less than 20 cases have been published. However, the outcomes from such an event could have disastrous consequences. This condition presents with high airway pressure, low expiratory tidal volume, and a lack of lung deflation (1, 2), resulting in gaseous exchange and hemodynamics.

In addition, type A aortic dissection (TAAD) is a serious and fatal disease, which usually requires emergency aortic surgery undergoing CPB to prevent death from aortic rupture (13). Up until now, no report has described severe bronchospasm, which was observed during surgery for TAAD. We report on a case of severe bronchospasm immediately following CPB during aortic surgery for TAAD. Bronchospasm did not respond to any conventional therapy, necessitating veno-venous extracorporeal membrane oxygenation (VV-ECMO).

Case report

A 50 year old male weighing 75 kg was transferred to our center for sudden chest pain lasting for 35 hours. He had an eight-year history of hypertension, but did not receive regular medical treatment. He had no known allergies, but had suffered from intermittent asthma for four years, treated with an albuterol inhaler, and theophylline irregularly. His last hospitalization was 13 months ago. He was febrile at 37.8 degree centigrade. His blood pressure was 65/45 mmHg, the heart rate was 97 beat per minute, and the central venous pressure was 20 mmHg. The distal skin was pale and cold. He had a normal neurological exam. The blood routine analysis showed that a white blood cell count of 18.2*109/L, and a neutrophil frequency of 86.4 percent. Computer tomography angiography (CTA) found a large tear on the ascending aorta, which extended to the common iliac artery, and presence of massive pericardial fluid and patchy exudation in both lungs. Pneumonia was diagnosed by fever, the blood routine and CT scans. Transthoracic echocardiography reconfirmed diagnosis of TAAD, and revealed massive pericardial fluid, and mild aortic valve regurgitation. The left ventricular ejection fraction was 50%. Considering the unstable dynamics, the patient was sent to the operating room immediately for emergency surgery. Two million units of Penicillin was given intravenously before he was sent to the operating room.

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For premedication, 10 mg of morphine and 1 mg of penehyclidine hydrochloride (anticholinergic drugs) was given intramuscularly, and 5 mg of dexamethasone and 0.25 g of aminophylline were given intravenously. Anesthesia was achieved with fentanyl, midazolam, atracurium besylate and isoflurane. After tracheal intubation, the lungs were clear to auscultation, and peak inspiratory pressure was 18 mmHg. Arterial blood gas analysis revealed no specific abnormalities. After median sternotomy, institution of CPB proceeded uneventfully. Then the aortic root repair and ascending aorta replacement was performed. When the rectal temperature decreased to 23, deep hypothermia circulation arrest (DHCA) and antegrade selective cerebral perfusion (SCP) was used during the procedure of total arch remodeling by a triple-stent graft implantation as we have previously described (14). After proximal and distal anastomoses complete the crossclamp was removed, rewarming was started. The aortic cross-clamp time was 54 minutes, and the total circulatory arrest time was five minutes, while the SCP time was 20 minutes. When mechanical ventilation was resumed, expiratory wheezing was heard, and the airway pressure was elevated to 50 mmHg. In addition, the lungs remained inflated. After a few minutes, the lungs over-expanded and bulged into the mediastinum.

The ventilator (Infinity C700 for IT, Drager medical GmbH, Germany) was carefully checked and was found to be working satisfactorily. A suction catheter was easily passed through the endotracheal tube, and no secretions were removed. The lungs deflated when they were disconnected from ventilator. However, they re-inflated after the ventilation was resumed, even though the inspiratory pressure was lower down to 5 mmHg. Fiber optic bronchoscopy was performed, and no tracheobronchial abnormality or obstruction was found. The left atrial pressure was 7 cm H_2O. Hemodynamics were stable and transesophageal echocardiogram confirmed normal biventricular function. No urticaria, skin erythema or edema were present and an initial diagnosis of severe bronchospasm was made.

To alleviate bronchospasm, ventilation with isoflurane was begun. Intravenous amino-phylline 0.5 g and methylprednisolone 0.5 g was given, followed by isoproterenol and metaproterenol inhalation by the intra-tracheal route. 0.1ug*kg-1*min-1 of epinephrine was continuously infused intravenously. However, the lungs showed sustained inflation for 60 minutes. Considering the long CPB time, VV- ECMO was performed. The outflow cannula was set in the right femoral vein, and the inflow site was the right jugular internal vein. A Quadrox oxygenator and a Rotaflow Centrifugal pump (Maquet, Hirrlingen, Germany) were connected to the extra-corporeal circuit. The flow rate was set at 3.5 L/min, and the fraction of inspired oxygen was 40 percent. The ventilator was then temporarily stopped, following which, the lungs were found to deflate gradually. Next, ventilation was resumed under the pressure controlled mode, and the peek inspiratory pressure was set as 10 mmHg. The positive end expiratory pressure was set at 5 mmHg to prevent the collapse of the alveoli. The patient was stabilized hemodynamically, and the patient was weaned off CPB. Blood gas analysis revealed that the partial pressure of oxygen was 175 mmHg, and the partial pressure of carbon dioxide was 40 mmHg. After careful hemostasis and sternal closure, the patient was the sent to the adult intensive care unit (ICU). While at the ICU, antibiotic therapy was continued. Methylprednisolone 80mg was given every eight hours during the first 48 hours. Then the dose was reduced gradually in 3 days. A bronchodilator was used for inhalation every four hours. Eight hours later, the inspiratory pressure of the ventilator was set to 18 mmHg with stable hemodynamics. ECMO was weaned 26 hours later without hemorrhagic complications. The patient was weaned from the mechanical ventilator and extubated four days post-operatively. The remainder of the patient's hospital stay was otherwise uneventful, and he was discharged 12 days post-surgery.

Discussion

Severe bronchospasm is a rare complication of open-heart surgery (3). However, the outcome can be extremely poor without timely and effective treatment. TAAD is a serious disease, which usually requires emergency surgery with a high rate of perioperative mortality (15). It is a very dangerous condition once the severe bronchospasm occurs following CPB during aortic repair for TAAD. Thus far, no literature has been previously reported on this topic.

Bronchospasm is known to be caused by high airway reactivity. The patient in the current case report presented with a history of asthma for four years, which carried a high probability of airway hyper-

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responsiveness. Pneumonia could be another inducement of bronchospasm. However, it was impractical to apply adequate dosing and duration of bronchodilator support and antibiotic therapy, because of the cardiac tamponade and unstable hemodynamics in this case.

The diagnosis of bronchospasm should exclude wheezing caused by other diseases, ventilator problems and mechanical obstruction of the airway. Mechanical causes for wheezing include cardiac wheezing and pneumothorax (1). Cardiac wheezing will be accompanied by elevated left atrial pressure and alveolar edema, and pneumothorax is unlikely to involve both lungs. Neither condition was noted in this patient. In addition, the faults of the ventilator and its circuit could lead to ventilator disorders, and the tracheal cannula could be obstructed by mucosal secretions or blood clots, or it could oppressed by the transthoracic echocardiographic probe. We checked the ventilator and its circuit very carefully, following which, no faults were found. In addition, the catheter suction and fiber optic bronchoscopy through the endotracheal tube was performed to rule out tracheobronchial obstruction.

The etiology of severe bronchospasm during CPB remains unclear. Light levels of anesthesia could cause high reactivity of the bronchial smooth muscle cells (16). Moreover, the β-adrenoreceptor antagonist is another trigger of bronchospasm, especially in patients with asthma (2). Deeper anesthetic depth and inhaled β-adrenergic agents are recommended once suspicious bronchospasm occurs. However, neither of these approaches appear to be an appropriate reason that might help explain presentation of bronchospasm in this patient. Drug allergy and transfusions could also induce bronchospasm. However, allergy is usually accompanied by cutaneous allergic manifestations, or cardiovascular abnormality, which did not appear in this patient.

CPB and low core body temperature could be potential triggers in this patient. Activation of the complement component C3a- and C5a-derived anaphylatoxins are known to occur in most patients during CPB, and the pump-oxygenator is the usual site of complement activation (17). Anaphylatoxins are inflammatory mediators that stimulate the release of mast cell-derived histamine, contraction of the bronchial smooth muscle and increases in vascular permeability (18). The levels of C3a and C5a complement components correlate with the duration of CPB (1). Surgery for TAAD is very time-consuming, which could result in systemic inflammatory responses, activation of complement, and activation of thrombin deposition, cytokines, endothelin, endotoxins, and neutrophils (19), which collectively carries a high risk of airway hyper-responsiveness. Decreased temperature might present as an additional inducing stimulus. Joseph Simpson et al., (9) reported on a case of cold-induced bronchospasm during coronary artery bypass surgery. DHCA is commonly used in surgery for TAAD. Although DHCA may preserve better cerebral function during circulatory arrest, the presence of hypothermia could quite possibly trigger bronchospasm in this case, despite there being no direct or firm evidence indicating their relationship. This possibility served to prompt us to consider shortening the duration of CPB and DHCA, which might decrease the incidence of bronchospasm, though it was already very low. Perhaps hemi-arch repair or the branch-first technique (20) could be more beneficial than total arch repair in such cohorts of patients that present with asthma.

Conventional treatments for managing acute bronchospasm include inhaled anesthetics, aerosolized β-adrenergic agents in combination with aminophylline, and intravenous epinephrine and corticosteroid support (1). However, these therapies were of no use to the patient described in this report. The mechanism remains unknown. To address bronchospasm to a more settled pattern, VV-ECMO was performed. ECMO rapidly restores gas oxygenation, corrects respiratory acidosis, removes carbon dioxide, and reverses hemodynamic deterioration, especially for severe refrac¬tory bronchospasm not responding to conventional therapy (21). The patient in the current report, had a good recovery by applying ECMO, showing the excellent effectiveness of this procedure. The inflow site of ECMO should be prudently determined in patients that present with TAAD. Arterial cannulation might potentially aggravate aortic dissection. Therefore, we propose instead of submit that VV ECMO might be a more suitable procedure than veno-arterial ECMO in this patient.

Conclusions

Severe bronchospasm may occur following CPB during aortic surgery in patients with TAAD. Possible

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inducing events might include preoperative history or presentation of asthma and pneumonia, CPB and hypothermia. The cardiac operating room has the distinct advantage of a cardiac surgeon, cardiac anesthesiologist and perfusionist. Emergency, high-risk surgery complicated by bronchospasm is a very tough situation. ECMO thus serves as an alternative and effective therapy for refractory bronchospasm.

Declarations

Conflict of Interest: All authors declare that they have no conflict of interest.

Informed consent: Informed consent was obtained from all individual participants included in the study.

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