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Severe Bronchospasm During Aortic Surgery for Type A Aortic Dissection

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ABSTRACT

Severe bronchospasm during cardiopulmonary bypass is an unusual but potentially fatal event. No literature previously has 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 [Tuman 1986; Neustein 1992; Shiroka 1982; Simpson 1993], with less than 20 cases that 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 [Tuman 1986; Neustein 1992], 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 [Yang 2018]. 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 he did not receive regular medical treatment. He had no known allergies, but he suffered from intermittent asthma for four years. He irregularly was treated with an albuterol inhaler and theophylline. His last hospitalization was 13 months prior. He was febrile at 37.8 degree centigrade. His blood pressure was 65/45 mmHg. The blood routine analysis showed a white blood cell count of 18.2*109/L and a neutrophil frequency of 86.4%. Computer tomography angiography (CTA) confirmed the diagnosis of TAAD and presented massive pericardial fluid and patchy exudation in both lungs. Pneumonia was diagnosed by fever and routine blood 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 intravenously was given before he was sent to the operating room.

For premedication, 10 mg of morphine and 1 mg of penehyclidine hydrochloride (anticholinergic drugs) intramuscularly was given, and 5 mg of dexamethasone and 0.25 g of aminophylline intravenously were given. 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. The surgical procedure was referred as we previously have described [Chen 2014]. 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. 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 found to be satisfactorily working. A suction catheter easily was passed through the endotracheal tube, and no secretions were removed. The lungs deflated when they were disconnected from the ventilator. However, they reinflated after ventilation was resumed.

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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 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 intratracheal route. 0.1ug*kg-1*min-1 of epinephrine continuously was infused intravenously. However, the lungs showed sustained inflation. 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%. The ventilator was then temporarily stopped, following which, the lungs were found to gradually deflate. 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 hemodynamically was stabilized, 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 gradually was reduced in three 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 postoperatively. 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 [Shiroka 1982]. 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 [Evangelista 2018]. It is a very dangerous condition once the severe bronchospasm occurs following CPB during aortic repair for TAAD. Thus far, no literature previously has been 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-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 [Tuman 1986]. 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 be 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 [Hirshman 1983]. Moreover, the β-adrenoreceptor antagonist is another trigger of bronchospasm, especially in patients with asthma [Neustein 1992]. 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 also could 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 [Chenoweth 1981]. Anaphylatoxins are inflammatory mediators that stimulate the release of mast cell-derived histamine, contraction of the bronchial smooth muscle and increases in vascular permeability [Fernandez 1978]. The levels of C3a and C5a complement components that correlate with the duration of CPB [Tuman 1986]. 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 [Wang 2013], which collectively carries a high risk of airway hyperresponsiveness. Decreased temperature might present as an additional inducing stimulus. Simpson et al. [Simpson 1993] 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 quite possibly could 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 already was very low.

Conventional treatments for managing acute bronchospasm include inhaled anesthetics, aerosolized β-adrenergic agents in combination with aminophylline, and intravenous epinephrine and corticosteroid support [Tuman 1986]. 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 refractory bronchospasm not responding to conventional therapy [Mikkelsen 2007]. 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 who present with TAAD. Arterial cannulation might potentially aggravate aortic dissection. Therefore, we propose 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 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.

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