

Case Report

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Early administration of venovenous extracorporeal life support for status asthmaticus during anaesthetic induction: case report and literature review

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Abstract: Here we report a case of a 40-year-old man who visited the emergency room with severe chest pain. He showed a Stanford type B aortic dissection on chest-computed tomography. Despite medical treatment and malperfusion of lower extremities, acute renal failure developed; hence thoracic endovascular aortic repair (TEVAR) was considered under general anaesthesia. After endotracheal intubation, ventilation with low tidal volume required high inspiratory airway pressure. An arterial blood gas analysis showed PaCO₂ of 61.8mmHg and PaO₂ of 26.4mmHg, indicating a status asthmaticus of hypoxaemia and hypercarbia, which did not respond to bronchodilator or mechanical ventilation. Impending cardiac arrest was treated using venovenous extracorporeal life support, which was administered by percutaneous femoral cannulation. Surgical procedure was completed without any complications. Extracorporeal life support was weaned at one day after the operation. The patient was discharged without any complications.

Keywords: Asthma; Extracorporeal membrane oxygenation

1 Introduction

Status asthmaticus, a reversible respiratory failure, does not respond to conventional bronchodilator. It presents

mild dyspnea to severe bronchospastic asphyxia with potential disaster during anaesthetic induction. It is usually developed during anaesthetic induction. However, it can be detected any time during perioperative period. Prompt recognition of this situation and appropriate management are very important to avoid morbidity and mortality [1]. Extracorporeal life support is characterized by drainage of venous blood, removal of carbon dioxide, adding oxygen in oxygenator, and returning blood by centrifugal pump. It is widely used for acute respiratory failure with pneumonia, acute respiratory distress syndrome (ARDS), bridge to lung transplantation, and primary graft dysfunction after lung transplantation [2]. A few cases of extracorporeal life support for severe status asthmaticus patient have been reported [3]. However, severe bronchospastic asphyxia during anaesthetic induction has not been reported yet.

2 Case report

A 40-year-old male (140kg, 165cm) patient was transferred to our emergency room with chest pain. He had hypertension, coronary artery disease, and bronchial asthma that were not treated. He visited the pulmonology department with exertional dyspnea one year before surgery. On pulmonary function test, he had mild obstructive airway disease and allergen skin prick test revealed bronchial asthma to specific allergen. On chest X-ray, there was no abnormal finding (Figure 1). Contrast-enhanced computed tomography scan revealed Stanford type B aortic dissection from descending thoracic aorta to iliac arteries (Figure 2). Despite medical treatment with labetalol (120mg/h) and nicardipine (2.5mg/h), lower limb ischaemia and acute renal failure developed due to malperfusion at HAD #2 (Figure 3). Urgent surgical intervention was needed. Thoracic endovascular aortic repair (TEVAR) followed. Before transferring him to the operating room, 0.2mg glycopyrrolate was injected intramuscularly. For anaesthetic induc-

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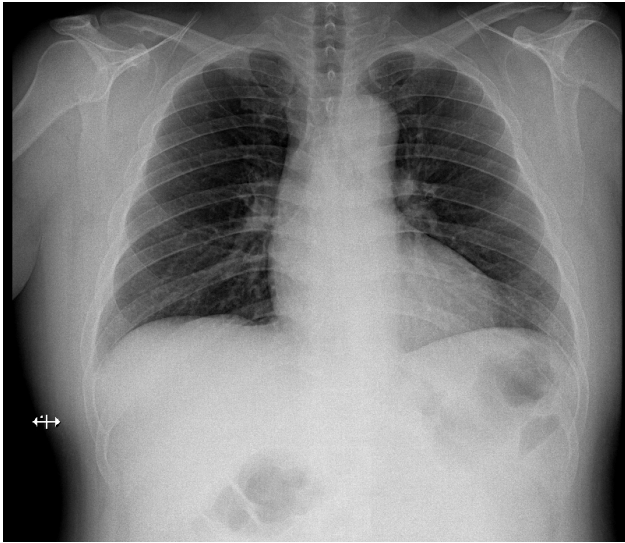


Figure 1: Preoperative chest X-ray without abnormal finding.



Figure 2: Contrast-enhanced computed tomography scan showing Stanford type B aortic dissection.



Figure 3: Contrast-enhanced computed tomography scan showing malperfusion of left kidney (white arrow) and left iliac artery (blue arrow)

tion, 2% propofol and remifentanyl were used. The patient was paralyzed with 50mg rocuronium. Anaesthesiologist had a difficult time in endotracheal intubation with poor laryngoscopic view. After endotracheal intubation, expiratory tidal volume was 100ml with high inspiratory airway pressure (70cmH₂O). On arterial blood gas analysis after 100% oxygen inhalation, severe hypoxemia and respiratory acidosis were found (pH 7.198, PCO₂ 61.8mmHg, PaO₂ 26.4mmHg, O₂ Saturation 36.1%). The anaesthesiologist performed flexible bronchoscopy. There was no endotracheal or endobronchial obstruction. Severe bronchospastic asphyxia was suspected and epinephrine 0.5 mg was given subcutaneously. Then 0.5ml albuterol of 0.5% formulation was inhaled into the endotracheal tube. Methylprednisolone (1mg/Kg) was also given intravenously. After conventional therapy for severe bronchospasm, his hypoxemia and respiratory acidosis were slightly improved (pH 7.264, PCO₂ 56.7mmHg, PaO₂ 43.2mmHg, O₂ Saturation 71.3%). However, his blood pressure dropped

down (systolic blood pressure < 70 mmHg) and bradycardia (40/min) developed. Conventional therapy was ineffective for him and cardiac arrest caused by persistent hypoxemia was impending. At 12 minutes after bronchospastic attack, emergent venovenous extracorporeal life support (VV ECLS) was initiated. A 21 French drainage cannula (Bio-Medicus™ Multi-Stage Femoral Venous Cannula, Medtronic, Minneapolis, MN, USA) was inserted into the right common femoral vein and advanced into right atrium. A 17 French return cannula (Bio-Medicus™ Femoral Arterial Cannula, Medtronic, Minneapolis, MN, USA) was placed at the left external iliac vein. The anaesthesiologist already inserted central venous catheter into the right internal jugular vein. Only both groins were cleansed and draped for TEVAR. Therefore, we planned secondary cannulation via the right internal jugular vein if needed after emergency VV ECLS via femoral venous cannulation. All procedures were done percutaneously with fluoroscopic guidance (Figure 4). Quadrox oxygenator and rotaflow pump (Maquet, Hirrlingen, Germany) were connected to extracorporeal circuit. Extracorporeal circulatory blood flow rate was 4.5 L/min. Peripheral O_2 saturation raised up to 80%. He was stabilized haemodynamically. We changed the mode of mechanical ventilation from volume controlled to pressure controlled ventilation. Limitation of inspiratory airway pressure was 40 cmH₂O. Expiratory tidal volume was less than 70 ml. After VV ECLS, hypoxemia and hypercarbia were improved on arterial blood gas analysis (pH 7.305, PCO_2 49.6 mmHg, PaO_2 56.2 mmHg, O_2 Saturation 86.2%). Intimal tearing portion of thoracic aorta was sealed with stent graft (Valiant thoracic stent graft with the Captiva delivery system, Medtronic) on VV ECLS. The patient was transferred to intensive care unit after surgery without any problem. Bronchodilator was used for inhalation every 4 hours. After 12 hours after VV ECLS, his expiratory tidal volume was increased up to 450 ml with appropriate inspiratory airway pressure (less than 40 cmH₂O). VV ECLS was weaned at POD#1 and decannulation was done without haemorrhagic complication. Patient could be weaned from mechanical ventilator and extubated at POD#2. He could be discharged at POD#7 without any respiratory or cognitive dysfunction. Malperfusions of left kidney and left iliac artery were resolved and there was no abnormal finding of lung parenchyma and pulmonary vessels on contrast-enhanced computed tomography scan at POD#14 (Figure 5). The patient has been treated with a bronchodilator at the Pulmonology Department. He has no more episode of bronchospastic asphyxia.

Ethical approval: The research related to human use has been complied with all the relevant national regula-

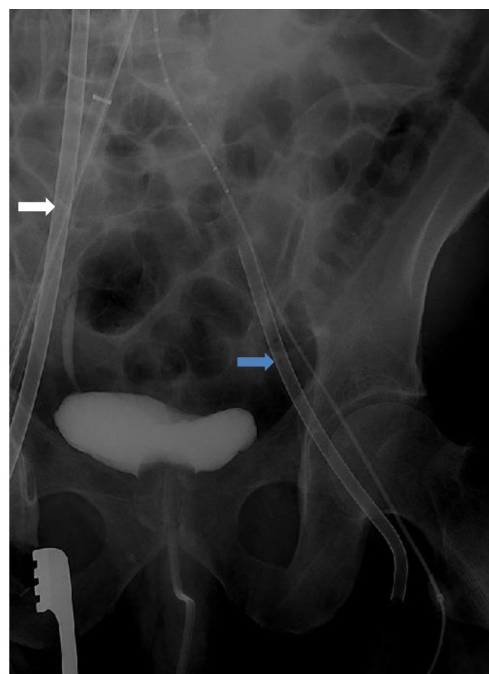


Figure 4: Venous cannulations for extracorporeal life support under fluoroscopic guidance. A 21 French venous cannula was inserted into the right common femoral vein for venous drainage (white arrow). A 17 French arterial cannula was inserted into the left common femoral vein for return (blue arrow).

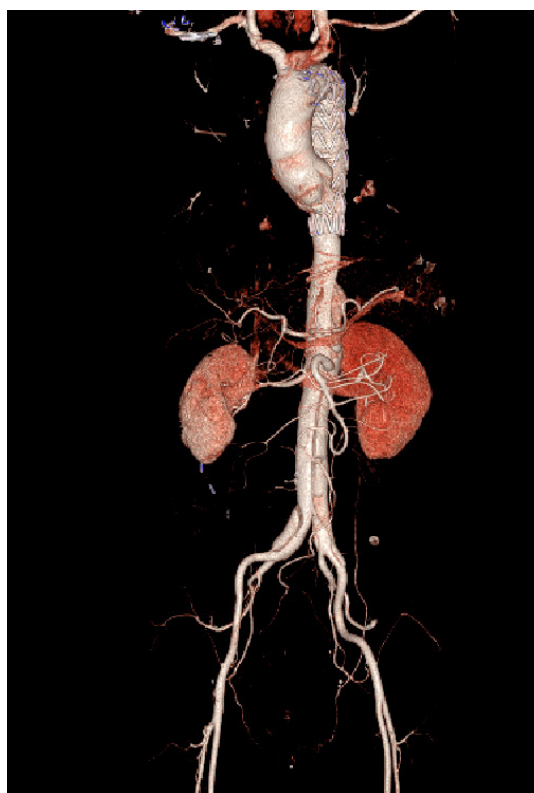


Figure 5: Malperfusions of left kidney and left iliac artery were resolved after operation at POD#14.

tions, institutional policies and in accordance the tenets of the Helsinki Declaration, and has been approved by the authors' institutional review board or equivalent committee.

Informed consent: Informed consent has been obtained from all individuals included in this study.

3 Discussion

Acute asthma is characterized by spasm of bronchial muscle, edema of bronchial wall, and increase of mucosal secretion. The goal of therapy for acute attack is to reverse obstructed airflow [4]. Bronchospasm is a severely exacerbated form of acute asthma. This usually leads to anaesthetic disaster. Such disaster encountered during anaesthetic induction period might involve IgE mediated anaphylaxis or nonallergic mechanism triggered by pharmacological and mechanical factors. A total of 4,000 bronchospasm events were reported in Australia, including 103 cases during the perioperative period. It might be triggered by pharmacological or mechanical stimulus [1]. A nonallergic mechanism (79%) is more frequently involved than an allergic mechanism. Among these non-allergic cases, most (44%) events occurred during the induction of anaesthesia [5]. In pharmacological factors, succinylcholine-induced anaphylaxis is the most common etiology. Cardiovascular shock is the hallmark of severe IgE mediated anaphylaxis. This sign might be associated with bronchospasm in 19-40% of patients with asthma or chronic obstructive pulmonary disease [6]. Perioperative bronchospasm commonly occurs during or after endotracheal instrumentation. Endotracheal tube is the irritant for well-innervated upper airway. It increases airway responsiveness [5]. Subcutaneously injection of epinephrine and inhalation of bronchodilator such as albuterol can be effective for rapid control of bronchospasm. Theophylline can increase intracellular cyclic adenosine monophosphate (cAMP) level, resulting in relaxation of bronchial smooth muscle. Corticosteroid can decrease inflammation of bronchial wall. Short-term therapy of corticosteroid is recommended when patient does not respond to bronchodilator therapy. However, its therapeutic effect will occur after 6 to 12 h. Inhalation of halothane as an anaesthetic agent is useful for bronchodilating effect. But, it has adverse effects such as increasing myocardial sensitization and cerebrospinal fluid pressure. Therefore, it is not commonly used. Despite these therapies, severe asthma attack can cause morbidity and mor-

tality during anaesthesia. For hypoxic brain damage and death during anaesthesia, respiratory events accounted for 28% of claims in the United States [7]. Extracorporeal life support could promptly restore gas oxygenation, resolve respiratory acidosis, and avoid haemodynamic deterioration. Excellent CO₂ removal, oxygenation, and rapid correction of respiratory acidosis for severe refractory asthma attack not responding to conventional therapy could be achieved by using extracorporeal life support. This could decrease intrinsic positive end expiratory pressure (PEEP) and prevent haemodynamic collapse and ventilator induced lung injury [3]. A total of 1,257 adult respiratory failure extracorporeal life support cases were reported to the international Extracorporeal Life Support Organization (ELSO) registry between January 1986 and September 2006. Asthma was the primary indication in 24 patients. They were treated by extracorporeal life support. Mean hours for mechanical ventilation until initiation of extracorporeal life support is 65.2 ± 67.7 h. Despite mechanical ventilation due to respiratory failure, mean pH is 7.17 ± 0.16 mmHg, PaCO₂ is 119 ± 58.1 mmHg, and PaO₂/FiO₂ ratio is 244 ± 180 before administration of extracorporeal life support. Extracorporeal life support was provided 111.9 ± 71.2 h and 83.3% of patients survived. Despite venoarterial extracorporeal life support (VA ECLS) was performed for only one because most cases were haemodynamically stable, mechanical, cardiovascular, and haemorrhagic complications developed in 19 (79.2%) patients [8]. In the current case, cardiac arrest impended due to hypoxemia and respiratory acidosis. We could not perform VA ECLS because cannulation into femoral artery could be dangerous. It might potentially aggravate aortic dissection in the patient. However, early administration of VV ECLS could correct hypoxemia and respiratory acidosis rapidly and haemodynamic stability could be achieved soon. Sudden resolution of severe asthma attack could occur after several days of pharmacologic therapy in some observational studies. However, we could not predict the exact time to recovery. Nevertheless, extracorporeal life support remains a reasonable therapy for treating adults with severe respiratory failure from status asthmaticus.

4 Conclusion

An obese patient with asthma developed severe status asthmaticus after endotracheal intubation. High oxygen mechanical ventilation, bronchodilator, or conventional medication including epinephrine was not effective. Early VV ECLS was initiated. The patient promptly recovered

from hypoxemia. He was kept stable haemodynamically. To the best of our knowledge, this is the first case report of a patient with impending cardiac arrest from severe asthmatic attack during anaesthesia who is successfully treated by VV ECLS. This case shows the effectiveness of VV ECLS for an acute, severe, and refractory status asthmaticus patient not responsive to mechanical ventilation or conventional therapy during anaesthesia. Therefore, early administration of VV ECLS can prevent haemodynamic instability, reduce complication, and optimize patient's outcome.

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Conflict of interest statement: Authors state no conflict of interest

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