J Anesth (2012) 26:265–268
DOI 10.1007/s00540-011-1288-z

CLINICAL REPORT

Successful treatment of severe asthma-associated plastic bronchitis with extracorporeal membrane oxygenation

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Received: 2 August 2011 / Accepted: 31 October 2011 / Published online: 17 November 2011

Abstract We describe a case of near-fatal asthma requiring extracorporeal membrane oxygenation (ECMO). The patient presented with severe respiratory distress, which was not responsive to conventional pharmacological therapy. The patient also failed to respond to mechanical ventilation and thus was placed on venovenous ECMO for temporary pulmonary support. A fiberoptic bronchoscopy revealed that large amounts of thick bronchial secretions had occluded the main bronchus, which suggested plastic bronchitis secondary to asthma. Aggressive airway hygiene with frequent bronchoscopies and application of biphasic cuirass ventilation for facilitation of secretion clearance were performed to improve the patient's respiratory status. The patient achieved a full recovery and suffered no neurological sequelae. This case illustrates that aggressive pulmonary hygiene with ECMO is a useful therapy for patients with asthma-associated plastic bronchitis.

Keywords Status asthmaticus · Plastic bronchitis · ECMO · Bronchoscopy · Biphasic cuirass ventilation

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Introduction

Despite recent advances in the management of asthma [1], the incidence of refractory asthma requiring mechanical support is still relatively high. Patients suffering from a combination of persistent or increasing hypercapnia, exhaustion, and a depressed mental status require ventilatory support and suffer significant mortality and morbidity [2]. Mechanical ventilation, especially in asthmatic patients, can cause increased air trapping and hyperinflation, predisposing the lungs to barotrauma. Equally, some patients fail to respond to conventional therapies and mechanical ventilation. Thus, mechanical ventilation is not always successful in critically ill asthma patients, and other therapies are being evaluated. Extracorporeal membrane oxygenation (ECMO) has shown some promise in pediatric patients with severe reversible respiratory failure [3–5], but its role in severe asthma has been limited. We report a case of status asthmaticus that was unresponsive to conventional ventilation and was successfully managed with venovenous ECMO. Massive atelectasis caused by plastic bronchitis in the course of asthma should be considered as an important cause of refractory respiratory failure. Plastic bronchitis following asthma is a relatively uncommon but life-threatening condition characterized by marked obstruction of the large airways by mucous plugs [6]. ECMO helps to ensure adequate oxygenation in such patients when pulmonary hygiene with frequent bronchoscopies is performed.

Case report

An 11-year-old girl suffering from severe dyspnea called an ambulance herself and was taken to the emergency department of Kyoto Second Red Cross Hospital. She presented with a history of poorly controlled asthma since the age of 2 years. Upon arrival, she received intravenous methylprednisolone and nebulized isoproterenol. Despite vigorous treatment of asthma, her condition did not improve, and her consciousness level decreased. Arterial blood gas analysis revealed severe hypercapnia and respiratory acidosis (pH, 7.02; $PaCO_2$, 122 mmHg; PaO_2, 112 mmHg). She was therefore intubated and transferred to the intensive care unit (ICU) after the initiation of mechanical ventilation. Pressure-regulated volume control was initiated at an inspiratory fraction of oxygen (FiO_2) of 0.6, a tidal volume of 210 ml, and a positive end-expiratory pressure (PEEP) of 5 cmH_2O. A muscle relaxant was used to improve patient–ventilator asynchrony under adequate sedation. However, hypercapnic respiratory failure persisted with no improvement in her blood gases. High-frequency oscillatory ventilation was also attempted for her worsening hypoxemia but was less efficient at achieving oxygenation. On day 2 after admission, she received continuous renal replacement therapy in response to a complicating acute kidney injury and hyperkalemia (7.3 mmol/l). On day 5, we decided to implement veno-venous (V–V) ECMO because of worsening refractory hypoxemia and hypercapnia. Thus, 15 Fr. Bio-Medicus cannulas (Medtronic, Minneapolis, MN, USA) were percutaneously inserted into both common femoral veins. Venous blood from the femoral vein was drained into the right atrium using a Capiox centrifugal pump (Terumo, Tokyo, Japan) and then passed through a Mera artificial lung (Senko Medical Instruments, Tokyo, Japan). The extracorporeal blood flow rate was initially set at 1.6 l/min, and the rate of oxygen flow was set at 2.0 l/min. Following the implementation of ECMO, her blood gases rapidly normalized (pH, 7.47; $PaCO_2$, 54 mmHg; PaO_2, 116 mmHg). For the subsequent management of the patient during ECMO, she was transferred to the ICU of Kyoto Prefectural University of Medicine.

An initial physical examination at our ICU revealed piping sounds in the bilateral lung fields and decreased breath sounds in the left lung field on auscultation. Chest radiography demonstrated atelectasis of the entire left lung (Fig. 1). Her laboratory data were as follows: white blood cell count (WBC), 14,500/mm^3; C-reactive protein (CRP), 0.04 mg/dl; aspartate aminotransferase (AST), 111 IU/l; alanine transaminase (ALT), 97 IU/l; lactic acid dehydrogenase (LDH), 836 U/l; blood urea nitrogen (BUN), 16.3 mg/dl; creatinine, 0.55 mg/dl. After the patient had been transferred to our ICU, the ventilator was set to

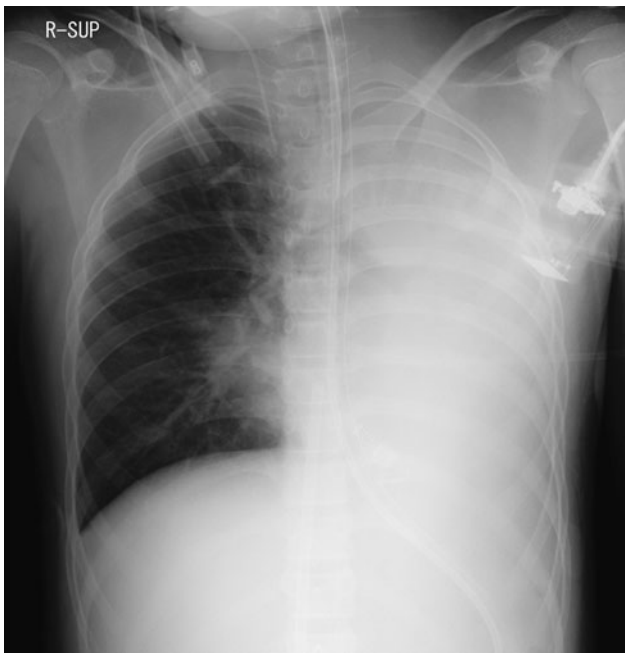

Fig. 1 A chest radiograph showed extensive atelectasis in the left lung and hyperinflation in the right lung

synchronized intermittent mandatory ventilation mode with peak inspiratory pressure of 20 cmH_2O, respiratory rate of 20 breaths/min, and FiO_2 value of 0.6. PEEP was set at 10 cmH_2O. These ventilator settings resulted in a tidal volume of 125 ml. The V–V ECMO system was set at constant blood flow of 1.0 l/min, oxygen flow of 4.0 l/min, and fraction of inspired oxygen of 1.0, which enabled adequate gas exchange. Arterial blood gas analysis produced the following results: pH, 7.54; $PaCO_2$, 44.0 mmHg; PaO_2, 125 mmHg.

A fiberoptic bronchoscopy was subsequently performed, which revealed that large amounts of thick bronchial secretions had occluded the main bronchus. Airway hygiene using fiberoptic bronchoscopy and alveolar recruitment with 10 cmH_2O PEEP resulted in increased tidal volume under pressure control ventilation. To remove the bronchial mucous plugs obstructing the bronchus, aggressive pulmonary hygiene with fiberoptic bronchoscopy was performed once or twice a day. She also received maximal treatment for her asthma; i.e., intravenous aminophylline and corticosteroids and a subcutaneous β_2-adrenergic agonist. On day 6, biphasic cuirass ventilation (BCV) was employed to promote drainage of the mucous secretions, which reduced the patient's atelectasis and improved her oxygenation. Weaning from ECMO was therefore attempted by reducing the blood flow and the fraction of inspired oxygen. On day 7, chest computed tomography (CT) demonstrated remaining atelectasis in the lower lobe of the left lung and middle lobe of the right

lung. At the same time, head CT showed evidence of intracranial hemorrhage. On day 8, her respiratory status progressively improved (Fig. 2), and she was weaned off ECMO after 4 days without any ECMO-related complications, except for the aforementioned intracranial hemorrhage. No change in the ECMO circuit consequent to thrombosis was required. Chest radiography performed on the subsequent day showed progressive resolution of her left lobe atelectasis (Fig. 3). She was finally extubated on day 13. She made a satisfactory recovery with no evidence of neurological or pulmonary complications and was discharged from hospital on day 36.

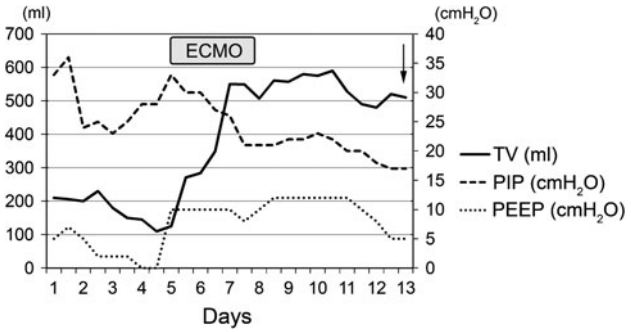

Fig. 2 Time courses of peak inspiratory pressure (*PIP*), tidal volume (*TV*), and positive end-expiratory pressure (*PEEP*). The ventilator was set to pressure control ventilation mode from day 5 onward. As the TV increased, inspiratory pressure setting was progressively decreased. The patient was extubated on day 13 after admission (*down-pointing arrow*). *ECMO*, extracorporeal membrane oxygenation

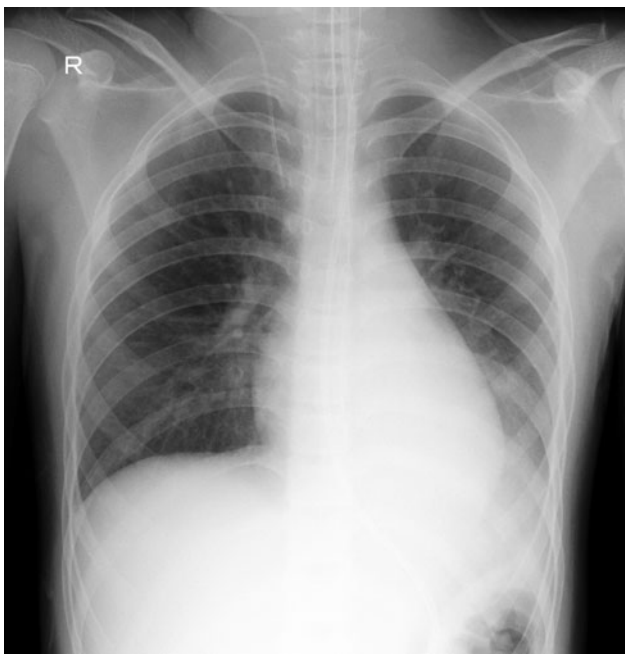

Fig. 3 A chest radiograph showed complete resolution of the left lobar atelectasis

Discussion

Asthma is a common chronic illness that is characterized by reversible inflammation, obstruction, and hyperresponsiveness of the airways. The worldwide prevalence of asthma is increasing and is currently estimated to range from 7 to 10% [7]. ICU admission for asthma is relatively uncommon but remains associated with appreciable in-hospital mortality [2]. The evidence-based therapy for asthma includes supplementary oxygen to achieve an arterial oxygen saturation of 90% or greater, inhaled short-acting β_2-adrenergic agonists, and systemic corticosteroids; a small number of patients with refractory asthma require mechanical ventilation as a life-saving intervention [1]. When status asthmaticus does not respond to this standard therapy, ECMO should be considered as a means of providing temporary pulmonary support to stabilize the patient and improve gas exchange. Several case reports have described the clinical efficacy of ECMO as a treatment for pediatric respiratory failure [3–5]. Additionally, accumulating evidence suggests that ECMO is more effective at improving survival in adults with reversible respiratory failure than conventional ventilation [8]. However, regarding the management of asthma, only a few case reports have addressed the indications for ECMO [9]. Previous reports have shown that the early use of ECMO resulted in a positive outcome in a case of near-fatal status asthmaticus that was unresponsive to conventional treatment and ventilation [10, 11]. In this case, the muscle relaxants administered to facilitate synchrony might have impaired the patient's expiratory muscle strength and exacerbated air trapping and lung hyperinflation by reducing expiratory flow, ultimately leading to hypoventilation. Hence, the patient did not receive any muscle relaxant from day 5 onward. Nevertheless, venovenous ECMO was especially effective because it eliminated the risk of life-threatening hypoventilation and barotrauma. Many patients with severe respiratory failure associated with 2009 influenza A (H1N1), in whom it is extremely difficult to achieve effective alveolar ventilation, have been provided with ECMO support, and patients treated in this manner display a low mortality rate despite the severity of their illness [12, 13]. These observational studies have also confirmed that ECMO is an effective therapy for patients with severe, but potentially reversible, respiratory failure. However, at present there are no evidence-based guidelines as to when to commence ECMO for status asthmaticus. Therefore, specific criteria for determining the necessity of ECMO are required.

Inflammation caused by severe asthma is regarded as one of the main causes of plastic bronchitis. The findings of this case, in which lobar atelectasis developed as a result of mucous plugging of the airways, were suggestive of plastic bronchitis. Plastic bronchitis is characterized by marked obstruction of the large airways by bronchial casts. In a previous study of patients displaying this condition, the underlying disease was asthma or allergic disease in 31% of patients, cardiac defects in 40% of patients, and other diseases in 29% of patients [6]. Preciado et al. [14] reported that bronchoscopic procedures for cast removal were useful in three children who developed recurrent plastic bronchitis after undergoing the Fontan procedure. In this case, aggressive pulmonary hygiene including frequent bronchoscopies was employed to improve the patient's respiratory status.

We conclude that, when patients with potentially fatal asthma develop progressive respiratory distress with lung atelectasis, clinicians should be aware of the possibility of plastic bronchitis and consider bronchoscopic evaluation. In severe cases, ECMO is a suitable treatment option.

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