

When mild asthma requires extracorporeal membrane oxygenation in a young child

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Key words: child; extracorporeal membrane oxygenation; status asthmaticus.

Childhood asthma deaths are rare and the higher death rates are reported in very young children and adolescents (1, 2). While the majority of children with status asthmaticus can be successfully managed with a combination of drugs, progressive respiratory failure may develop. The management of the intubated child with refractory status asthmaticus continues to be a difficult problem that may be complicated by iatrogenic problems and barotraumas. We describe a young child with mild intermittent asthma, who developed life-threatening status asthmaticus refractory to maximal conventional treatment.

Childhood asthma deaths are rare and the higher death rates are reported in very young children and adolescents (1, 2). While the majority of children with status asthmaticus can be successfully managed with a combination of drugs, progressive respiratory failure may develop. The management of the intubated child with refractory status asthmaticus continues to be a difficult problem that may be complicated by iatrogenic problems and barotraumas. We describe a young child with mild intermittent asthma, who developed life-threatening status asthmaticus refractory to maximal conventional treatment (3) and required extracorporeal membrane oxygenation (ECMO) support for survival. A 3-year-old boy presented to a local community hospital with a 24-h history of symptoms of upper respiratory infection and wheezing. His past medical history was notable for two mild wheezing episodes, with no recurrence in the last 12 months.

On arrival at the hospital at 9:00 p.m., the child had moderate respiratory distress and an oxygen saturation (SaO_2) of 89% on air. Lung auscultation revealed diminished breath sounds and diffuse

wheezing. Therapy with oxygen, nebulized albuterol and i.v. methylprednisolone was started, achieving only a transient improvement. A chest radiograph revealed, patchy infiltrates in the perihilar regions, with an area of atelectasis in the right upper lobe. The peripheral leucocyte count was $25\,250\text{ cells/mm}^3$ (85% neutrophils) and C-reactive protein level was 33.8 mg/l. After 8 h, the child was transferred to the paediatric intensive care unit of our department due to increasingly severe respiratory distress.

On arrival at 5:15 a.m., the child looked ill and was agitated with an axillary temperature of 37.7°C , a heart rate of 169 per min, a respiratory rate of 50 per min, an SaO_2 of 88% on air and severe respiratory distress. Arterial blood gas analysis showed a pH of 7.31 and a PaCO_2 of 48 mmHg. Therapy was continued with oxygen, continuous nebulized albuterol, nebulized ipratropium bromide

and i.v. methylprednisolone (1 mg/kg every 6 h). In addition, i.v. cefotaxime and clarithromycin were started. At 6:50 a.m., further deterioration prompted an i.v. infusion with albuterol ($0.2\text{ }\mu\text{g/kg/min}$ up to $2\text{ }\mu\text{g/kg/min}$). At 11:30 a.m., arterial blood gas analysis showed: pH 7.17, pO_2 67 mmHg, pCO_2 74 mmHg; i.v. aminophylline was started and a bolus of i.v. magnesium sulphate (40 mg/kg) was administered.

At 2:00 p.m., because increasingly severe hypoxia and hypercapnia, the child was intubated and started on conventional mechanical ventilation. As his respiratory failure progressed (pH 7.14, PaCO_2 104 mmHg, PaO_2 57 mmHg with FiO_2 100%), the child was switched to high-frequency oscillatory ventilation. No improvement was obtained: the A-a DO_2 (difference between alveolar and arterial O_2) was $>500\text{ mmHg}$ on three consecutive blood samples and the oxygenation index was 85.

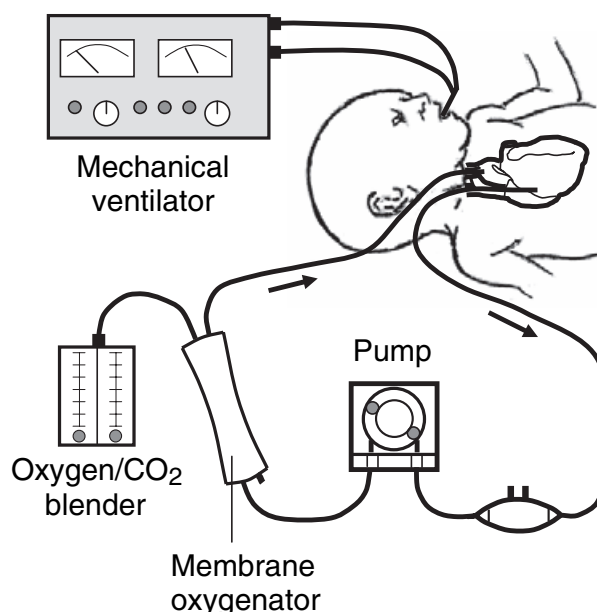


Figure 1. Schematic drawing of a veno-arterial extracorporeal membrane oxygenation circuit. Blood is drained through a catheter inserted into the right internal jugular vein, and after being oxygenated, is returned into the right carotid artery.

After speaking to the family, the right internal jugular vein and common carotid artery were cannulated and veno-arterial ECMO was initiated (Fig. 1). Fiberoptic bronchoscopy revealed a mucous plug in the right upper bronchus, removed by aspiration. Broncho-alveolar lavage fluid (BALF) analysis showed 88% of neutrophils and 8% of eosinophils; BALF culture yielded influenza A virus. The child's respiratory function gradually improved and he was successfully weaned from ECMO after 5 days, when he required an additional 2 days of mechanical ventilation. No major complications occurred during ECMO.

Serial chest radiographs showed progressive normalization. A month later, a chest computed tomography scan revealed normal lungs and bronchi and pulmonary function tests (interrupter resistance technique) showed a normal airway resistance (0.60 kPa/l-s). The range of factors associated with increased risk of asthma death include delay in diagnosis and under-treatment of disease, both long term and in the final acute event (1).

Our patient had no risk factors for life-threatening asthma. He suffered from mild intermittent asthma and the near fatal asthma exacerbation was triggered by a viral respiratory infection. Respiratory viruses are well recognized as major triggers of asthma exacerbations and a viral detection rate of 47% has been reported in children treated for near fatal asthma (4).

Very few data are available on ECMO as a therapeutic option for children with life-threatening status asthmaticus (5, 6). Patients with status asthmaticus die because their condition deteriorates or because they experience treatment-associated complications. The decision to place our patient on ECMO was based on a progressive deterioration of respiratory function despite maximal medical therapy.

This case indicates that even children with severe pulmonary dysfunction related to status asthmaticus may recover within a few days, if they are treated with ECMO, which allows the lungs to rest, together with standard aggressive therapies. However, as significant adverse effects may occur with ECMO, it should

be reserved as rescue therapy when all other therapies fail.

In conclusion, ECMO may represent a rescue therapy for children with status asthmaticus refractory to maximal conventional treatment. In addition, while it is useful for doctors and patients to recognize the features that give certain asthmatic subjects a high-risk profile, physicians should be constantly aware that no asthmatic child is really free of the risk of a life-threatening attack of asthma.

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DRESS syndrome from cefadroxil confirmed by positive patch test

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Key words: cefadroxil; DRESS syndrome; positive patch test.

A 51-year-old man was diagnosed with osteomyelitis, a complication that developed in the aftermath of an orthopedic surgery on his right tibia. He took cefadroxil 500 mg bid for 9 weeks until his admission to our hospital. A week prior to his admission, he had developed fever, myalgia, arthralgia, and skin rash.

Physical examination showed that he had mild fever, generalized maculopapular exanthem, and multiple purpuric patches which gradually developed to exfoliative dermatitis. No lymphadenopathy was found. Pus was found on the gaping wound on his right tibia. Laboratory tests found leucocytosis ($80.43 \times 10^3/\mu\text{l}$), absolute eosinophilia ($33.780/\mu\text{l}$), lymphocytosis ($20.911/\mu\text{l}$), atypical lymphocytes, abnormal liver, and renal function (AST 112.4 IU/l, ALT 57.6 IU/l, BUN 32.9 mg/dl, CRE 1.48 mg/dl), increasing ALP (286 IU/l) and decreasing total protein and albumin (TP 6.23 g/dl, ALB 2.78 g/dl). HBsAg were negative. *Streptococcus epidermidis* was positive on pus culture; however, the blood culture showed no bacteria.

Cefadroxil was stopped; we gave him methylprednisolone equivalent to 1.3 prednisone/mg BW/day, clindamycin 300 mg tid and wound care for a week. The patient's health condition improved remarkably, attested by laboratory tests. He was afterwards discharged from the hospital. The methylprednisolone was continued and tapered-off for a week. Two weeks later, the DRESS syndrome relapsed. Treatment with a similar dosage of methylprednisolone, with a slower gradual tapering-off for 4 weeks was performed until complete recuperation.

The ability of patch testing to confirm cefadroxil DRESS syndrome.