

Successful treatment of fatal asthma combined with a silent chest: A case report

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Abstract

Fatal asthma is a rare and potentially fatal disease. This disease requires suitable treatment to achieve rehabilitation, especially when accompanied by other complications, such as a silent chest and severe bronchial spasm. A 36-year-old man presented with a 10-year history of asthma that broke out into persistent asthma attacks and cardiac arrest, and was accompanied by a silent chest for 18 hours. He recovered and was discharged without any sequelae after being treated by a ventilator, hormones, epinephrine, analgesics, sedation, and muscle relaxants. Comprehensive treatment with a ventilator, hormones, epinephrine, analgesics, sedation, and muscle relaxants has a good effect on fatal asthma combined with a silent chest.

Keywords

Fatal asthma, silent chest, cardiac arrest, analgesic sedation, muscle relaxant, ventilator

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Introduction

Asthma is a chronic inflammatory respiratory disease and is characterized by increasing airway responsiveness, reversible airflow obstruction, chest tightness, and other factors.¹ Asthma affects approximately 300 million people worldwide and is a considerable health burden.^{2,3} Severe asthma comprises only a minority (5%–10%) of the population, but accounts for

a large proportion of morbidity from asthma and health care expenditures.^{4,5} This is because severe asthma cannot only

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be treated by high-dose inhaled corticosteroids or by oral corticosteroid (for at least 6 months per year).⁶ Fatal asthma is a special type of severe asthma, and it is characterized by being combined with cardiac arrest and a silent chest.⁷ Silent chest with a sign of severe bronchial spasm or extensive blockage of a mucous plug results in wheezing and a significant reduction or disappearance in breathing sounds.⁸ Fatal asthma can cause rapid onset of severe hypoxemia, hypoxic brain injury, cardiopulmonary failure, and even cardiac arrest.

We report a case of a 36-year-old man who presented with fatal asthma, cardiac arrest, and 18 hours of a silent chest. The patient recovered and was discharged without any sequelae after being treated by a ventilator, hormones, epinephrine, strong analgesic sedation, and muscle relaxation.

Case presentation

A 36-year-old man presented to our hospital at 15:31 hours on 23 July 2018 because of 3 hours of dyspnea and 1.5 hours of unconsciousness. He had asthma for longer than 10 years and had a drug history, including salbutamol aerosol and theophylline sustained release tablets. An examination showed that he was in a coma, cyanotic, with both pupils dilated, and without a carotid pulse. Urgent chest compression and ventilator-assisted breathing were carried out, and he regained a spontaneous heart rhythm after 2 minutes (ventilator mode: pressure-controlled synchronized intermittent mandatory ventilation [PSIMV], pressure control [PC]=12 cmH₂O, pressure support [PS]=12 cmH₂O, positive end-expiratory pressure [PEEP]=6 cmH₂O, fraction of inspired oxygen [FiO₂]=100%, and respiratory frequency [F]=12 times/minute). Blood gas analysis showed the following: pH=6.98, PCO₂=118 mmHg, PO₂=120.7 mmHg, FiO₂=100%, base excess [BE]=7.4 mmol/L,

lactate level=3.9 mmol/L, glucose level=10.4 mmol/L, and potassium level=6.1 mmol/L. An X-ray chest radiograph and troponin levels showed no abnormalities. He was administered intravenous methylprednisolone (80 mg) and 5% glucose solution (100 mL) with doxofylline (0.2 g), followed by rapid infusion of 1000 mL saline. At 16:00 hours, his oxygen saturation was 60% with no audible breathing sounds or wheezing, while there were obvious airway spasms and a silent chest. The ventilator mode was changed to synchronized intermittent mandatory ventilation (tidal volume [VT]=300 mL, inspiration:expiration=1:2, F=12 times/minute, and PEEP=0 cmH₂O) until the patient's airway pressure was between 45 and 60 cmH₂O. He was instantly administered propofol (3 mg/kg/hour) for sedation, with slow titration of terbutaline (0.25 mg) with 0.9% normal saline (100 mL), intravenous injection of methylprednisolone (160 mg), and infusion of vecuronium bromide (100 mg) with 0.9% normal saline (40 mL) for muscle relaxant. The patient still showed a silent chest, arterial oxygen saturation (SPO₂) had decreased to 50%, and there was pneumothorax with no pulmonary edema. The ventilator mode was changed to PSIMV (PC=20 cmH₂O, PS=20 cmH₂O, PEEP=0 cmH₂O, FiO₂=100%, and F=12 times/minute). After 2.5 hours, blood gas analysis showed the following: pH=6.82, PCO₂=150 mmHg, PO₂=105.5 mmHg, FiO₂=100%, potassium level=7.35 mmol/L, and glucose level=6.5 mmol/L. Additionally, an electrocardiogram showed a wide QRS and high T-wave. Because of respiratory acidosis and hyperkalemia, he was intravenously administered 2 g calcium gluconate, 10% glucose solution (500 mL) with insulin (10 U), and 5% sodium bicarbonate (250 mL). Intravenous methylprednisolone (160 mg), doxofylline (0.2 g), and terbutaline (0.25 mg) were injected again.

The patient's blood pressure had decreased to 70/20 mmHg, although it increased to 110–120/60–70 mmHg after subcutaneous injection of 0.5 mg epinephrine, intravenous injection of 0.5 mg epinephrine, and intravenous infusion of norepinephrine 0.5 µg/kg/minute. This was followed by intravenous infusion of 0.2 µg/kg/min epinephrine and intravenous 0.9% normal saline (500 mL) with 500 mg methylprednisolone. During this treatment, an ice cap was provided to protect the brain, epinephrine was intermittently injected through the skin and a vein, and there was persistent sedation with fentanyl and propofol, as well as vecuronium bromide infusion. The ventilator mode was changed to PSIMV (PC = 20 cmH₂O, PS = 20 cmH₂O, PEEP = 5–8 cmH₂O, FiO₂ = 100%, and F = 12 times/minute). Breathing sounds were heard in both lungs. At 22:00 hours, the ventilator mode was PSIMV with the following settings: PC = 20 cmH₂O, PS = 20 cmH₂O, PEEP = 8 cmH₂O, F = 12 times/minute, FiO₂ = 80%, and SPO₂ = 91%. VT was approximately 200 mL and maintained with 0.3 µg/kg/minute of norepinephrine.

The next day, at 03:00 hours, the patient was injected with norepinephrine (0.1 µg/kg/minute) and epinephrine (0.1 µg/kg/min) to maintain a heart rate of 80 to 100 beats/minute and blood pressure of 100–120/60–70 mmHg. At 08:00 hours, audible breathing sounds were enhanced and wheezing filled both lungs. The ventilator mode was adjusted to PSIMV with the following settings: PC = 20 cmH₂O, PS = 20 cmH₂O, inspiration:expiration = 1:2, PEEP = 12 cmH₂O, VT = 300 mL, FiO₂ = 60%, F = 12 times/minute, and SPO₂ = 93%. Blood gas analysis showed the following: pH = 7.36, PO₂ = 98.2 mmHg (FiO₂ = 60%), PCO₂ = 90.6 mmHg, BE = –3.5 mmol/L, and lactate level = 3.1 mmol/L. At 10:00 hours, wheezing was reduced. Blood gas analysis showed the

following: pH = 7.46, PO₂ = 112.5 mmHg (FiO₂ = 40%), PCO₂ = 60.1 mmHg, SPO₂ = 93%, BE = –2.3 mmol/L, and lactate level = 1.9 mmol/L. The ventilator mode was PSIMV with the following settings: PC = 12 cmH₂O, PS = 12 cmH₂O, PEEP = 8 cmH₂O, and FiO₂ = 40%. At 17.32 hours, the patient appeared to have a tic disorder and hyperpyrexia. An ice cap was provided to protect his brain and for mild hypothermia treatment. He was continually administered fentanyl combined with propofol for sedation and provided mannitol dehydration to decrease intracranial pressure and methylprednisolone (80 mg). Blood gas analysis showed the following: pH = 7.48, PO₂ = 100.8 mmHg (FiO₂ = 40%), PCO₂ = 49.5 mmHg, BE = –3.0 mmol/L, and lactate level = 2.1 mmol/L.

On the third day (08:30 hours), the patient showed a normal temperature, he had regained consciousness, and the tics had disappeared. On the fourth day (08:30 hours), the patient was transferred to the general ward. He was administered methylprednisolone (80 mg, every 6 hours, gradually reduced) and doxofylline (0.2 g, every 12 hours). All of the drugs were stopped after 7 days. On the ninth day, he was fully recovered without any sequelae.

This study was approved by Hebei General Hospital Ethics Committee and the participant signed an approved informed consent form. We also obtained consent for publication of the patient's medical data. All procedures were performed in accordance with the World Medical Association's Declaration of Helsinki.

Discussion

Severe asthma may fall into two categories of difficult-to-treat asthma and severe therapy-resistant asthma.⁶ Fatal asthma is a special type of severe asthma, and it is

accompanied by a silent chest, which may involve wheezing and inaudible breathing sounds because of severe bronchial spasms or extensive mucous blockage.⁸ In our case, the patient had a history of asthma and experienced an abrupt asthma attack, which might have been due to a sensitized state after being exposed to allergens. He experienced difficulty in breathing and lost consciousness when he was exposed to an allergen. Furthermore, a silent chest was diagnosed by the symptoms of inaudible breathing sounds and wheezing, and large resistance to breathing occurred when he was assisted by a simple breathing apparatus.

Mechanical ventilation is an important method for treating fatal asthma. Adequate positive PEEP can help inspiration, change the isobaric point of the small airways, correct ventilation, and improve the patients' blood gas status.⁹ In the present case, the patient showed severe airway spasms, a low tidal volume, and still had a silent chest when the PEEP was 0 cmH₂O. During progress of gradually adjusting PEEP from 5 to 12 cmH₂O, breathing sounds became clearly audible with a considerable reduction in wheezing. Additionally, VT increased from 100 to 300 mL, SPO₂ increased from 50% to 93%, FiO₂ decreased from 100% to 60%, and blood gases were improved.

In the current case, severe airway spasms, bronchial hyperresponsiveness, and a silent chest were the main problems, especially airway spasms. During the patient's 18-hour continuous airway spasms, along with mechanical ventilation, methylprednisolone, epinephrine, doxofylline, terbutaline, and vecuronium bromide were successively used for therapy. Glucocorticoids, which are one of the most effective medicines for asthma, can enhance sensitive resistance of the trachea, inhibit synthesis of acid mucopolysaccharides in the bronchial glands, and relieve

airway obstruction.¹⁰ Methylprednisolone easily penetrates lung tissue, is difficult to degrade, and has a short biological half-life, and thus can be used as a synthetic glucocorticoid. Our patient showed weak sensibility to muscle relaxants, and his symptoms improved after injection of approximately 1000 mg methylprednisolone. Epinephrine can shrink bronchial arterioles to eliminate mucosal congestion and edema, and this can be used to treat asthma.² Low-dose subcutaneous and intravenous infusion of epinephrine are beneficial to the heart and airway expansion when cardiac arrest, anoxia, and acidosis occur. Our patient had subcutaneous and intravenous administration of small doses of epinephrine because of the severe airway spasms and this resulted in hardly inhaling the atomized epinephrine. In principle, this treatment can contract airway mucosal blood vessels, reduce edema of the airway, and expand the bronchus. Terbutaline inhibits mast cell transmitters, increases mucus transport, prevents mucosal edema, and stimulates chloride ion pumps, with minimal side effects.¹¹ Low-dose epinephrine combined with terbutaline for fatal asthma achieved good clinical results in our case. Silent chest is a rare disease that threatens patients' safety and prognosis. Our patient was discharged without sequelae, which might have been due to some protective measures such as analgesia, sedation, and an ice cap.

In conclusion, fatal asthma combined with a silent chest is a rare clinical condition. This condition can be alleviated by comprehensive treatment with a ventilator, hormones, epinephrine, analgesics, sedation, and muscle relaxants.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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