



# Successful adrenaline treatment of perioperative severe bronchospasm combined with a silent lung: two case reports

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**Background:** Silent lung is a rare and potentially fatal disease. It is a critical sign of strong bronchospasm or extensive mucus plug blockage, which can result in the obvious weakening of breathing sounds or even disappearance of breathing sounds. Silent lung has an acute onset and rapid progress, which seriously threatens the life of patients. It needs early diagnosis, timely and effective treatment to reverse the persistent severe bronchospasm of patients. If not handled in time, silent lung can cause rapid onset of severe hypoxemia, hypoxic brain injury, and even cardiac arrest. Few studies have been reported on the causes and specific treatments for silent lungs.

**Case Description:** We report 2 rare cases of silent lung in this article and summarize the pathogenesis, inducing factors, clinical manifestations of perioperative silent lung. We also review the literature and discuss our solutions and propose other possible solutions for the treatment of silent lung emergencies in clinical settings in order to provide reference for clinical practice of anesthesiologists. Of the patients, 1 displayed a sudden decrease in ventilation volume, an increase in airway resistance, and was changed to pure oxygen. The manual ventilation failed, and there was no fluctuation of the thorax and no respiratory sound during auscultation. Cardiopulmonary resuscitation (CPR) was initiated when cardiac arrest was imminent after hypoxia. The other patient had high airway resistance after anesthesia-induced endotracheal intubation, could not be ventilated, and the carbon dioxide (CO<sub>2</sub>) waveform at the end of breathing disappeared.

**Conclusions:** Both patients had severe bronchospasm; that is, silent lung. The 2 patients improved after hand-controlled ventilation and the administration of adrenaline and methylprednisolone, and ultimately recovered ventilation.

**Keywords:** Silent lung; bronchospasm; adrenaline; case report

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## Introduction

Silent lung is an acute and critical sign of bronchial asthma (1). In the perioperative period, the pathophysiological changes and clinical manifestations similar to “silent lung” due to strong bronchospasm caused by various reasons, such as asthma, anaphylaxis, are called “perioperative silent lung”.

The clinical manifestations of silent lung are as follows: airway resistance increases rapidly, and the resistance of manual extrusion of respiratory sac is very high, the thorax has no fluctuation, the respiratory sound of both lungs disappears, the end-tidal carbon dioxide (ETCO<sub>2</sub>) waveform changes (i.e., airway peak pressure increases, and the expiratory phase is prolonged) or disappears, and oxygen

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saturation (SPO<sub>2</sub>) decreases continuously.

For severe bronchospasm, existing reports mostly focus on the treatment of bronchospasm in asthmatic patients. There are many reasons for the silent lung in the perioperative period, and most of the time it is not caused by asthma. The use of salbutamol cannot effectively relieve the silent lung. Once delayed, it will lead to catastrophic results. There is no report on the specific treatment measures for the silent lung of severe bronchospasm in the perioperative period.

Anesthesia related bronchospasm itself may manifest as an event or may be a component of another event, such as an allergic reaction (2). It is characterized by prolonged expiratory time and complete silence during lung auscultation in severe cases. Most types of less severe bronchospasm can be relieved by appropriate treatment; however, once silent lung occurs, it can cause severe hypoxemia due to its acute onset and rapid progress. Thus, the timely diagnosis and treatment of patients can save lives.

In relation to the 2 patients, 1 displayed rapid remission from silent lung after the administration of low-dose adrenaline, and underwent surgery on schedule. While the other patient experienced severe bronchospasm during surgery and cardiac arrest after silent lung and was sent to the intensive care unit (ICU) after cardiopulmonary resuscitation (CPR). After 6 days, the tracheal tube was successfully extubated, and the patient underwent further rehabilitation treatment. Both patients were successfully treated after suffering from silent lung. We present the following case in accordance with the CARE reporting checklist (available at <https://tcr.amegroups.com/article/view/10.21037/tcr-22-617/rc>).

## Case presentation

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Declaration of Helsinki (as revised in 2013). Written informed consent was obtained from the patients for publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

### Case 1

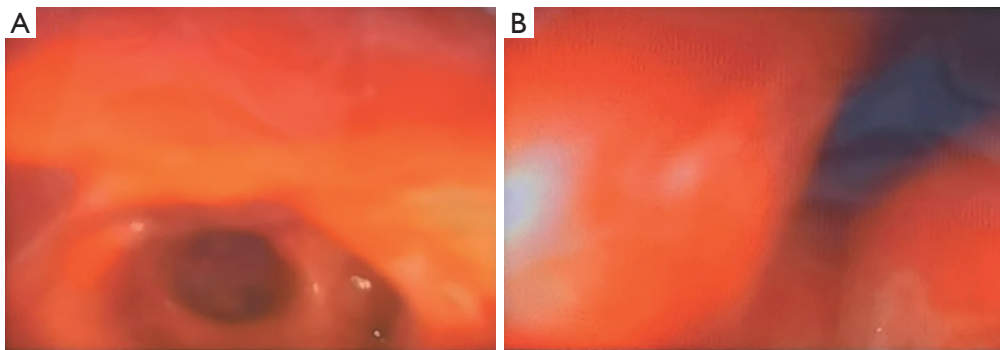
Case 1 was a 45-year-old female patient whose main complaint was a thyroid tumor that had been found at a

physical examination some 10 months ago. The patient was diagnosed with thyroid papillary carcinoma. The doctors planned to adopt the endoscopic transthoracic approach, and perform a right near total thyroidectomy, right recurrent laryngeal nerve exploration, and right central lymph node dissection. In relation to her previous history, the patient had a 3-year history of hepatitis B, and no medication history. The patient had undergone a Cesarean section at a local hospital some 3 years ago. The patient denied any history of smoking, drug use, or food allergies. The chest computed tomography (CT) scan showed no obvious abnormalities in either lung. The patient's preoperative physical examination results were as follows: blood pressure: 115/75 mmHg; heart rate: 78 bpm, and SpO<sub>2</sub>: 99%.

The anesthesia induction was smooth and stable; the enhanced nerve monitoring endotracheal tube was used. During the operation, the surgeon complained that the signal of the nerve monitoring tube was weak, and repeatedly asked for muscle relaxation antagonists to be administered. After the administration of 0.5 mg of neostigmine and 0.25 mg of atropine, the surgeon was able to detect the nerve signal. After 20 minutes, the patient's ventilation decreased, and the patient's airway resistance then increased. The mechanical obstruction factors (i.e., the endotracheal tube and anesthesia machine pipeline) were immediately excluded, and the patient was changed to pure oxygen and placed on manual ventilation. The airway resistance was very high, and the patient could not be ventilated. There was no fluctuation of the thorax, no respiratory sound during auscultation, and the carbon dioxide (CO<sub>2</sub>) waveform of breathing disappeared.

Given the occurrence of a severe airway spasm, 120 µg of epinephrine and 40 mg of methylprednisolone were injected intravenously, but the airway spasm was not relieved. The patient's blood SPO<sub>2</sub> decreased rapidly to 0, and her heart rate decreased rapidly from 85 to 30 bpm. CPR was started, and 1 mg of epinephrine was injected intravenously, and 1 mg of epinephrine was added again some 2 minutes later. Some 5 minutes after CPR had started, the patient's airway pressure decreased, and the blood oxygen gradually increased to 90% after manual ventilation.

The right radial artery was successfully punctured for the manometry and blood gas analysis. It showed metabolic acidosis. Sodium bicarbonate was used to correct the acidosis. Auscultation showed that the respiratory sounds of both lungs were thick. A fiberoptic bronchoscopy showed an obvious edema of the tracheobronchial mucosa (see



**Figure 1** Intraoperative fiberoptic bronchoscopy showed obvious edema of tracheobronchial mucosa.

*Figure 1*). The patient's pupils on both sides were checked and were equal in size and circle (about 4 mm in diameter), and slightly dull in light reflection. The pathological reflex of nervous system was negative. An ice cap was used to protect brain. The patient's oxygenation gradually improved, and the partial pressure of blood oxygen was 128 mmHg. After consultation with the surgeon, the operation was continued to completion.

After stopping the inhalation of anesthetics after the operation, the patient recovered and was able to breathe autonomously, with a tidal volume of about 450 mL and a respiratory rate of 16–18 times/min. The patient's vital signs were stable, but her consciousness was poor. Given that the patient had hypoxic-ischemic nerve injury, the patient was sent to the ICU for further observation and treatment. When leaving the operating room, the patient's bilateral pupils were equal in circle and size (about 3–4 mm in diameter), and sensitive to light reflection. Additionally, a thick respiratory sound was heard in both lungs during auscultation, and oxygen inhalation was administered via an oxygen bag. The patient's results were as follows: SpO<sub>2</sub>: 100%; blood pressure: 110/78 mmHg; and heart rate: 120 bpm.

On the 1st day after the operation, the patient experienced intermittent seizures and had a binocular gaze. As ischemic hypoxic encephalopathy was possible, continuous ice-cap cooling was administered to reduce brain oxygen consumption. Mannitol combined with glycerol sodium chloride dehydration, midazolam, phenobarbital sodium, and sodium valproate were administered to control the seizures, and edaravone and monosialate tetrahexose ganglioside were administered to nourish the nerves. The sedative drugs were stopped on the 4th day after the operation, and the patient's consciousness gradually

improved. On the 5th day after the operation, the patient was conscious and could follow instructions. On the 6th day after the operation, the tracheal intubation was removed, bilateral nasal catheters were given low-flow oxygen, and the blood SpO<sub>2</sub> was able to be maintained at 100%. The patient was then transferred to Neurology Department for rehabilitation treatment and discharged from the hospital.

### Case 2

Case 2 was an 84-year-old male patient whose main complaint was bloody stool for >4 months. The patient was diagnosed with rectal cancer. Da Vinci-assisted radical resection of the rectal cancer was planned. The patient's disease history was as follows: he had had a history of asthma for >20 years, but his condition was well controlled, and he had not experienced any attacks recently. However, in the past 2 months, he had suffered from upper respiratory syndrome, accompanied by shortness of breath and suffocation. The patient denied any history of hypertension, diabetes, or heart disease. He also denied any history of smoking, drug use, allergies to food, and blood transfusion. A chest CT scan showed multiple fibroproliferative foci in the upper lobe of both lungs, the medial segment of the middle lobe of the right lung and the outer basal segment of the lower lobe, and small calcification in the lingual segment of the upper lobe of the left lung, and the aortic arch and left coronary artery, and the pleural thickening and calcification of both lungs. The patient's physical examination results were as follows: blood pressure: 106/57 mmHg; heart rate: 84 bpm; and SpO<sub>2</sub>: 94%. The auscultation left lung breathing sound was clear, but the right lung breathing sound was weak.

Preoperative arterial puncture and catheterization were

performed for real-time blood pressure monitoring, and the patient was monitored for vital signs according to the American Society of Anesthesiologists Standard. General anesthesia was induced via the intravenous injection of methylprednisolone (40 mg), propofol (100 mg), and sufentanil (10 µg), and cisatracurium (14 mg). The intubation was guided by a visual laryngoscope, and the endotracheal tube was fixed after the tube had passed through the glottis at a distance of 22 cm from the incisor. As the patient's airway resistance was high when he was connected to the ventilator, he was changed to manual ventilation. However, there was no ventilation, no fluctuation of the thorax, no respiratory sound during auscultation, and the CO<sub>2</sub> waveform at the end of expiration disappeared. Adrenaline (180 µg) was injected intravenously, and a defibrillator was prepared. The airway resistance decreased gradually, and as the auxiliary ventilation was >300 mL, the patient was changed to controlled ventilation. We communicated with the patient's family members to explain the risks of continuing the operation, and the family members directed us to continue and complete the operation. After the operation, the endotracheal tube was successfully removed, and the patient was returned to the ward.

## Discussion and conclusions

Silent lung is a rare and potentially fatal clinical crisis. Due to severe bronchospasm or extensive mucus obstruction, wheezing and inaudible breathing may occur and develop into silent lung. If the rescue is not timely, the condition can easily evolve into hypoxic-ischemic encephalopathy and even cardiac arrest. Silent lung generally has 2 causes: (I) airway hyperresponsiveness, such as a history of asthma, chronic obstructive pulmonary disease, smoking, respiratory tract infection (3); (II) perioperative trigger factors, including the stimulation of endotracheal intubation, irritant inhalation anesthetics (e.g., desflurane), the stimulation of airway secretions and blood, histamine releasing drugs (e.g., muscle relaxants and morphine), non-steroidal anti-inflammatory drugs, and cholinesterase inhibitors (e.g., neostigmine). Another cause of silent lung is severe bronchospasm caused by an allergic reaction (4). When silent lung occurs during the perioperative period, anesthesiologists must be familiar with the inducing factors, identify the signs, and quickly exclude other factors, such as tracheal tube displacement, sputum blockage, anesthesia machine failure, and too shallow anesthesia.

Both patients with silent lung were successfully treated. In Case 1, the patient's allergy to neostigmine was thought to have resulted in severe bronchospasm and silent lung. Perioperative allergic reactions may manifest as a rash, erythema, angioedema, gastrointestinal manifestations (e.g., nausea, vomiting, and diarrhea), respiratory manifestations (e.g., bronchospasm), and circulatory manifestations (e.g., tachycardia and hypotension). In cases of perioperative hypotension and bronchospasm, allergic reactions should always be considered, unless other causes can be identified. More importantly, when an allergic reaction occurs, a rash, bronchospasm, hypotension and cardiac arrest can be regarded as independent symptoms. The patient may not display all of the above-mentioned symptoms. Bronchospasm can occur as a single manifestation of an allergic reaction (5).

In Case 1, the silent lung was caused by severe bronchospasm. The clinical signs indicated that the patient had reached a grade-III allergic reaction. For allergic reactions > grade III, a first dose of adrenaline (100–200 µg) should be administered, and additional doses should be administered every 2 minutes. For patients undergoing cardiac arrest, epinephrine (1 mg) should be injected intravenously immediately. The β<sub>2</sub> receptor of adrenaline agonism can alleviate bronchial smooth muscle spasms, while the α receptor of adrenaline activation can make the skin, mucosa and visceral blood vessels contract, excite myocardium, increase cardiac output and increase blood pressure, and also inhibit the release of inflammatory mediators. Thus, epinephrine is the first choice for the rescue of anaphylactic shock (6).

The cortisol hormone is a second-line drug for rescuing allergic reactions. Terbutaline can be used in patients with allergic reactions that mainly manifest as bronchospasm. In this case, the patient developed an intractable airway spasm. Initially, the patient was intravenously injected with a small dose of adrenaline, but this did not effectively alleviate airway spasm. The patient's blood SPO<sub>2</sub> decreased to 0, and the patient's heart rate decreased rapidly. We immediately began CPR and increased the dose of adrenaline. The bronchospasm lasted for about 10 minutes before it was gradually relieved. In this case, the timely application of adrenaline won valuable time for the rescue of the patient, and timely protective measures were implemented to treat the cerebral ischemia and hypoxia, including the application of the ice cap. The patient's vital signs were stable, and the patient's spontaneous breathing was good, we did not remove the endotracheal tube immediately, but

administered analgesia, sedation, and decrease intracranial pressure. As the edema peak of hypoxic-ischemic encephalopathy occurred at 24–48 hours, brain oxygen consumption was reduced, the seizures were controlled, and the patient's brain was protected, enabling the patient to wake up safely and smoothly, and regain consciousness. The endotracheal tube was then removed.

In Case 2, the patient had a history of asthma, high airway reactivity and suffered from severe bronchospasm after endotracheal intubation. After administering a small dose of adrenaline, the patient was relieved without any sequelae. After the operation, the endotracheal tube was successfully removed, and the patient was returned to the ward. The duration of silent lung in both Cases 1 and 2 was about 10 minutes. Compared to Case 1, Case 2 experienced no serious consequences. This may be because Case 2 was oxygenated and denitrified before intubation. Sufficient pre-oxygenation can maintain SPO<sub>2</sub>. When ventilation is impossible, the time of hypoxia is greatly reduced when the patient had pre-oxygenation. In Case 1, the inhaled oxygen concentration was 50%, and the SPO<sub>2</sub> decreased rapidly after silent lung. When silent lung occurs clinically, the pure oxygen flow should be immediately increased to 8 L/min, manual ventilation should be performed, oxygenation should be maintained, and lung compliance should be evaluated. Increasing the concentration of volatile anesthetics (sevoflurane and isoflurane) can relax the smooth muscle and reduce airway resistance (7).

β<sub>2</sub> receptor agonists, such as salbutamol aerosol, can be injected into the trachea 8–10 times through an endotracheal tube to dilate the bronchus. Glucocorticoids have anti-inflammatory effects and reduce airway edema. An intravenous drip of hydrocortisone (100 mg) or methylprednisolone (80 mg) can be used. If the bronchospasm is still not relieved, 50 μg of adrenaline can be injected intravenously, and the dose can be increased according to the remission degree of the patient's "silent lung".

During the rescue of these two patients, we used epinephrine in time, which effectively relieved the silent lung of the two patients. But we also have some limitations, for refractory silent lung, such as case 1, if we can use more different measures, such as magnesium sulfate may be effective in refractory bronchospasm. Magnesium sulfate (1–2 g) can be administered intravenously over 20 minutes (8). In addition, high-frequency pulsed ventilation can also be used as a rescue modality for refractory bronchospasm (9). High nasal flow may be

beneficial for respiratory support and oxygenation in emergency patients with severe asthma and hypoxemia (10). It is possible that after using epinephrine in combination with these measures, the patient's rescue can be more successful, and the patient of case 1 may not develop ischemic hypoxic encephalopathy.

In short, during the perioperative period, silent lung directly threatens the life and safety of patients due to difficult ventilation. Anesthesiologists should have the ability to quickly identify and diagnose silent lung during the perioperative period. For refractory bronchospasm, the timely and reasonable use of adrenaline can win valuable time for the rescue of patients.

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## Footnote

*Reporting Checklist:* The authors have completed the CARE reporting checklist. Available at <https://tcr.amegroups.com/article/view/10.21037/tcr-22-617/rc>

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*Ethical Statement:* The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Declaration of Helsinki (as revised in 2013). Written informed consent was obtained from the patients for publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

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