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Silence is No Longer Golden - Reflection on Successful Endotracheal Intubation but Unable to Provide Normal Ventilation - A Case Report

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Abstract

Bronchospasm is an anesthetic emergency, and early diagnosis and timely treatment are crucial for patient recovery. If not reversed in time, it may further progress to silent lung, potentially leading to life-threatening respiratory and cardiac complications. This case report details the crisis encountered by a 54-year-old male after tracheal intubation and proposes some reflections and airway management strategies based on this experience and lesson.

Keywords

Bronchospasm, Airway Hyperresponsiveness, Silent Lung, Hormone Abuse, Endotracheal Intubation, Airway Management Strategies, Anesthesia Complications

Introduction

Silent lung is a rare and potentially fatal disease, characterized primarily by airway hyperresponsiveness, severe bronchospasm, and mucosal edema, which subsequently leads to narrowing of the airway lumen, airflow limitation, and even inability to breathe. Failure to promptly reverse bronchospasm may result in severe hypoxemia, hypoxic ischemic encephalopathy, and even cardiac and respiratory arrest [1]. Although there have been some case reports on this type of patient [1,2], research on early diagnosis and treatment remains scarce.

Case report

A 54-year-old male (160 cm, 71 kg) came to the hospital for treatment after discovering severe hydronephrosis in his left kidney and non-functioning left kidney during a physical examination, intending to undergo left nephrectomy. Upon admission, the doctor conducted a detailed inquiry and physical examination of the patient, who denied any underlying diseases, long-term medication history, or any special events during previous hemorrhoidectomy. Next, the patient underwent auxiliary examinations such as chest CT, electrocardiogram, and blood tests. Only the chest CT indicated mild chronic inflammation in both lungs, and the remaining examinations showed no significant abnormalities.

Anesthesia Induction Process:

After the patient entered the operating room, the electrocardiogram monitoring showed stable vital signs. We noticed that the patient was relatively obese with a short neck and a full moon face appearance.

However, the patient denied any previous use of steroid medications and informed us that all auxiliary examinations were normal. Based on the patient's preoperative physical condition, we planned to perform a routine anesthesia induction: intravenous administration of ciprofol 32.5 mg, sufentanil 25 μg, and rocuronium 60 mg. After the patient's consciousness disappeared, pressure-controlled mechanical ventilation was initiated. The ventilator indicated a low tidal volume of only about 200 ml (support pressure 18–20 mmHg). Considering the patient's body type, we suspected that the poor mask ventilation may be due to a smaller oropharynx, posterior tongue fall, or temporary failure of the muscle relaxant to take effect.

After 90 seconds, with the same support pressure, the tidal volume remained at only about 200 ml, prompting us to perform emergency tracheal intubation. Under the guidance of a video laryngoscope, we clearly saw the endotracheal tube passing through the glottis, with a depth of 22 cm from the incisor. However, we discovered that the ventilator was not working properly. Under manual assisted ventilation, the patient's chest showed no obvious undulations, auscultation of both lungs revealed no respiratory sounds, and there was no clear waveform of end-tidal carbon dioxide on the monitor. We immediately increased the depth of anesthesia by administering 3% sevoflurane and intravenous administration of 5 μg of adrenaline and 40 mg of methylprednisolone. Under continuous manual ventilation with pure oxygen at 6–8 L/min, the patient's chest gradually showed undulations, tidal volume slowly increased, and end-tidal carbon dioxide showed at 50–60 mmHg. Switching to mechanical ventilation, the patient's tidal volume could be maintained at 400–500 ml, with a support pressure of 20–24 mmHg, and auscultation of both lungs revealed a few wheezing sounds. The entire process lasted about 3 minutes, with no severe fluctuations in the patient's circulation and no rash or papules on the body surface. Subsequently, the surgery began, and with continuous infusion of adrenaline at 0.03–0.05 μg/kg/min, mechanical ventilation maintained normal levels.

Tracheal Extubation Process:

Considering the crisis that occurred during the intubation process of the patient, we anticipated that there might also be an increase in airway reactivity and bronchospasm during the extubation process, which could lead to ventilation failure. Throughout the entire surgical procedure, we monitored the patient's depth of anesthesia (bispectral index, BIS). We ensured that the infusion of anesthetic drugs was stopped after all surgical procedures were completed. At this moment, the patient was in a relatively deep sedation state, so we administered sugammadex to reverse the muscle relaxation caused by rocuronium and ensured adequate analgesia for the patient.

When the patient's BIS value exceeded 60, he once again experienced ventilation failure and wheezing throughout the lungs. Fortunately, we called out to the patient, and he opened his eyes and was able to follow instructions. We quickly removed the endotracheal tube and administered ventolin (salbutamol inhalation) to the patient. The patient's rapid breathing gradually improved, and he reported feeling well. Subsequently, we asked the patient again if he had a long-term medication history. The patient told us that due to long-term colds and wheezing, he regularly took oral steroids for treatment, but he did not experience any discomfort at ordinary times.

Discussion and Reflection

Asthma is a disease characterized by varying degrees of airway obstruction, inflammation, and hyperreactivity [3]. Although the patient in this case repeatedly denied having a history of asthma, considering his usual wheezing symptoms and improvement after long-term regular oral hormone therapy, it is highly likely that he concealed his medical history.

According to research reports [4], the probability of bronchospasm occurring in asthmatic patients during surgery is relatively low, and it mostly occurs during the induction and recovery periods. This may be related to the patient's own physical state at that time, such as shallow anesthesia depth, imbalance of vagal-sympathetic nervous system response, and excessive secretions. Due to the airway hyperresponsiveness,

bronchospasm is easily induced by stress (intubation/extubation) reactions, various drugs (such as muscle relaxants, muscle relaxant antagonists, antibiotics), and perioperative complications (such as aspiration, infection, trauma) [3].

In this case, the patient's rapid progression from bronchospasm to silent lung after intubation stress is a poor prognostic indicator. Fortunately, under our diagnosis and treatment, the patient ultimately turned the corner and returned to the ward safely. We believe this is mainly due to the following key points:

1. During the preoperative assessment, we paid close attention to details and immediately thought of possible triggers for the patient. This patient exhibited a typical full moon face, and at the first sign of crisis, we immediately suspected that he had been taking steroids for a long time and had recently stopped taking them.
2. Pure oxygen manual ventilation ensures adequate pre-oxygenation to maintain patient oxygenation, providing sufficient treatment time for the patient.
3. Provide first-line treatment drugs [1] promptly: adrenaline. As a sympathomimetic agent, adrenaline activates α receptors, which not only cause vasoconstriction of skin, mucosa, and internal organs, stimulate myocardial cells to increase cardiac output and elevate blood pressure, but also inhibit the release of inflammatory mediators. Meanwhile, the β2 receptors activated by adrenaline can alleviate bronchospasm.
4. The use of volatile anesthetic sevoflurane: on the one hand, it increases the depth of anesthesia, inhibits the production of stress response, and reduces oxygen consumption; on the other hand, it can relax bronchial smooth muscles and decrease airway resistance.
5. Rapidly acting glucocorticoids provide multiple anti-inflammatory effects.
6. Adequate analgesia during the operation.
7. Monitor the depth of anesthesia to guide extubation.
8. Avoid using cholinesterase inhibitors.
9. Short-acting β-agonists for inhalation. However, it is important to note that these drugs are only recommended for short-term symptom relief or use before known triggers. And because they require active inhalation by the patient, inhaled medications can better penetrate the bronchi to alleviate symptoms, making them more suitable for conscious patients, often resulting in poorer efficacy in intubated patients. This is also the reason why this patient was administered them after regaining consciousness.

Conclusion

Bronchospasm is an anesthetic emergency. If it is not recognized early and treated promptly when it occurs, it may further progress to silent lung, leading to life-threatening respiratory and cardiac complications. Early recognition and diagnosis benefit from paying attention to details during preoperative evaluation and quickly investigating when a crisis occurs. Timely treatment involves avoiding predisposing factors, selecting key medications, and providing adequate care.

Conflict of Interest

The author has read and approved the final version of the manuscript. The author declares no conflicts of interest.

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