Bronchospasm during Anesthesia Induction in A Patient with Undiagnosed Asthma

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ABSTRACT--- In large study of 9287 children undergoing general anesthesia, the incidence of perioperative bronchospasm is 2.1 percent. During anesthesia in patients with well-controlled asthma, airway complications such as bronchospasm are rare. However, poorly controlled asthma is closely related to the pathophysiologic mechanisms of nonallergic and allergic bronchospasm. More than 80% of asthma patients have allergic rhinitis, and 10-40% of allergic rhinitis patients have asthma.

We report a case of a 10-year-old male with undiagnosed asthma who developed bronchospasm during induction of anesthesia. This patient had been treated for allergic rhinitis before surgery. Unexpected bronchospasm occurred immediately after induction of anesthesia and was treated with salbutamol nebulizer and intravenous dexamethasone. In addition, a massive hypotension-suspected anaphylactic reaction occurred and was treated with intravenous epinephrine, after which airway pressure and vital signs improved. For safety, the operation was canceled, anesthesia was discontinued, and the patient was discharged without specific complications. Asthma was diagnosed upon further evaluation two weeks after discharge.

Because this patient had been treated for allergic rhinitis before surgery, asthma should have been diagnosed before surgery. If asthma symptoms before anesthesia were well-controlled using a bronchodilator, steroid, etc., bronchospasm could have been prevented during anesthesia in the current case.

This case suggests that, when possible, asthma should be diagnosed before surgery in allergic rhinitis patients. This case also suggests that anesthesia should be performed after good control of asthma symptoms before surgery to prevent life-threatening perioperative events.

Keywords--- Bronchospasm, Asthma, Allergic rhinitis

1. INTRODUCTION

Many disease processes induce bronchospasm in children [1], and acute asthma exacerbation, acute viral bronchiolitis, and anaphylaxis are important causes [1]. Bronchospasm can occur at any time during anesthesia, but it most often occurs after endotracheal intubation. Although bronchospasm is usually treated without sequelae, it may result in a serious life-threatening perioperative event and significant morbidity and mortality [2, 3]. In fact, 7% of anesthesia-related deaths in France are attributed to intraoperative bronchospasm [4].

Several causes of bronchospasm encountered during the perioperative period have been suggested [2]: 1. An immediate hypersensitivity reaction including IgE-mediated anaphylaxis; 2. A nonallergic bronchospasm triggered by mechanical factors (intubation-induced bronchospasm) in patients with uncontrolled underlying airway hyperreactivity; and 3. A nonallergic pharmacologic-induced bronchospasm (caused by histamine-releasing drugs such as atracurium or mivacurium) in patients with uncontrolled underlying airway hyperreactivity [2]. Poorly controlled asthma may induce perioperative pulmonary complications, a serious life-threatening perioperative event, and significant morbidity and mortality. In contrast, controlled asthma does not promote additional risk [5, 6]. If asthma is diagnosed and treated before surgery and anesthesia, bronchospasm may be prevented [2].

In this case study, the patient was treated for allergic rhinitis 2 months before surgery. Although more than 80% of asthma patients have allergic rhinitis, and 10-40% of allergic rhinitis patients have asthma [2], the patient’s asthma was not diagnosed before surgery. Therefore, we were not fully prepared for a dangerous situation to arise during anesthesia in this patient with uncontrolled asthma. Here, we report a case in which bronchospasm occurred during induction of anesthesia in a 10-year-old male with undiagnosed asthma.
2. CASE

A 10-year-old male patient, 143.4 cm tall and 35.95 kg, was diagnosed with chronic tonsillitis and admitted for a tonsillectomy. This patient was treated for allergic rhinitis before surgery. Two months before surgery, several preoperative evaluations were performed. In the 'MAST immunoglobulin E (IgE) Inhalant' test, total IgE increased to 193.28 IU/ml (reference range <100 IU/ml). Mite-pollen responded as class 4, Birch responded as class 3, Mite-farinae responded as class 3, and House Dust responded as class 2. There were no abnormal findings in other laboratory results, chest X-ray, or electrocardiogram. For allergic rhinitis, oral Montelukast sodium 5 mg (Singulair 5 mg chew tab) was prescribed once daily for 14 days, and spray-type mometasone furoate 0.5mg/ml (Nasonex nasal spray) was prescribed twice a day for two months.

Presurgically, the patient had no specific symptoms and no abnormalities in vital signs. He was moved to the operating room without premedication. We attached noninvasive monitors for blood pressure, ECG standard leads II, and pulse oximeter. Preliminary vital signs were as follows: blood pressure, 114/67 mmHg; heart rate, 156 beats/min; oxygen saturation rate, 98%; and ECG, normal. To calm the patient, 5 minutes of denitrogenation with 100% oxygen was performed through a face mask. We confirmed vital signs of heart rate 124 beats/min and oxygen saturation rate 100%. Thereafter, 0.1 mg of glycopyrrolate, 10 mg of lidocaine, and 70 mg of propofol (Anepol) were administered into the patient’s intravenous line. After approximately 10 seconds, we assessed lid reflex and confirmed loss of consciousness. After confirming face mask ventilation without problems through several attempts at mask bagging, we administered intravenous 15 mg of rocuronium (Rocumeron) and set the sevoflurane concentration as 3 vol.%.

The peak airway pressure increased rapidly 2.5 min after anesthetic induction, so we attempted endotracheal intubation. When the vocal cord was visualized using a video laryngoscope, no airway obstruction was observed, and we performed intubation using an endotracheal tube with an inner diameter of 5.5 mm. After intubation, manual ventilation was attempted, but the tidal volume did not exceed 50 ml, and the airway pressure was also very high, about 30 cm H2O. Wheezing was auscultated on both lungs, and the oxygen saturation rate continued to drop to 60%.

We suspected airway obstruction caused by bronchospasm, maintained FiO2 of 1.0 with mechanical ventilation, and immediately administered intravenous dexamethasone at 5 mg and sprayed two puffs of nebulized salbutamol (Ventolin, β2 adrenergic receptor agonist, bronchodilator). Afterward, the tidal volume was not sufficient, with a respiratory volume of 100 mL at 30 mmHg. After 5 minutes of intubation, blood pressure dropped to 61/28 mmHg with a heart rate of 115 beats per min. We administered 80 mcg intravenous epinephrine, 5 mg dexamethasone, and 2 puffs sprayed nebulized salbutamol (Ventolin). After the trial, the patient’s blood pressure recovered to 85/35 mmHg with a heart rate of 103 beats per min, a respiratory volume of 250-300 ml; the peak airway pressure gradually decreased to 22 cmH2O over 10 minutes. The wheezing that was heard in both lungs during auscultation improved, and respiration and vital signs remained stable.

For safety reasons, we decided to postpone the operation. We confirmed that the TOF rate was stable at 0.95 through a nerve stimulator. To reverse the effect of the neuromuscular blocking agent, we administered intravenous glycopyrrolate of 0.1 mg and pyridostigmine of 5 mg. The patient spontaneously opened his eyes and responded to voice commands. After confirming stabilized spontaneous breathing, extubation was performed.

The patient was transferred to the post-anesthetic care unit and observed by blood pressure and oxygen saturation monitors. We applied facial mask with oxygen of 5 liters per min. One day after anesthesia, the patient showed no specific complications and was released from the hospital. Two weeks after discharge, the patient underwent pulmonary function test and nonspecific bronchial provocation test, based on which he was diagnosed with asthma.

3. DISCUSSION

Allergic rhinitis is characterized by nasal symptoms such as sneezing, nasal blockage, and/or itching of the nose; is either intermittent or permanent; and is classified as mild, moderate, or severe [2]. More than 80% of asthma patients have allergic rhinitis, and 10-40% of allergic rhinitis patients have asthma [2]. Because the presence of asthma must be considered in all patients with allergic rhinitis, it is recommended that those with severe and/or persistent uncontrolled allergic rhinitis be evaluated for asthma before surgery [2].

A previous history of asthma has been identified in 50% and 60% of patients with nonallergic and allergic bronchospasm, respectively. Thus, uncontrolled asthma/chronic obstructive pulmonary disease is closely related to the pathophysiologic mechanisms of nonallergic and allergic bronchospasm, regardless of the stage of anesthesia (induction or maintenance). During induction or maintenance of anesthesia, airway irritation-induced bronchospasm was more common in patients who had one or more predisposing factors such as asthma or bronchitis [2].
Of the 4,000 bronchospasm incidents reported in Australia, perioperative bronchospasm represented 103 (3%) (Reported by Westhope et al.) [7]. Of these 103 incidents, 21% were associated with allergy or anaphylaxis, and 79% were associated with a non-allergic mechanism [2, 7]. Of the 79% nonallergic cases, 44% occurred during induction of anesthesia, 36% during the maintenance phase, and 20% during the emergence/recovery stage. During induction of anesthesia, bronchospasm was mainly related to airway irritation (64%), followed by misplacement of endotracheal tube (17%), aspiration of gastric contents (11%), and other pulmonary edema or unknown causes (8%). During maintenance of anesthesia, bronchospasm was related to anaphylaxis or severe allergy (34%), endotracheal tube malposition (25%), and airway irritation (11%). In contrast to Westhope et al.'s report that 21% of bronchospasm is related to allergy, Fisher et al. reported that bronchospasm occurs as an allergic mechanism in 60% of patients during induction of anesthesia [8].

Here, we did not perform a tryptase test immediately after bronchospasm; tryptase elevation indicates mast cell activation such as that occurring during IgE-mediated anaphylaxis. Because this patient was an allergic rhinitis patient with elevated total IgE in the preoperative test, if the tryptase test had been performed, the serum tryptase level would have been high. In the current case, because there was hypotension, bronchospasm was more likely to be associated with IgE-mediated anaphylaxis. In patients with bronchospasm after commencement of general anesthesia, development of hypotension and oxygen desaturation were 27 and 21 times, respectively, more likely to be associated with IgE-mediated anaphylaxis [8]. However, because the tryptase test was not performed in this patient, we cannot assert that the cause of bronchospasm is IgE-mediated anaphylaxis. It is possible that bronchospasm may be triggered by endotracheal tube insertion because uncontrolled asthma is closely related to the pathophysiologic mechanisms of nonallergic and allergic bronchospasm.

When possible, asthma should be diagnosed and treated before surgery. Because uncontrolled asthma is considered the main risk factor for bronchoconstriction during surgery [5], control of airway inflammation and corresponding asthma symptoms is essential for perioperative and postoperative care [2]. The National Asthma Education and Prevention Program Expert Panel Report 3 recommends that level of asthma control, medication use, and pulmonary function be reviewed before administration of anesthesia [2]. To identify individuals with uncontrolled or poorly controlled asthmatic symptoms, the following should be assessed preoperatively: 1. The degree of asthma control (increased use of inhaled short-acting 2-agonists, previous/current use of inhaled corticosteroids, recent use of oral/injected corticosteroids, recent exacerbations of asthma symptoms, emergency department or hospital visit within the last month) and 2. Other potential risks or complication factors (recent infection of the respiratory tract, previous bronchospasm after intubation, pulmonary complications during/after previous surgical procedure, long-term use of a systemic corticosteroid for severe asthma, associated gastroesophageal reflux or smoking) [2]. If asthma was diagnosed and controlled before surgery in the current case, bronchospasm may have been prevented during anesthesia.

Neuromuscular blocking agents are the most common cause of intraoperative allergic (IgE-mediated) and nonallergic mechanisms and may cause bronchospasm through histamine release [3]. Rocuronium is the most commonly used agent associated with IgE-mediated allergic bronchospasm [9]. Furthermore, cardiovascular signs are the hallmark of severe IgE-mediated anaphylaxis [2, 10]. In patients with neuromuscular blocking agent-induced perioperative anaphylaxis, cardiovascular signs including hypotension are usually the initial clinical event and occur within minutes after drug administration. These cardiovascular signs are related to or followed by bronchospasm in 19 - 40% of patients and are more commonly seen in those with underlying asthma or chronic obstructive pulmonary disease.

Except for rocuronium, the anesthetics (propofol, sevoflurane, and remifentanil) used in this case are considered safe for anesthesia. Although there are a number of case reports of significant bronchospasm with propofol use in patients allergic to the active components of the drug, propofol reduces the bronchospastic response to intubation in both asthmatic and nonasthmatic patients [3]. Because sevoflurane is an effective bronchodilator, it is the preferred anesthetic for patients with asthma [3]. Although morphine can release histamine and cause bronchospasm, synthetic opioids (remifentanil, fentanyl, sufentanil) tend to release much less histamine and have been used safely in asthmatic patients [3].

In this case, the cause of bronchospasm could have been: 1. Uncontrolled asthma; 2. Allergic rhinitis; 3. Rocuronium. Because neuromuscular blocking agents (rocuronium) are a leading cause of perioperative anaphylaxis, and since we did not perform a skin test for rocuronium, bronchospasm caused by rocuronium cannot be excluded. In a recent case report, a 7-year-old boy who suffered from anaphylaxis during induction of anesthesia was subjected to an intradermal test of the drugs used during anesthesia; it was concluded that rocuronium was the cause of anaphylaxis [11].

Anaphylaxis should be treated quickly and aggressively according to established protocols with administration of intramuscular adrenaline, nebulized salbutamol, and fluid resuscitation. Corticosteroids and antihistamines should be considered [12]. We administered intravenous dexamethasone and nebulized salbutamol immediately after symptom onset and intravenous epinephrine, additional intravenous dexamethasone, and nebulized salbutamol 5 minutes after symptom onset. Thereafter, bronchospasm and anaphylactic symptoms gradually improved.
4. CONCLUSION

This case suggests that some patients undergoing anesthesia may have undiagnosed asthma, and the possibility of allergy to anesthesia cannot be ruled out. Even in healthy patients, careful preoperative evaluation should be performed, and anesthesiologists should always be prepared for respiratory complications related to poorly controlled or uncontrolled asthma. This case implies that anesthesia should be performed after asthma symptoms are well-controlled to prevent life-threatening perioperative events.

5. CONFLICT OF INTEREST DECLARATION

No potential conflicts of interest were reported by the authors.

6. REFERENCES


