

Perioperative Anaphylaxis Presented with High Peak Airway Pressure

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It is difficult to diagnose anaphylactic reactions during anesthesia, because the signs and symptoms can be masked by the anesthetized status and sterile draping. The most common causes of perioperative anaphylaxis are neuromuscular blocking agents, latex, and antibiotics. Identifying the offending agent may be difficult because multiple medications are administered in a short period. We report two cases of perioperative anaphylaxis that developed along with high peak airway pressure. Subsequent free circulating immunoglobulin E antibody concentration was elevated in both patients, and the two patients underwent surgery successfully. We present these two cases to alert clinicians to the warning signs for these conditions and to raise awareness of the possibility of hypersensitivity reactions involving any drug or substance used to assure patient safety.

Key words: Anaphylaxis, general anesthesia, immunoglobulin E (IgE), high peak airway pressure

INTRODUCTION

Anaphylactic reactions during anesthesia are rare events that occur in 1/5,000 to 1/20,000 applications of anesthesia^{1,2}. The diagnosis is difficult to make, and treatment needs to start promptly to ensure the best outcome for the patient³. In most cases, a large number of drugs will be administered during anesthesia, and it is not possible to identify the exact allergen⁴. Follow-up investigation is necessary to avoid a potentially life-threatening situation if the patient is reexposed to the offending substance^{5,6}. We report on two patients without a previous allergy history who experienced anaphylaxis and high peak airway pressure.

CASE REPORTS

Patient 1

A 32-year-old man with no history of atopy or drug allergies was admitted to our hospital to undergo a Bankart repair of the right shoulder. Multiple anesthetics, including

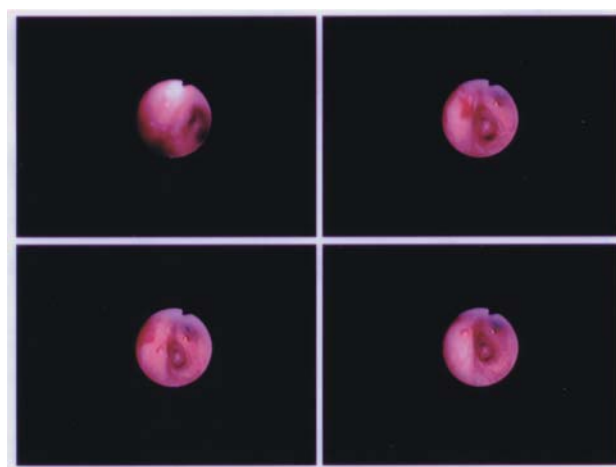


Fig.1 Bronchoscopic examination in Case 1 showed bronchial congestion and mucosa edema with partial lower airway obstruction.

cisatracurium, propofol, fentanyl, and cefazolin, were administered in a 10-minute interval during anesthesia induction. Within 10 minutes of the administration of these medications, the patient developed bronchospasm (high peak airway pressure > 40 cmH₂O) without cutaneous manifestations such as wheal or urticaria. Auscultation of breathing sounds by stethoscope revealed no breathing sounds bilaterally. Bronchoscopic examination showed edematous mucosa with congestion and partial lower airway obstruction (Fig. 1). Surgery was deferred and the symptoms subsided after treatment with epinephrine,

Received: October 31, 2007; Revised: March 27, 2008;
Accepted: September 11, 2008

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diphenhydramine, and hydrocortisone. Free circulating immunoglobulin E (IgE) antibody concentration was elevated (208 IU/ml, normal range < 165 IU/ml). The patient underwent an uneventful surgery and received all the reagents except cefazolin. The rest of the hospital stay was unremarkable and the patient was discharged on postoperative day 10 without any sequelae.

Patient 2

A 33-year-old man with no history of atopy or drug allergies was admitted to our hospital to receive septorhinoplasty. Multiple anesthetics, including cisatracurium, propofol, fentanyl, and cefazolin, were given sequentially within a 10-minute interval during anesthesia induction. The endotracheal tube position was confirmed by auscultation of bilateral breath sounds and capnography. About 30 minutes after endotracheal intubation, the patient developed bronchospasm (high peak airway pressure < 40 cmH₂O) without hypotension or cutaneous manifestations of allergic reactions. Auscultation revealed bilateral diminished breath sounds. The symptoms resolved after treatment with epinephrine, diphenhydramine, and hydrocortisone, and the surgery proceeded and was completed uneventfully. The subsequent free circulating immunoglobulin E (IgE) antibody concentration was elevated (185 IU/ml). The rest of the hospital stay was unremarkable, and he was discharged on postoperative day 5 without any sequelae.

DISCUSSION

Allergic anaphylaxis is most commonly caused by the interaction of an allergen with specific immunoglobulin E (IgE) antibodies, which are present on mast cells and basophils in sensitive individuals. This reaction stimulates the cells to release inflammatory mediators (e.g., histamine, leukotrienes, and tryptase), which cause clinical features involving the respiratory system (bronchospasm and upper airway edema), cardiovascular system (vasodilation and increased capillary permeability), and cutaneous system (wheal and flare)⁷. Some substances that induce allergic anaphylaxis (e.g., dextrans) may do so by releasing IgG antibodies, which produce immune complexes with the antigen (dextran macromolecules) and activate the complement system⁸. In nonallergic anaphylaxis, the clinical features are induced by direct, pharmacological, or “toxic” stimulation of mast cells and basophils, causing them to release inflammatory mediators. Nonallergic anaphylaxis does not involve an immunological mechanism and previous contact with the substance is not necessary^{9,10}.

Anesthesiologists use numerous drugs during the provision of anesthesia. Many of these drugs have side effects that are dose related, and some can lead to severe immune-mediated adverse reactions. Allergic reactions are among the major factors contributing to morbidity and mortality during anesthesia and in the postoperative period¹¹. Anaphylactic reactions during anesthesia are rare events that occur in 1/5,000 to 1/20,000 applications of anesthesia^{1,2}. The agents most likely to cause an anaphylactic reaction are neuromuscular blocking agents (NMBAs), latex, and antibiotics¹². NMBAs account for 50-70% of all cases of generalized reactions during anesthesia¹³. The quaternary ammonium ions shared by NMBAs are the immunodominant epitopes recognized by IgE antibodies¹⁴, leading to significant cross-sensitivity of about 75% among the NMBAs¹². NMBAs can be classified according to their risk of sensitization as high (succinylcholine and rocuronium), medium (pancuronium, vecuronium, and mivacurium), and low (atracurium and cisatracurium)¹³. The second most common agent to cause generalized reactions during anesthesia is latex, which accounts for 10-16% of cases¹³. The third most common cause of generalized reactions during anesthesia are the antibiotic drugs, which account for about 15% of cases¹³. Other causes include the hypnotic induction agents, colloids, opioids, radiocontrast agents, and local anesthetics, which combined account for about 10% of all reactions¹³.

Anaphylaxis is an unanticipated severe allergic reaction, which is often explosive immediately and which can occur perioperatively, especially during a surgical procedure when multiple drugs are administered during the induction of anesthesia. Most anaphylactic reactions are caused mainly by agents administered intravenously¹⁵ and can occur within minutes during anesthesia induction—up to 90% reported in one study¹. Recognizing an allergic reaction during anesthesia may be difficult. Multiple medications are administered sequentially in a short period, making the temporal relationship between drug administration and clinical reactions unhelpful. Anaphylaxis during anesthesia may present in different ways and may be masked by hypovolemia, light or deep anesthesia, or extensive regional blockade. Cutaneous symptoms such as flushing, urticaria and edema are common, but these are usually hidden by surgical drapes during anesthesia and are often unrecognized. Thus, bronchospasm and cardiovascular collapse may be the first recognized signs of anaphylaxis¹⁶. Cardiovascular symptoms often comprise hypotension and tachycardia, which may progress rapidly into severe arrhythmias and cardiovascular collapse if not recognized and treated. These are the most common and serious

symptoms. In some patients, cardiovascular collapse may be the only presenting symptom. Respiratory symptoms such as bronchospasm after the induction of anesthesia occur slightly less often but may predominate in patients with preexisting asthma¹⁵.

High peak airway pressure can be caused by increased airway resistance and decreased thoracic dynamic compliance. In the common three-step checking method, airway problems are explored from the patient's side, breathing circuit, and anesthetic machine in that order. From the patient's side, the common causes are pneumothorax, pulmonary edema, hyperreactive airway, and bronchospasm or secretion, which can be detected with a stethoscope. In the case of pneumothorax, ETCO₂ decreases because of decreased cardiac output. Hemodynamic changes and capillary oxygen desaturation are not constant, but tension pneumothorax with cardiorespiratory compromise can occur. Pneumothorax can be confirmed by the absence of breath sounds on the affected side. Immediate decompression of the chest with a chest tube is mandatory. The most frequent causes of problems involving the breathing circuit are kinking and malposition of the endotracheal tube. This is usually solved after repositioning of the tube to the correct position under the guidance of a fiberoptic bronchoscope. The final check is the anesthesia machine, whose problems can be eliminated by a preoperative test. In our two patients, auscultation revealed bilateral diminished or no breathing sounds, so we used a fiberoptic bronchoscope to examine the patients and found bilateral bronchial mucosa edema and congestive changes with partial lower airway obstruction.

Ideally, all patients experiencing a moderate or severe anaphylactic reaction during anesthesia should be followed up with immediate blood tests and a secondary check with allergy testing¹⁷. In the hours following an anaphylactic reaction, blood samples should be analyzed for serum tryptase or IgE. The secondary investigation comprises the skin prick test, intradermal test, and supplementary *in vitro* tests (e.g., basophil allergen challenge tests) if necessary. The type of secondary investigation may differ between hospitals depending on experience and available resources. The purpose of the follow-up is to identify the drug or substance responsible and the mechanism behind the reaction to make subsequent anesthesia for that patient as safe as possible. In our hospital, only IgE antibodies analysis is available. In our country, we must document such reactions in interhospital referrals for follow-up investigation to avoid a potentially life-threatening situation caused by reexposure to the offending substance.

In conclusion, physicians should be alert to the possibil-

ity of an allergic reaction when a patient develops a sudden onset of high peak airway pressure during general anesthesia. We present these case studies to alert surgeons to the warning signs of these conditions. Healthcare providers should be aware of the possibility of hypersensitivity reactions involving any drug or substance administered before and during surgery and should use this information to assure the safety for patients.

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