

Anaphylaxis in the operating room treated with an anaphylaxis response kit: A case report

ABSTRACT

Anaphylaxis is a rapidly fatal complication of anesthesia, and it needs to be appropriately diagnosed and treated by anesthesiologists. A 37-year-old man underwent surgery for pneumothorax under general anesthesia. Postoperatively, the train-of four count was four, and sugammadex was administered. Soon after, the patient's oxygen saturation and blood pressure decreased, and skin flushing was noted. We suspected anaphylaxis and administered adrenaline. Once the blood pressure normalized, we administered hydrocortisone, famotidine, and d-chlorpheniramine maleate. Thereafter, his condition stabilized, he recovered well, and he was discharged. Blood samples obtained after the onset of the episode indicated anaphylaxis, and a prick test performed 10 weeks postoperatively confirmed sugammadex as the causative agent. We report a case of sugammadex-induced anaphylaxis, which we could properly diagnose and treat because an anaphylaxis kit was available in our department.

Key words: Anaphylaxis, postoperative diagnosis, shock

Introduction

Anaphylaxis is a hypersensitivity reaction that can cause systemic allergic symptoms because of invasion by allergens. In 2021, Japanese Society of Anesthesiologists developed a practical guide for anaphylaxis during anesthesia.^[1] Drug-induced anaphylaxis can rapidly develop, with the speed depending on the severity of the anaphylaxis.^[1] Therefore, the anesthesiologist should immediately begin treatment and subsequently make the diagnosis. We report a case of sugammadex-induced anaphylaxis where we could respond quickly as an anaphylaxis response kit was available at our department.

Written patient consent was obtained for the publication of this report, which was prepared according to the CARE guidelines.

Case History

A 37-year-old man (height: 179 cm, weight: 63 kg), with no comorbidities and no history of allergies or surgery presented to our hospital with dyspnea. Left-sided pneumothorax was diagnosed and thoracic drainage was performed. However, because of persistent leakage, thoracoscopic surgery was scheduled.

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Intradermal infiltration anesthesia was performed with 0.5% lidocaine to secure the peripheral venous route, and general anesthesia was induced with remifentanyl (0.47 µg/kg/min), propofol (90 mg), and rocuronium (50 mg). Anesthesia was maintained with desflurane (4–5%), remifentanyl (0.08–0.3 µg/kg/min), and fentanyl (500 µg). Before the surgery ended, acetaminophen (1000 mg) was administered, and an intercostal nerve block was performed by the surgeon using 20 mL of 0.375% ropivacaine. After the surgery ended, sugammadex (130 mg) was administered after confirming a train-of-four count of four by neuromuscular monitoring. Four minutes after sugammadex administration, the patient's oxygen saturation decreased to 82%, followed by a decrease in blood pressure to 51/30 mmHg. Ephedrine (15 mg) and phenylephrine (0.3 mg) were administered, but the blood pressure did not increase; it was 46/26 mmHg. The peak airway pressure increased from 18 to 28 cmH₂O, conjunctival edema and skin flushing were observed, and we strongly suspected anaphylactic shock. The patient was ventilated with 100% oxygen and adrenaline (0.1 mg) was intravenously injected three times with blood pressure monitoring.

The patient's blood pressure was subsequently stabilized, and he was intravenously administered hydrocortisone (200 mg), famotidine (20 mg), and d-chlorpheniramine maleate (5 mg). Eighteen minutes after anaphylaxis onset, blood samples were drawn for plasma tryptase and histamine tests. The patient was extubated after the cufflink cuff-leak test result was negative and transferred to the critical care unit. His progress in the ward was uneventful and he was discharged 5 days postoperatively. The diagnosis of anaphylaxis was confirmed based on the plasma tryptase and histamine levels measured 18 min and 24 h after onset [Table 1]. We performed prick tests for lidocaine, propofol, ropivacaine, acetaminophen, rocuronium, sugammadex, histamine, and saline, but not for opioids, 10 weeks postoperatively. Fifteen minutes after injection, 4 × 4 mm wheals appeared for histamine and sugammadex [Figure 1], and sugammadex was diagnosed as the causative agent.

Discussion

The frequency of perioperative anaphylaxis rate in Japan has not changed much from 2012 to 2016,^[1] but the overall rate of emergency department visits for anaphylaxis in Europe has risen.^[2] The causes of perioperative anaphylaxis

also differ between Western countries and Japan; the most frequently reported agents in Western countries are, in order, neuromuscular blockers, latex, antibiotics, plasma substitutes, and morphine-like substances,^[3] whereas those in Japan are sugammadex, rocuronium, cefazolin, and antibiotics other than cefazolin.^[4] In 90% of cases, perioperative anaphylaxis occurs during anesthesia induction,^[1] but it is highly likely to occur before or after extubation because of the high rate of sugammadex use in Japan.

For definitive diagnosis, it is useful to note the increase in plasma tryptase and histamine levels during the early stages of anaphylaxis.^[1] As plasma histamine levels return to normal within 15–30 min of anaphylaxis onset, blood samples for hematological examinations should be collected within 5–10 min of onset to detect increased histamine levels. However, these examinations are often conducted at a subcontracted laboratory. Therefore, a system for requesting tests in advance should be established in Japan to enable prompt performance of these tests. Our hospital has an anaphylaxis response kit in the operative department, which contains vacutainer tube, test request forms, hydrocortisone, famotidine, and d-chlorpheniramine maleate along with a paper describing the diagnosis and treatment process. When the anesthesiologist in charge of the case patient called for assistance, he requested to bring this kit from the central storage area of our department, so that treatment and diagnosis could be performed promptly.

In patients with drug-induced anaphylaxis, identifying the causative agent is important for when the patient subsequently undergoes anesthesia. Skin tests such as the prick test should be performed 4–6 weeks after anaphylaxis



Figure 1: Skin test results. Arrows indicate the positive control histamine (1) and sugammadex 10 mg/ml (2) induced wheals. Other negative tested drugs were lidocaine 10 mg/ml, propofol 10 mg/ml, ropivacaine 10 mg/ml, acetaminophen 10 mg/ml, rocuronium 10 mg/ml, sugammadex 1 mg/ml, sugammadex 0.1 mg/ml, and saline

Table 1: Laboratory findings

	Tryptase (µg/l)	Histamine (ng/ml)
18 min after onset	58	305
24 h after onset	2.9	0.98

Plasma tryptase and histamine levels 18 min and 24 h after anaphylaxis onset

onset to avoid false-negative reaction.^[3] Performing skin testing later than the recommended can lead to false-negative results.^[5] However, many patients with a history of anaphylaxis induced by muscle relaxants show positive skin tests even after several years.^[6] Therefore, skin testing may be worthwhile even after the recommended period.

Conclusion

When anaphylaxis occurs, the anesthesiologist must be able to quickly treat the patient and determine the diagnosis, considering future surgical procedures that the patient might undergo. The availability of an anaphylaxis response kit will enable rapid diagnosis and treatment.

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Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initials will not be published and due efforts will

be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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