Scholars Journal of Medical Case Reports **ISSN 2347-6559 (Online)**
Sch J Med Case Rep 2016; 4(6):453-455 **ISSN 2347-9507 (Print)**

(An International Publisher for Academic and Scientific Resources) DOI: 10.36347/sjmcr.2016.v04i06.028

Improvement of suspected rocuronium-induced anaphylaxis after sugammadex administration: A case report

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Abstract: Although rare, anaphylactic and anaphylactoid reactions during anesthesia may result in fatal outcomes. A 26-year-old male patient was scheduled to undergo septoplasty to correct a septal deviation. The patient's preoperative medical history and laboratory findings revealed no specific abnormalities. Anesthesia was induced with propofol and rocuronium. Following the intravenous injection of additional rocuronium, hypotension, tachycardia, and pink frothy secretions within the endotracheal tube were observed. The patient's vital signs and pulmonary edema improved after the administration of sugammadex.
Keywords: anaphylaxis; rocuronium; sugammadex.

INTRODUCTION

Anesthesiologists must always consider the possibility of anaphylaxis, as it can occur as a result of factors unrelated to anesthesia, such as various types of anesthetics used during surgery, as well as exposure to blood and latex [1]. Neuromuscular relaxants are known to be the most frequent cause of anaphylactic reactions during surgery, and rocuronium is one frequently used neuromuscular relaxant [2]. Sugammadex, which has been recently introduced, selectively binds rocuronium to reverse neuromuscular blockade [3]. The author reports a case of a severe hemodynamic reaction that was suspected to be anaphylaxis following the injection of rocuronium bromide for induction of anesthesia, which was successfully reversed by the administration of sugammadex.

CASE REPORT

A healthy 26-year-old male patient (78 kg, 178 cm) was scheduled to undergo septoplasty for the correction of a septal deviation. The patient had no abnormal medical history or family history, and had no atopy or hypersensitivity to drugs or food. Preoperative blood tests, biochemical tests, urinalysis, electrocardiogram (ECG), and chest x-ray revealed no abnormal findings. For general anesthesia, the patient received an intramuscular injection of 0.2 mg glycopyrrolate as premedication 30 minutes prior to arrival in the operating room (OR). After arrival in the OR, the patient was attached to monitoring devices to monitor ECG, heart rate, noninvasive blood pressure, pulse oximeter, and end tidal carbon dioxide (CO_2). Immediately prior to the induction of anesthesia, the patient's vital signs were as follows: blood pressure of 132/83 mmHg, heart rate of 87 beats/minute (bpm), and oxygen saturation of 99%. Anesthesia was induced by the slow intravenous (IV) injection of 150 mg propofol, and 50 mg rocuronium was IV injected after confirming the patient's loss of consciousness. To minimize hemodynamic changes during endotracheal intubation, remifentanil was continuously infused and manual ventilation was performed with 100% oxygen and 3 vol% sevoflurane prior to intubation. After intubation, the patient's blood pressure was 85/60 mmHg, heart rate was 111 bpm, and oxygen saturation was 100%. An additional 10 mg rocuronium was injected prior to surgery as the patient showed movement. His blood pressure was maintained at 70–90/50–60 mmHg, and heart rate at 100–130 bpm, so we stopped the continuous injection of remifentanil and instead continuously injected phenylephrine. However, the patient's blood pressure dropped to 65/46 mmHg while his heart rate increased to 140 bpm; maximum inspiratory pressure (airway pressure) gradually increased to 29 cmH_20 while oxygen saturation gradually dropped to less than 85%, at which point we alerted the surgeon that the surgery could not proceed further. We suspected that the patient was experiencing a hypersensitivity reaction that occurred following the injection of rocuronium, so we immediately stopped the administration of sevoflurane and provided 5 L/min of 100% O_2, fluid, and 0.1 mg phenylephrine, but the blood pressure remained low. Airway pressure increased to 32 mmHg, and pink frothy secretions were observed within the endotracheal tube; these were removed via endotracheal suction. We immediately injected epinephrine 0.03 mg and solumedrol 125 mg, at which point the blood pressure was 80/40 mmHg with a heart rate of 140 bpm and oxygen saturation of 92%. We determined this phenomenon to indicate a

hypersensitivity reaction to rocuronium, so we injected 600 mg (7.6 mg/kg) of sugammadex for reversal of neuromuscular blockade. About 3 minutes after the injection of sugammadex, the patient recovered spontaneous breathing with a blood pressure of 115/70 mmHg, heart rate of 98 bpm, and oxygen saturation of 98%, and the pink frothy secretions within the tube disappeared over time. After the patient's vital signs stabilized, we stopped injections of all drugs and transferred to the patient to the intensive care unit (ICU) without extubation. The patient's vital signs continued to demonstrate stability in the ICU, and pulmonary edema also was reduced. The patient was extubated and transferred to the general ward, and he was discharged the following day without complications. The patient was scheduled for skin prick and intradermal testing as an outpatient in the dermatology department, but was lost to follow-up.

DISCUSSION

The author experienced a case of anaphylaxis accompanied by severe hypotension, tachycardia, and lung inflation impairment after induction of anesthesia in a young male patient.

Whereas the main symptoms of anaphylactic reactions are hypotension, cardiovascular collapse, bradycardia, and bronchospasm, those of anaphylactoid reactions mostly involve skin responses to a much milder degree [4].

Muscle relaxants are the most frequent cause of anaphylactic or anaphylactoid reactions during anesthesia [4]. Among numerous muscle relaxants, succinylcholine is known to be the most common cause of anaphylaxis, followed by rocuronium and vecuronium [5]; however, with the reduced popularity of succinylcholine, rocuronium has been the most common cause in recent cases [1]. Quaternary ammonium ions are considered the major epitopes in muscle relaxant-induced anaphylaxis [6]. In the present case, the blood pressure dropped following the injection of rocuronium for intubation, which we determined to be due to remifentanil; however, we determined the patient to have rocuronium-induced anaphylaxis because the patient's blood pressure dropped and heart rate and maximum inspiratory pressure increased following the injection of additional rocuronium as the patient showed movement before surgery.

Sugammadex, an antagonist of neuromuscular-blocking drugs, binds rocuronium to form a complex that reverses the neuromuscular blockade [7]. It is predicted that the side chains of sugammadex electrostatically interact with rocuronium's quaternary ammonium ions to form a rocuronium-sugammadex complex, which removes the epitopes of quaternary ammonium ions of rocuronium molecules [8]. We considered this possibility in the present case and thus injected 600 mg sugammadex (7.7 mg/kg).

Diagnostic methods include taking blood samples during anaphylactic or anaphylactoid reactions to measure tryptase, histamine, complement, and IgE levels, and performing skin prick and intradermal testing six weeks after the event [9].

In this case, we could not perform a blood test during surgery, and intended to perform the skin prick test and intradermal test six weeks later, but lost track of the patient during follow-up. We were planning to identify the exact cause of anaphylaxis through skin prick and intradermal testing and examine cross-reactions with other muscle blockers as rocuronium was suspected to be the causative agent. However, the patient did not show up for his appointment, preventing us from making a definitive diagnosis and examining any cross-reactions.

The treatment of anaphylaxis primarily involves correcting hypoxia, further suppressing chemical mediated materials, and supplementing intravascular volume. First, 100% oxygen is administered, and fluid as well as epinephrine is immediately injected to increase blood pressure [11]. However, Due to the increased permeability of capillary when anaphylaxis occurs, edema can be seen to the lung or respiratory tract if a massive amount of fluid therapy is given, so the presence of the pulmonary edema must be assessed through chest x-ray prior to extubation.

In the present case, the exact reason why sugammadex resulted in stabilization of the hemodynamic state of the patient is unknown. Presumably, as previously mentioned, the sugammdex and rocuronium complex might have inhibited further secretion of vasoactive mediators, or sugammadex might have reversed the muscular blockade, resulting in increased muscle tone, which in turn promoted venous return and recovery of cardiac output [12].

CONCLUSION

In conclusion, this case showed that the patient's hemodynamic state which results from suspected rocuronium-induced anaphylaxis has been improved after the injection of sugammadex. Although the exact mechanism or reason behind this phenomenon is unclear, sugammadex could be an alternative when suspected rocuronium-induced anaphylaxis does not respond to traditional treatment.

REFERENCES

1. Laxenaire MC, Mertes PM, Groupe d'Etudes des R éactions Anaphylactoïdes Peranesthésiques; Anaph ylaxis during anaesthesia. Results of a two-year sur vey in France. Br J Anaesth, 2001;87(4):549–558.
2. Fisher MM, Doig GS; Prevention of anaphylactic r eactions to anaesthetic drugs. Drug Saf, 2004;27(6)

:393–410.
3. Naguib M; Sugammadex: another milestone in clin ical neuromuscular pharmacology. Anesth Analg, 2 007;104(3):575–581.
4. Mertes PM, Laxenaire M-C, Alla F, Groupe d'Etud es des Réactions Anaphylactoïdes Peranesthésiques ; Anaphylactic and anaphylactoid reactions occurri ng during anesthesia in France in 1999-2000. Anest hesiology, 2003;99(3):536–545.
5. Harboe T, Guttormsen AB, Irgens A, Dybendal T, Florvaag E; Anaphylaxis during anesthesia in Nor way: a 6-year single-center follow-up study. Anest hesiology, 2005;102(5):897–903.
6. Matthey P, Wang P, Finegan BA, Donnelly M; Roc uronium anaphylaxis and multiple neuromuscular b locking drug sensitivities. Can J Anaesth,2000;47(9):890–893.
7. Bom A, Bradley M, Cameron K, Clark JK, Van Eg mond J, Feilden H, et al; A novel concept of reversi ng neuromuscular block: chemical encapsulation of rocuronium bromide by a cyclodextrin-based synth etic host. Angew Chem Int Ed Engl, 2002;41(2):26 6–270.
8. Kawano T, Tamura T, Hamaguchi M, Yatabe T, Y amashita K, Yokoyama M; Successful management of rocuronium-induced anaphylactic reactions with sugammadex: a case report. J Clin Anesth, 2012;2 4(1):62–64.
9. Watkins J; Investigation of allergic and hypersensit ivity reactions to anaesthetic agents. Br J Anaesth, 1987;59(1):104–111.
10. Mertes PM, Demoly P, Malinovsky JM; Hypersens itivity reactions in the anesthesia setting/allergic re actions to anesthetics. Curr Opin Allergy Clin Imm unol, 2012;12(4):361–368.
11. Heier T, Guttormsen AB; Anaphylactic reactions d uring induction of anaesthesia using rocuronium fo r muscle relaxation: a report including 3 cases. Act a Anaesthesiol Scand, 2000;44(7):775–781.
12. Motamed C, Baguenard P, Bourgain JL; Possible m itigation of rocuronium-induced anaphylaxis after a dministration of sugammadex. J Anaesthesiol Clin Pharmacol, 2012;28(1):127–128.