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 (CO2). 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 cmH2O 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% O2; 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 solomedrol 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...
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 anaphylactic or anaphylactoid reactions. Furthermore, rocuronium might have reversed the muscular blockade, resulting in increased muscle tone, which in turn promoted venous return and recovery of cardiac output [12].

In conclusion, this case showed that 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.

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