Perioperative Precision Medicine
ISSN: 2957-5443

Zentime

Case report https://doi.org/10.61189/655401cvkjpp

Rescue of rocuronium-induced refractory anaphylaxis with sugammadex in a laparoscopic hysterectomy: A case report

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Declaration of conflict of interest: None.

Ethics approval and consent to participate: This case report was approved by the Ethics Committee of Birat Medical College (Ref: IRC-082/083), and written informed consent was obtained from the patient.

Received June 24, 2025; Accepted August 29, 2025; Published September 30, 2025

Highlights

- Anaphylaxis is a potentially life-threatening event during general anesthesia, with a rare incidence estimated at 1 in 10,000 to 1 in 20,000 cases.
- Neuromuscular blocking agents, antibiotics, and latex are the primary triggers of perioperative anaphylaxis, with rocuronium being one of the most commonly implicated drugs.
- Sugammadex is a valuable drug for reversing rocuronium-induced refractory anaphylaxis.

Abstract

Neuromuscular blocking agents, particularly rocuronium, are a major cause of perioperative anaphylaxis. This report presents a rare and severe case of rocuronium-induced anaphylaxis in a 50-year-old woman undergoing an elective total laparoscopic hysterectomy at a tertiary care center in Eastern Nepal. Despite multiple doses of intravenous epinephrine, the patient did not achieve adequate hemodynamic recovery. However, a single bolus of sugammadex resulted in the rapid and complete resolution of symptoms. This case highlights the potential of sugammadex as a life-saving adjunct in treating neuromuscular blocking agents-induced anaphylaxis that is unresponsive to standard interventions.

Keywords: Rocuronium, sugammadex, anaphylaxis, epinephrine, refractory

Introduction

Anaphylaxis is a potentially life-threatening event following general anesthesia, with a rare incidence estimated to be between 1 in 10,000 and 1 in 20,000 cases [1]. Neuromuscular blocking agents (NMBAs), antibiotics, and latex account for the majority of perioperative anaphylactic reactions, with rocuronium being one of the most frequently involved agents [2]. NMBA-induced anaphylaxis develops rapidly, typically within minutes after drug administration, and manifests as cardiovascular collapse, bronchospasm, urticaria, or angioedema [3]. Hypersensitivity reactions to rocuronium, a non-depolarizing aminosteroidal NMBA, occur due to the presence of quaternary ammoniums, which are strong allergens [4]. Standard treatment for anaphylaxis focuses on immediate epinephrine administration (intramuscular or intravenous), fluid replacement, corticosteroids, and antihistamines [5].

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Table 1. Timeline of Events and Vital Sign Changes

Time (min)	MAP (mmHg)	SpO_2 (%)	HR (bpm)
0 (Baseline)	85	98	75
2 (Induction)	80	97	89
5 (5 minutes after rocuronium administration)	63	94	116
10 (10 minutes after rocuronium administration)	50	86	133
15 (15 minutes after rocuronium administration indicating severe reaction)	40	80	142
20 (Post-sugammadex)	60	90	110

Note: MAP, mean arterial pressure; SpO_2, peripheral capillary oxygen saturation; HR, heart rate.

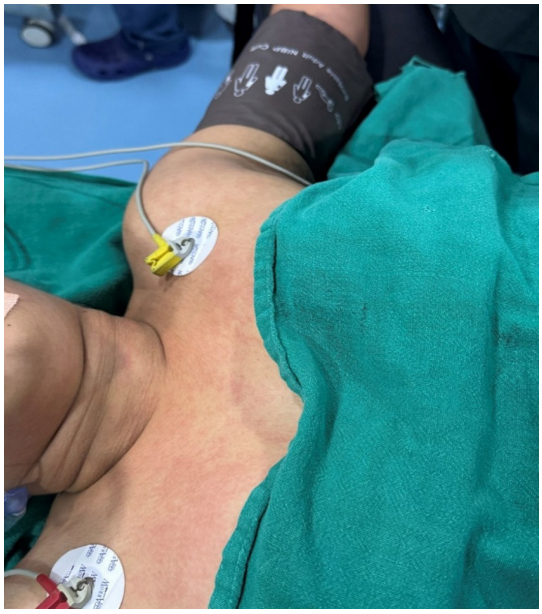

Figure 1. Erythematous rashes over the chest and arm.

However, not all cases respond to first-line therapy. Sugammadex, a modified form of gamma-cyclodextrin, has been investigated as a reversal agent for aminosteroidal neuromuscular blockade, with recent interest in its application for NMBA-induced anaphylaxis [6]. By binding free circulating rocuronium molecules, sugammadex may accelerate the resolution of symptoms [7]. This case presents a rare instance of rocuronium-induced anaphylaxis that was unresponsive to epinephrine but successfully reversed with sugammadex.

Case presentation

A 50-year-old woman weighing 65 kg with ASA physical status, I underwent an elective total laparoscopic hysterectomy for abnormal uterine bleeding. She had no significant medical history, drug allergies, or prior surgeries. Laboratory data, including complete blood count, renal and hepatic function, electrolytes, ECG, and chest X-ray, were all within normal limits.

Upon entering the operating room, standard ASA monitoring was initiated. Pre-anesthetic medications included intravenous glycopyrrolate (0.2 mg), midazolam (1 mg), and fentanyl (100 μg). The patient received intravenous propofol (120 mg) and rocuronium (50 mg). Shortly after rocuronium administration, erythematous rashes appeared on the chest and neck, accompanied by hypotension (mean arterial pressure [MAP] 63 mmHg), tachycardia (heart rate [HR] 116 bpm), and oxygen desaturation (SpO_2 94%) (**Figure 1**). Within one to two minutes, her condition worsened significantly, with MAP decreasing to 40 mmHg, HR increasing to 142 bpm, and SpO_2 dropping to 80%. These signs were consistent with severe anaphylaxis.

Despite timely intravenous fluid resuscitation and a total of 300 μg epinephrine, the patient's hemodynamic instability and hypoxia persisted, rendering the anaphylactic reaction refractory. Given the temporal relationship with rocuronium administration and the failure of conventional therapy, sugammadex was considered as a potential rescue therapy.

Treatment: At 15 minutes post-induction, 200 mg intravenous sugammadex was administered. Within three minutes, her MAP increased to 60 mmHg, SpO_2 improved to 95%, and HR decreased to 110 bpm. Simultaneously, the erythematous rashes and respiratory symptoms rapidly resolved rapidly. Additional treatments, including 100 mg intravenous hydrocortisone and 10 mg chlorpheniramine, were administered to further manage the reaction. The timeline of events and changes in vital signs are presented in **Table 1**.

Due to the severity of the reaction, the surgery was postponed. The patient was transferred to the intensive care unit for close monitoring and did not experience any further complications. She was discharged in good condition on the third postoperative day. A skin prick test six weeks later confirmed rocuronium as the causative agent.

Discussion

This case presents a rare and severe episode of rocuronium-induced anaphylaxis, which was refractory to conventional epinephrine treat-

ment. It highlights the challenges in managing severe hypersensitivity reactions and underscores the emerging role of sugammadex as a potential rescue agent.

Perioperative anaphylaxis is a critical emergency, with NMBAs being the most frequent cause, responsible for 50%-70% of reactions [8]. Rocuronium, a steroidal NMBA, is particularly implicated due to its quaternary ammonium ion, which can trigger IgE-mediated cross-linking on mast cells and basophils in sensitized individuals [4, 9]. The acute onset of hypotension, tachycardia, hypoxia, and erythema observed in our patient immediately following rocuronium administration is characteristic of an IgE-mediated anaphylactic response.

While epinephrine remains the cornerstone of anaphylaxis management, its efficacy can be limited if the inciting antigen remains in circulation, perpetuating mast cell and basophil degranulation [10]. This likely explains the refractory hypotension in our patient despite multiple epinephrine boluses.

Sugammadex, a modified gamma-cyclodextrin, is designed to encapsulate steroidal NMBAs like rocuronium in a tight 1:1 complex, rapidly reducing the free plasma concentrations of the drug [6, 11]. Beyond its primary role in reversing neuromuscular blockade, sugammadex's mechanism of action offers a rational basis for its use in treating rocuronium-induced anaphylaxis by eliminating the triggering antigen [12]. The immediate and dramatic improvement in hemodynamics and respiratory function following sugammadex administration in this case supports its potential as a life-saving therapy. Emerging evidence suggests that sugammadex may also stabilize mast cells by preventing further cross-linking of IgE antibodies, thereby attenuating ongoing degranulation [13, 14].

However, sugammadex is highly specific. It is effective only in cases of anaphylaxis induced by steroidal NMBAs such as rocuronium and vecuronium, and is not useful for treating reactions caused by non-steroidal NMBAs (e.g., succinylcholine), antibiotics, latex, or other agents. Additionally, clinicians must be aware of potential drug interactions, such as its ability to bind to steroidal contraceptives and other medications like torcemide, necessitating alternative non-hormonal contraceptive methods for a period following sugammadex administration. While sugammadex's effect on coagulation is minimal, a transient increase in activated partial thromboplastin time and prothrombin time has been observed, although there is no clinical evidence of an increased bleeding risk [11].

The potential for adverse drug reactions to sugammadex itself must also be considered. Although rare, hypersensitivity reactions, including anaphylaxis, have been reported [15]. Common adverse effects include altered taste sensation, nausea, vomiting, headache, bradycardia and QTc prolongation. These are generally managed with symptomatic treatment, such as antiemetics for nausea and vomiting, analgesics for headache, atropine or glycopyrollate for bradycardia, and vigilalant hemodynamic monitoring for arrhythmias and QTc prolongation [11]. Thus, its use should be reserved for clearly refractory cases in which rocuronium-induced anaphylaxis is strongly suspected. Sugammadex is not a substitute for first-line anaphylaxis management with epinephrine, fluids, corticosteroids, and antihistamines, but rather serves as an adjunctive therapy when these measures fail.

This case is particularly relevant in resource-limited settings where advanced diagnostics like serum tryptase assays may not be available. In such situations, clinical acumen and a high index of suspicion are crucial. The empirical use of sugammadex in a refractory anaphylactic scenario temporally linked to rocuronium administration can be life-saving.

Author contributions: Conceptualization: Rahul Kumar Chaudhary, Prajwal Bista, Lalit Kumar Rajbanshi, Kanak Khanal. Writing–original draft: Rahul Kumar Chaudhary. Writing–review & editing: Rahul Kumar Chaudhary.

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