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ROCURONIUM-INDUCED INTRAOPERATIVE ANAPHYLAXIS PRESENTING AS ACUTE PULMONARY OEDEMA: A CASE REPORT

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ABSTRACT **Introduction:** Perioperative anaphylaxis is a rare but life-threatening emergency, and neuromuscular blocking agents (NMBAs), especially rocuronium, are among its leading triggers. Its presentation may be atypical and overlapping with other anaesthetic complications, making prompt diagnosis challenging. We report an unusual case of rocuronium-induced anaphylaxis that initially presented as acute intraoperative pulmonary oedema, was successfully managed with adrenaline and sugammadex, and highlights the critical importance of recognising delayed or atypical anaphylaxis in the perioperative period. **Case Presentation:** A 35-year-old female patient, weighing 50 kg, underwent right ear tympanoplasty with mastoid exploration under general anaesthesia. After induction with propofol and rocuronium bromide (40 mg), she developed acute tachycardia, hypertension, desaturation (SpO2 <86%), and bilateral crepitations with frothy endotracheal secretions consistent with acute pulmonary oedema. Despite management with furosemide, hydrocortisone, and vasodilators, haemodynamic instability persisted. A second dose of rocuronium was administered for elective ventilation, after which the patient developed further haemodynamic deterioration. Rocuronium-induced anaphylaxis was diagnosed. She was administered adrenaline 0.5 mg intramuscularly (1:1000) into the anterolateral thigh, repeated after 5 minutes, along with intravenous sugammadex 200 mg. Within 5–6 minutes, haemodynamics and oxygen saturation normalised. The patient was successfully extubated, shifted to the ICU, managed with BiPAP oxygen therapy for 8 hours, and discharged uneventfully after 3 days. **Conclusion:** This case underscores the atypical presentation of rocuronium anaphylaxis masquerading as acute pulmonary oedema. Sugammadex, by encapsulating free rocuronium molecules and rapidly reversing neuromuscular blockade, served as a valuable adjunct in management alongside adrenaline. Anaesthetists should maintain a high index of suspicion for NMBA-related anaphylaxis even when symptoms deviate from classical patterns.

KEYWORDS : Rocuronium, Anaphylaxis, Perioperative, Acute Pulmonary Oedema, Sugammadex, Neuromuscular Blocking Agents, Intraoperative Emergency

INTRODUCTION

Perioperative anaphylaxis is a severe, immediate hypersensitivity reaction that can rapidly progress to cardiovascular collapse and death if not promptly identified and treated. The overall incidence of perioperative anaphylaxis has been estimated to range from 1 in 3,500 to 1 in 20,000 anaesthetic procedures, with a reported mortality of up to 9%.[1,2] Neuromuscular blocking agents (NMBAs) are the single most common trigger for perioperative anaphylaxis, accounting for 50–70% of all reported cases.[3] Among NMBAs, rocuronium bromide, a non-depolarising amino steroidal agent widely used for rapid sequence intubation and routine intubation, is one of the most frequently implicated agents, responsible for up to 56% of NMBA-related anaphylactic events.[4,5]

Allergic reactions to NMBAs are predominantly IgE-mediated; however, MRGPRX2 (Mas-related G-protein-coupled receptor X2)-mediated non-IgE mast cell activation has also been described as an alternative pathway, particularly relevant because up to 75% of reactions occur on the first known exposure to the agent.[6,7] The clinical presentation of perioperative anaphylaxis is variable and may include cutaneous features (urticaria, angioedema), cardiovascular collapse (hypotension, tachycardia), bronchospasm, and desaturation. However, in the absence of cutaneous manifestations—which may be obscured by surgical draping—the diagnosis can be delayed or missed entirely.[1]

Sugammadex, a modified gamma-cyclodextrin, was developed as a selective reversal agent for rocuronium and vecuronium. Its mechanism involves encapsulation of free rocuronium molecules in a 1:1 ratio, thereby rapidly terminating neuromuscular blockade. Several reports have documented the role of sugammadex as an adjunct in the management of rocuronium-induced anaphylaxis by reducing the circulating antigen load, particularly in cases refractory to standard epinephrine therapy.[8,9,10]

Here, we report an unusual case of rocuronium-induced perioperative anaphylaxis with an atypical initial presentation as acute pulmonary oedema, successfully managed with adrenaline and sugammadex, with complete recovery.

Case Report

A 35-year-old female patient, weighing 50 kg, presented to the pre-anaesthetic clinic with a chief complaint of right ear discharge for 2 years. She was a diagnosed case of right ear Chronic Suppurative Otitis Media (CSOM), mucosal type, with atelectatic otitis media of the left ear and bilateral moderate conductive hearing impairment. She was scheduled for right ear tympanoplasty with mastoid exploration.

Pre-operative assessment revealed no history of recent upper respiratory tract infection, asthma, atopy, or allergic reactions to foods or drugs. Physical examination was unremarkable. Routine blood investigations, electrocardiogram (ECG), and chest X-ray were within normal limits. The patient was classified as ASA Physical Status Grade I.

On the day of surgery, the patient was transferred to the Ear, Nose, and Throat (ENT) operating room. Standard ASA monitoring was established, including non-invasive blood pressure (NIBP), pulse oximetry (SpO2), ECG, and end-tidal CO2 (EtCO2) monitoring. Written informed consent was obtained from the patient prior to anaesthesia.

Premedication was administered with injection ondansetron 4 mg IV, injection omeprazole 40 mg IV, injection nalbuphine 10 mg IV, injection midazolam 2 mg IV, and injection glycopyrrolate 0.2 mg IV. Preoxygenation was performed with 100% oxygen via face mask for 3 minutes. General anaesthesia was induced with injection propofol 100 mg IV administered slowly. Satisfactory bag-mask ventilation was confirmed before administering injection rocuronium bromide 40 mg IV (0.8 mg/kg). The patient was then intubated with a cuffed 7.0 mm internal diameter endotracheal tube (ETT). Correct ETT placement was confirmed by bilateral chest auscultation and end-tidal CO2

waveform capnography. Anaesthesia was maintained with nitrous oxide (N2O), oxygen (O2), and sevoflurane.

Approximately 10–15 minutes following intubation, the patient abruptly developed tachycardia with a pulse rate (PR) exceeding 150 beats per minute and hypertension with blood pressure (BP) of 162/94 mmHg. Treatment was commenced with infusions of injection nitroglycerin and injection esmolol. Despite observation for 10 minutes, the haemodynamic instability continued to escalate. Subsequently, the patient developed progressive oxygen desaturation with SpO2 falling below 86% on 100% oxygen. Bilateral crepitations were heard on chest auscultation, and endotracheal tube suctioning yielded whitish frothy secretions. The clinical picture was consistent with acute pulmonary oedema.[11]

The patient was managed accordingly with head-end elevation, 100% oxygen supplementation, injection hydrocortisone 200 mg IV, and injection furosemide 40 mg IV. Haemodynamic parameters (BP and PR) showed partial stabilisation; however, chest crepitations did not improve significantly. A decision was made to shift the patient to the postoperative ICU for elective mechanical ventilation.

For facilitation of intubation and transfer, a second dose of injection rocuronium bromide was administered intravenously. Following this second administration, the patient again developed acute hypertension and tachycardia. The recurrence of haemodynamic instability specifically after re-administration of rocuronium strongly suggested the diagnosis of rocuronium-induced anaphylaxis.[8,9] This diagnosis was supported by the clinical triad of cardiovascular compromise, respiratory compromise (pulmonary oedema), and the temporal relationship with rocuronium administration.

The patient was promptly managed with adrenaline (epinephrine) 0.5 mg (1:1000 solution, 0.5 mL) administered intramuscularly into the anterolateral thigh, the preferred site as per current WHO and EAACI anaphylaxis guidelines, repeated after 5 minutes, along with injection sugammadex 200 mg IV administered as a single bolus.[1,12] The intramuscular route via the anterolateral thigh ensures faster and more reliable absorption compared to subcutaneous or deltoid routes, owing to the rich vascularity of the vastus lateralis muscle.[12] Within 5–6 minutes of sugammadex administration, haemodynamic parameters began to stabilise. SpO2 improved progressively, and on repeat chest auscultation, bilateral crepitations showed marked improvement.[8,9,10] The patient was assessed for adequacy of spontaneous neuromuscular recovery and was then extubated safely. She was shifted to the postoperative ICU for further monitoring.

Arterial blood gas (ABG) analysis performed on admission to the ICU revealed: pH = 7.36, PaCO2 = 24.7 mmHg, PaO2 = 78 mmHg, and Base Excess (BE) = −11 mmol/L, indicative of a compensated metabolic acidosis with respiratory compensation. A bedside chest X-ray performed at this time (Figure 1) showed features consistent with resolving pulmonary oedema, with bilateral perihilar haziness and vascular engorgement, confirming the intraoperative diagnosis. The patient was administered oxygen supplementation via BiPAP for 8 hours.

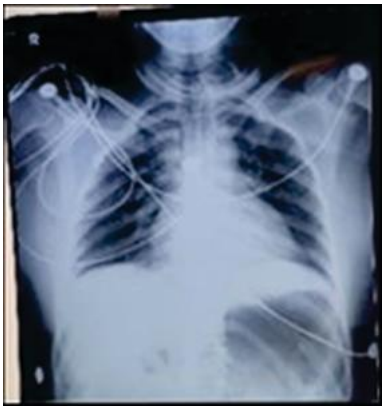

Figure 1: Bedside Chest X-ray Obtained in the Postoperative ICU Showing Bilateral Perihilar Haziness, Vascular Engorgement, and Features Consistent with Resolving Pulmonary Oedema Following Rocuronium-induced Anaphylaxis. Endotracheal Tube and Monitoring Leads are in Situ.

A repeat ABG after 8 hours of BiPAP therapy showed significant improvement: pH = 7.49, PaCO2 = 26.6 mmHg, PaO2 = 150 mmHg, and BE = −3 mmol/L. Following 3 days of uneventful ICU monitoring, the patient was successfully discharged. She was advised to follow up in both the ENT and Anaesthesia outpatient departments. Allergy evaluation, including skin prick testing and serum IgE measurement for rocuronium, was planned for follow-up visits. The patient was counselled regarding her sensitisation and advised to avoid rocuronium for all future anaesthetic procedures.

DISCUSSION

This case illustrates an unusual and clinically challenging presentation of rocuronium-induced perioperative anaphylaxis, where the predominant initial manifestation was acute pulmonary oedema rather than the classical hypotension and urticaria. The absence of cutaneous signs—commonly masked by surgical draping—and the prominence of respiratory and haemodynamic features led to an initial diagnosis of acute pulmonary oedema, delaying the recognition of the underlying anaphylactic aetiology.

Rocuronium, a non-depolarising aminosteroidal NMBA, is a well-established trigger for perioperative anaphylaxis. Epidemiological data from France and Australia have consistently identified rocuronium and suxamethonium as the NMBAs with the highest anaphylaxis incidence rates, with rocuronium accounting for up to 56% of NMBA-related cases.[4,5] The incidence of rocuronium-induced anaphylaxis has been variably reported from 1:3,500 to 1:445,000, reflecting differences in reporting practices, diagnostic criteria, and regional drug usage patterns.[1,2]

The pathophysiological mechanism of NMBA-induced anaphylaxis is predominantly IgE-mediated, involving the quaternary ammonium ion structure shared by all NMBAs. However, an IgE-independent mechanism via MRGPRX2 on mast cells has increasingly been recognised, which may partly explain why reactions can occur even on first exposure.[6,7] In our patient, who had no prior known NMBA exposure or documented allergic history, this first-exposure sensitisation mechanism is plausible.

The clinical presentation in our case was dominated by acute pulmonary oedema, as evidenced clinically and radiologically (Figure 1). This can be explained by anaphylaxis-induced capillary leak and massive histamine release causing increased pulmonary vascular permeability, as well as possible anaphylaxis-triggered negative pressure pulmonary oedema due to laryngospasm or bronchospasm following mediator release.[11] The key diagnostic clue was the temporal relationship of haemodynamic instability reoccurring immediately following the second dose of rocuronium, prompting reconsideration of the diagnosis.

Adrenaline (epinephrine) remains the cornerstone of anaphylaxis management, acting on α1-adrenergic receptors to cause vaso-constriction and on β2-adrenergic receptors to cause bronchodilation and inhibit further mediator release.[1] Current WHO and EAACI guidelines unequivocally recommend adrenaline 0.5 mg (1:1000) administered intramuscularly into the anterolateral thigh as the first-line treatment for anaphylaxis in adults, with the dose repeatable every 5 minutes if required.[1,12] The anterolateral thigh is preferred over the deltoid or subcutaneous routes due to its higher peak plasma concentration and faster time to peak effect, attributable to the rich vascularity of the vastus lateralis muscle.[12] In perioperative anaphylaxis, IV access is readily available, and if IM adrenaline (1:1000) does not rapidly achieve haemodynamic stability, IV adrenaline titrated as a dilute infusion (1:100,000 or 10 mcg/mL) should be initiated without delay.[1,7]

The role of sugammadex in the management of rocuronium-induced anaphylaxis has been extensively described in the literature. By encapsulating free plasma rocuronium molecules in a 1:1 complex, sugammadex effectively reduces the antigenic trigger load, thereby attenuating the ongoing immune response.[8,9,10] Several case reports and a growing body of evidence support sugammadex as a valuable adjunct when adrenaline alone is insufficient or when anaphylaxis is refractory.[8,9,10] In our patient, the dramatic clinical improvement within 5–6 minutes of sugammadex administration corroborates this therapeutic benefit. The dose used (200 mg, equivalent to 4 mg/kg for a 50 kg patient) provides standard reversal coverage. However, some authors advocate higher doses of 16 mg/kg (800 mg) in the acute anaphylactic setting to maximally scavenge circulating rocuronium

molecules, and this merits consideration in future similar cases.[8,10]

Postoperative BiPAP therapy for 8 hours was instrumental in supporting the patient's respiratory recovery, with repeat ABG demonstrating significant improvement in oxygenation and acid-base status. All patients recovering from perioperative anaphylaxis should receive careful post-event monitoring, allergy workup including serum tryptase (ideally within 1–2 hours of the reaction), and skin prick/intradermal testing after 4–6 weeks to confirm the causative agent and assess cross-reactivity.[1,2] Future anaesthesia in this patient should avoid rocuronium and succinylcholine, with cisatracurium identified as the NMBA with the lowest cross-reactivity profile.[4]

CONCLUSION

This case highlights the importance of maintaining a high index of suspicion for rocuronium-induced anaphylaxis even when clinical presentation is atypical, such as predominantly pulmonary oedema without classical urticaria or hypotension. The temporal correlation with rocuronium re-administration was the key diagnostic clue. Adrenaline administered intramuscularly into the anterolateral thigh (0.5 mg, 1:1000) remains the definitive first-line treatment, and combined use with sugammadex led to rapid and complete haemodynamic recovery in our patient. Anaesthetists should be familiar with this potentially life-saving role of sugammadex in rocuronium anaphylaxis and must ensure post-event allergy evaluation to guide safe future anaesthetic management.

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Conflict of Interest

None.

Source of Funding

None.

Patient Consent

Written informed consent was obtained from the patient for publication of this case report, including the clinical X-ray image.

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