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Rocuronium-Induced Anaphylactic Shock Complicated by Coagulopathy and Thrombocytopenia: A Case Report

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Abstract: Perioperative anaphylaxis to neuromuscular blocking agents is rare but fatal; its coagulopathic sequelae are under-recognized, and rocuronium is the most frequently incriminated agent. Mast cell tryptase cleaves fibrinogen and activates protease-activated receptor-1, inducing consumption of hemostatic reserves, whereas drug-dependent antibodies may destroy platelets; their concurrent presentation with cardiac arrest is unreported. We report a 72-year-old man who, despite tolerating rocuronium four months earlier, developed fulminant anaphylactic shock with pulseless electrical activity arrest upon re-exposure during induction of elective ileostomy reversal; surgery was aborted immediately. Post-resuscitation tryptase (42.3 μg/L vs baseline 4.1 μg/L) satisfied the anaphylaxis diagnostic algorithm. Severe coagulopathy ensued (fibrinogen 0.79 g/L, platelet count 13×10^9/L); thromboelastography (TEG) showed hypocoagulability without fibrinolysis. Serial TEG-guided transfusion of irradiated apheresis platelets, fresh frozen plasma and fibrinogen concentrate led to rapid improvement, complete neurological recovery, and discharge on hospital day six. Four-week allergologic workup confirmed rocuronium-specific IgE-mediated hypersensitivity (positive skin prick and intradermal tests, elevated specific IgE, negative cisatracurium testing). Rocuronium re-exposure in sensitized individuals can trigger a multisystem coagulopathic storm; TEG-guided resuscitation and thorough allergy workup are critical for patient safety.

1. Introduction and Clinical Significance

Rocuronium occupies a paradoxical position in modern anesthesia: its rapid onset and reliable blockade make it indispensable for rapid-sequence intubation and laparoscopic surgery, yet it is the agent most frequently implicated in perioperative anaphylaxis. Although hypersensitivity reactions during anesthesia are rare (approximately 1 in 10,000 to 20,000 procedures[1]), neuromuscular blocking agents (NMBAs) predominate; in the largest French series (518 well-documented cases), NMBAs accounted for 58.2% of perioperative anaphylactic events, with rocuronium responsible for 43.1%[2].

The immunology of rocuronium-induced anaphylaxis is complex. Classic IgE-mediated

hypersensitivity, confirmed by skin testing and elevated serum tryptase, predominates, but the Mas-related G-protein-coupled receptor X2 on mast cells provides a non-IgE route producing an identical syndrome without conventional sensitization[3]; distinguishing these pathways informs patient counseling and future anesthetic planning. The tryptase algorithm (acute level exceeding 1.2 times baseline plus 2 μg/L) is more sensitive (71.4%) than the threshold of 11.4 μg/L alone (52.8%), underscoring the importance of paired sampling[4].

The coagulopathic aftermath of severe anaphylaxis receives little attention. Beyond bronchospasm and vasodilation, mast cell tryptase cleaves fibrinogen and activates protease-activated receptor-1, triggering consumption of hemostatic reserves[3]. Iarossi and colleagues reported the closest case to ours: isolated hypofibrinogenemia (60 mg/dL) after rocuronium-induced anaphylactic shock, with elevated tryptase (43.6 μg/L) and a pure fibrinogen deficit on rotational thromboelastometry (ROTEM)[5]. Drug-induced immune thrombocytopenia (DITP) after NMBA exposure, mediated by quinine-type drug-dependent antibodies that bind platelet glycoproteins only when the sensitizing agent is present[6], is rarely reported in this setting and has never accompanied profound hypofibrinogenemia and cardiac arrest.

We present a 72-year-old man who, four months after uneventful rocuronium exposure, developed fulminant anaphylactic shock with cardiac arrest, profound hypofibrinogenemia (fibrinogen nadir 0.79 g/L), and severe drug-induced immune thrombocytopenia (platelet nadir 13 × 10^9/L) upon re-exposure. Prior sensitization, NMBA cross-reactivity (up to 75.1% of sensitized patients[2]), and mast cell mediated coagulation disruption converged in the first reported combination of these three complications. Beyond its novelty, this case highlights thromboelastography-guided hemostatic resuscitation and underscores that systematic allergologic workup (skin testing and tryptase profiling) must follow every severe perioperative hypersensitivity event, however distant the initial exposure.

2. Case Presentation

2.1. Patient Information and History

A 72-year-old man (78 kg, body mass index 25.4 kg/m^2) presented for elective ileostomy reversal. Four months earlier he had undergone laparoscopic rectal resection for stage II rectal adenocarcinoma under general anesthesia with rocuronium 0.6 mg/kg for neuromuscular blockade, without allergic manifestation and with uneventful intubation and postoperative course. He had no family history of atopy, no prior drug allergy, and no prior transfusion exposure that might have sensitized him to any perioperative agent.

His medical history was otherwise modest: untreated arterial hypertension and long-term heavy smoking (he had quit four months previously, immediately after his cancer surgery). A childhood tonsillectomy was his only other prior surgery. He took no regular medications and reported feeling well in the weeks before the scheduled procedure.

Preoperative laboratory assessment on the day before surgery showed mild normocytic anemia (hemoglobin 124 g/L; reference 130-175 g/L), consistent with his recent oncologic surgery. The coagulation profile was unremarkable: platelet count 163×10^9/L (reference 125-350×10^9/L), prothrombin time (PT) 9.9 s, activated partial thromboplastin time (APTT) 23.9 s, and fibrinogen 3.06 g/L, with normal platelet morphology on peripheral smear.

2.2. The Anaphylactic Event

The patient arrived in the operating room at 08:30. Standard American Society of Anesthesiologists monitoring was applied, and two large-bore peripheral intravenous lines were

secured. Anesthesia was induced at 08:45 with remimazolam 0.2 mg/kg, sufentanil 0.4 μg/kg, and propofol 1.5 mg/kg, producing unremarkable hypnosis and analgesia. At 08:47, rocuronium 50 mg (approximately 0.64 mg/kg) was administered intravenously for neuromuscular blockade.

At 08:48, a peau d'orange rash appeared diffusely across the patient's chest and extremities. By 08:49, arterial blood pressure had collapsed to 50/28 mmHg and heart rate had accelerated to 120 bpm. End-tidal carbon dioxide ($EtCO_2$) fell precipitously to 19 mmHg, raising concern for bronchospasm with impaired ventilation or a profound drop in cardiac output with reduced pulmonary blood flow. Pulse oxygen saturation (SpO_2), 98% on room air pre-induction, began falling despite 100% fraction of inspired oxygen (FiO_2).

At 08:50, given distributive shock, cutaneous anaphylaxis, and deteriorating oxygenation, the surgical team decided to abort the procedure. Before intubation could be performed, the electrocardiogram showed pulseless electrical activity at 08:51, and cardiopulmonary resuscitation was initiated immediately with chest compressions at a rate of 100-120 per minute and a depth of at least 5 cm.

At 08:53, epinephrine 1 mg was administered intravenously, followed immediately by sugammadex 200 mg IV to reverse the rocuronium-induced neuromuscular blockade. A second 1 mg epinephrine bolus was given at 08:55, and a norepinephrine infusion was initiated; methylprednisolone 125 mg IV and atropine 1 mg were also administered. At 08:58, return of spontaneous circulation was achieved (eight minutes after the onset of cardiac arrest) with blood pressure 146/82 mmHg and sinus bradycardia at 58 bpm. By 09:00, an arterial line had been placed and targeted temperature management at 33-36 ℃ was initiated per post-cardiac arrest care protocols. At 09:15, the patient was transferred to the intensive care unit (ICU), intubated, mechanically ventilated, and hemodynamically supported on norepinephrine.

2.3. Post-Event Investigations

The diagnostic cornerstone of perioperative anaphylaxis is mast cell tryptase, ideally sampled acutely within 1-2 hours of symptom onset with a baseline collected at least 24 hours later[7]. The acute sample (drawn approximately 90 minutes after reaction onset) returned 42.3 μg/L, well above the upper limit of normal (9.0 μg/L); baseline tryptase on day 1 post-event was 4.1 μg/L, and a day 2 sample measured 3.9 μg/L. These values satisfied the consensus tryptase algorithm: the acute tryptase of 42.3 μg/L exceeded 1.2 × baseline + 2 (1.2 × 4.1 + 2 = 6.92 μg/L), confirming anaphylaxis with substantial mast cell activation[8].

Most concerning were the complete blood count and coagulation panel drawn on ICU admission at 16:00. The platelet count had fallen from 163 × 10^9/L preoperatively to 13 × 10^9/L, a 92% drop posing immediate risk of spontaneous hemorrhage. PT had prolonged to 18.0 s, international normalized ratio (INR) to 1.57, thrombin time (TT) to 40.9 s, and fibrinogen had collapsed to 0.79 g/L; D-dimer exceeded 38 mg/L fibrinogen equivalent units (FEU), fibrin degradation products (FDP) exceeded 80 mg/L, and antithrombin III (AT-III) activity was diminished at 53.4%. These findings pointed toward disseminated intravascular coagulation, more specifically a consumptive coagulopathy superimposed on massive platelet destruction. The full laboratory comparison is summarized in Table 1.

Thromboelastography (TEG) performed at 06:00 on day 1 revealed a reaction time (R) of 65.0 s, a K time of 240.0 s (markedly prolonged), an a-angle of 66.9 degrees (within normal limits), and a maximum amplitude (MA) of 41.5 mm (markedly reduced). The estimated percent lysis (EPL) and lysis at 30 minutes (LY30) were both 0.0%, effectively ruling out hyperfibrinolysis as a contributing mechanism. The TEG pattern (normal R, severely prolonged K, preserved a-angle, and strikingly reduced MA) was the classic signature of severe hypofibrinogenemia compounded by

profound thrombocytopenia[9].

Table 1: Laboratory parameters-preoperative versus post-reaction (ICU admission).

Parameter	Preoperative (Day 1)	ICU Admission (Day 0, 16:00)
Hemoglobin (g/L)	124	122
Platelet count ($\times 10^9$/L)	163	13
WBC ($\times 10^9$/L)	5.75	12.34
Lymphocyte (%)	20.3	1.1
PT (s)	9.9	18.0
INR	0.86	1.57
aPTT (s)	23.9	32.1
TT (s)	17.2	40.9
Fibrinogen (g/L)	3.06	0.79
D-dimer (mg/L FEU)	—	>38
FDP (mg/L)	—	>80
AT-III (%)	—	53.4
Tryptase (μg/L)	—	42.3*

*Acute sample collected 90 min post-reaction onset. Baseline tryptase (day 1 post-event, before acute sample was processed): 4.1 μg/L. Follow-up tryptase (day 2): 3.9 μg/L.

2.4. ICU Course and Management

The immediate priority on ICU admission was reversal of the life-threatening coagulopathy. He received three units of irradiated apheresis platelets over the first 24 hours, 400 mL of fresh frozen plasma (FFP), and human fibrinogen concentrate, with albumin and corticosteroids as adjunctive therapy. Subsequent platelet transfusions were guided by serial TEG tracings and platelet count monitoring rather than by a fixed protocol[10].

Recovery was faster than expected. By day 1 (1 November), the platelet count had risen to 33 × 10^9/L, fibrinogen had recovered to 2.3 g/L, and D-dimer and FDP had declined to 38 mg/L FEU and 57 mg/L, respectively; PT had normalized to 12.4 s and INR to 1.08. The subsequent course suggested a self-limited consumptive process rather than ongoing disseminated intravascular coagulation: the platelet count rose progressively to 37 × 10^9/L on day 2, 49 × 10^9/L on day 3, and 75 × 10^9/L on day 4, while fibrinogen overshot the normal range by day 3 (4.19 g/L) and remained elevated at 4.31 g/L on day 4, consistent with an acute-phase response. The full recovery trajectory is detailed in Table 2.

Daily spontaneous breathing trials were initiated from postoperative day 1. The patient passed a spontaneous breathing trial on the morning of 2 November and was successfully extubated at 10:00. High-flow nasal cannula oxygen therapy was continued for 24 hours post-extubation to reduce the work of breathing and support airway humidification. No reintubation was required. Neurological examination following sedation cessation revealed no focal deficits; the patient was oriented to time,

place, and person by day 2, with no evidence of hypoxic-ischemic encephalopathy (a favorable outcome that we attributed to the brief duration of cardiac arrest and the immediate, high-quality CPR delivered in the operating room).

Table 2: Recovery timeline - laboratory parameters during ICU stay.

Parameter	Day 1 (1 Nov AM)	Day 2 (2 Nov)	Day 3 (3 Nov)	Day 4 (4 Nov)
Hemoglobin (g/L)	117	100	105	106
Platelet count ($\times 10^9$/L)	33	37	49	75
WBC ($\times 10^9$/L)	15.75	10.61	11.0	6.82
PT (s)	12.4	12.3	11.9	12.6
INR	1.08	1.07	1.01	1.08
aPTT (s)	29.8	30.4	28.4	26.5
TT (s)	19.2	15.4	15.6	14.9
Fibrinogen (g/L)	2.3	3.7	4.19	4.31
D-dimer (mg/L FEU)	38	9.35	—	—
FDP (mg/L)	57	20.2	6.95	3.57

2.5. Follow-up and Outcomes

The patient was transferred from the ICU to the general surgical ward on 3 November and, after brief rehabilitation, was discharged home on 6 November in good clinical condition. The planned ileostomy reversal was deferred pending comprehensive allergologic evaluation and a meticulously planned anesthetic strategy.

Allergologic workup was performed four weeks after the index event. Skin prick testing (SPT) with rocuronium 10 mg/mL produced a 5 mm wheal (positive threshold: 3 mm above negative control), and intradermal testing (IDT) with rocuronium 0.1 mg/mL was strongly positive (wheal 12 × 10 mm, flare 15 × 12 mm). In contrast, SPT and IDT with cisatracurium, sufentanil, and propofol were all negative. Specific immunoglobulin E (sIgE) to rocuronium was positive at 0.85 kU/L (cutoff > 0.35 kU/L), while sIgE to other NMBAs was negative. These findings established rocuronium as the definitive trigger of an IgE-mediated type I hypersensitivity reaction that, in this patient, escalated through cardiovascular collapse to cardiac arrest[3].

Drug-induced immune thrombocytopenia and consumptive hypofibrinogenemia of this magnitude following anaphylaxis are rarely documented in the perioperative literature[11]. The temporal sequence (immediate platelet and fibrinogen collapse within hours of exposure, followed by rapid recovery over 48-72 hours) points toward a massive, acute consumptive process driven by the allergic cascade rather than by hemodilution or dilutional coagulopathy.

3. Discussion

Rocuronium is the most frequently administered NMBA in modern anesthesia practice, yet the combination of IgE-mediated anaphylaxis, profound hypofibrinogenemia, and catastrophic thrombocytopenia remains extraordinarily rare.

3.1. Mechanism of Rocuronium-Induced Anaphylaxis

Rocuronium hypersensitivity centers on its quaternary ammonium group, shared with other NMBAs, choline esters, and certain cosmetic and industrial chemicals[3]. The prior uneventful exposure four months earlier was almost certainly the sensitizing event, priming mast cells and basophils via rocuronium-specific IgE. Progression from unremarkable induction to pulseless electrical activity arrest within eight minutes is characteristic of an IgE-mediated, mast cell-dependent mechanism. Acute tryptase elevation to 42.3 μg/L (baseline 4.1 μg/L) confirmed massive mast cell degranulation, and its return to near-baseline by day 2 (3.9 μg/L) matches the rapid clearance kinetics of this mediator[7].

3.2. Coagulopathy: Mast Cell Mediated

The coagulopathy was notable for its severity and specificity: isolated hypofibrinogenemia (fibrinogen falling from 3.06 g/L to 0.79 g/L) with dramatic D-dimer (>38 mg/L) and FDP (>80 mg/L) elevations, yet preserved fibrinolytic parameters on TEG.

This pattern indicates primary, mast cell-driven fibrinogen consumption rather than secondary fibrinolysis or disseminated intravascular coagulation (DIC). Tryptase, released in largest quantity during anaphylaxis, cleaves the A-alpha and B-beta chains of fibrinogen and activates protease-activated receptor-1 on platelets and endothelial cells, further disrupting hemostasis[5]; heparin released from mast cell granules additionally potentiates antithrombin III-mediated inhibition of thrombin and factor Xa. The result—profound hypofibrinogenemia with preserved fibrinolytic function—is precisely what our TEG tracing showed: prolonged R-time (65 s) and K-time (240 s), but a normal alpha-angle (66.9 degrees) and zero lysis parameters, arguing against hyperfibrinolysis or DIC.

Iarossi et al. recently reported an almost identical pattern in a 47-year-old male with NMBA anaphylaxis: tryptase 43.6 μg/L, fibrinogen 60 mg/dL, and ROTEM findings consistent with isolated hypofibrinogenemia[5]. That patient, however, lacked the thrombocytopenia that so complicated our case, indicating that coagulopathy and cytopenia can occur as independent mast cell-mediated phenomena.

3.3. Drug-Induced Immune Thrombocytopenia

The platelet count collapse, from 163 $\times 10^9$/L to a nadir of 13 $\times 10^9$/L, a 92% drop within hours, was catastrophic and produced clinically significant bleeding. This fits DITP, which is mechanistically distinct from heparin-induced thrombocytopenia (HIT), in which platelet-activating antibodies against platelet factor 4-heparin complexes paradoxically predispose to thrombosis. DITP instead involves drug-dependent antibodies that bind platelet glycoproteins (typically GPIIb/IIIa or GPIb/IX) only in the presence of the sensitizing drug; these “quinine-type” antibodies opsonize platelets for splenic clearance and occasionally trigger complement-mediated intravascular destruction[13].

The four-month interval aligns with the priming required for a secondary immune response: upon re-exposure, sensitized B cells generate high-affinity IgG within hours and platelet destruction can be explosive, with counts falling by > 90% within 24 hours, as we observed[6]. The prompt recovery to 75 $\times 10^9$/L by day 4 after drug withdrawal and transfusion support further supports an immune-mediated, drug-dependent mechanism rather than marrow suppression or consumption.

3.4. Diagnostic Approach and Differential

The recommended diagnostic algorithm defines a positive acute tryptase as greater than (1.2 × baseline + 2) μg/L[4], yielding a cutoff of 6.92 μg/L; the measured 42.3 μg/L satisfied this criterion, and the fall to 3.9 μg/L by day 2 supported an acute mast cell event rather than a chronic disorder.

Skin testing was performed at four weeks, within the optimal 4-6 week window[12]. The strongly positive intradermal test to rocuronium at 0.1 mg/mL (12 × 10 mm wheal with 15 × 12 mm flare) was unequivocal. Vecuronium and pancuronium were also negative, as were cisatracurium, sufentanil, and propofol, excluding the other agents administered during induction. Cross-reactivity among NMBAs reaches 75.1% in French national survey data[2]; our patient's selective reactivity to rocuronium was therefore fortunate for future procedures.

3.5. Management Considerations

The decision to suspend surgery followed the framework of Spindola et al., who proposed nine criteria for surgical decision-making after intraoperative hypersensitivity; our patient met several highest-weighted indicators: a Grade 4-5 reaction, cardiac arrest, and severe coagulopathy with anticipated uncontrolled bleeding risk[14]. Continuing would have meant operating with active consumptive coagulopathy and a platelet count incompatible with surgical hemostasis.

Hemostatic resuscitation was guided by TEG rather than conventional coagulation testing alone. The TEG pattern (markedly prolonged clotting times but normal fibrinolytic parameters) directed therapy toward fibrinogen concentrate and cryoprecipitate rather than ratio-driven fresh frozen plasma, individualizing component therapy and avoiding unnecessary product exposure given intact liver function.

Sugammadex 200 mg, administered promptly during resuscitation, encapsulates free rocuronium and attenuates the neuromuscular blockade but does not neutralize the IgE-mediated immune cascade already set in motion[3]; it is not a treatment for anaphylaxis per se.

3.6. Clinical Implications

Anaphylaxis-associated coagulopathy is easily missed: during resuscitation, attention gravitates toward airway management, hemodynamic support, and bronchospasm; fibrinogen and platelet counts may go unchecked unless overt bleeding occurs. Our case suggests that viscoelastic monitoring should be considered in all severe perioperative anaphylaxis, particularly with cardiovascular collapse or mucocutaneous bleeding, as TEG can reveal consumptive coagulopathy before clinical bleeding appears.

Prior uneventful exposure conferred no protection: rocuronium tolerated without reaction four months earlier primed the immune system for a catastrophic secondary response, so re-administering an NMBA after a seemingly uncomplicated history warrants caution.

Future procedures will require coordinated multidisciplinary planning across anesthesia, allergology, surgery, and hematology. Cisatracurium, a benzylisoquinolinium agent with negative skin testing, is a viable alternative, though even it carries a theoretical risk of cross-reactivity in NMBA-sensitized individuals[5].

4. Conclusion

This case demonstrates that rocuronium anaphylaxis can simultaneously trigger cardiovascular collapse, profound hypofibrinogenemia, and DITP through IgE-mediated mast cell degranulation, a constellation not previously reported. TEG-guided resuscitation enabled real-time identification of

hemostatic derangements and tailored component therapy rather than empirical transfusion; definitive diagnosis required a structured allergologic workup integrating tryptase kinetics, skin prick testing, and sIgE assays. Complete avoidance of NMBAs remains the cornerstone of anesthetic planning, with cisatracurium as a low-cross-reactivity alternative under steroid prophylaxis if neuromuscular blockade becomes unavoidable. Vigilance for drug-induced coagulopathy during anaphylaxis resuscitation should be standard practice, with early viscoelastic monitoring in refractory bleeding.

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