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A Case Report of Severe Anaphylactic Reaction to Sugammadex with Cardiac Arrest and Neurological Sequelae Following Robot-Assisted Partial Nephrectomy

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Case Report

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Abstract

Background: Sugammadex is widely used to reverse steroidal neuromuscular blockade, and while it is considered safe, rare cases of severe hypersensitivity reactions and fatal cardiovascular collapse have been reported.

Case Presentation: A 43-year-old man with no history of drug allergies underwent robot-assisted partial nephrectomy under general anesthesia. Shortly after intravenous administration of sugammadex to reverse neuromuscular blockade, the patient developed apnea, convulsions, and ventricular fibrillation, progressing to prolonged cardiac arrest. Despite multiple defibrillations and advanced cardiopulmonary resuscitation, the return of spontaneous circulation was achieved only after initiating extracorporeal cardiopulmonary resuscitation (ECPR) with extracorporeal membrane oxygenation (ECMO). The patient subsequently developed hypoxic-ischemic encephalopathy with persistent neurocognitive sequelae. Provocation testing performed one month later confirmed sugammadex-induced anaphylaxis.

Conclusions: In rare instances, sugammadex can trigger severe anaphylaxis and catastrophic cardiovascular collapse. Continuous monitoring during emergence and prompt aggressive resuscitation are critical to improve survival and neurological outcomes.

Background

Sugammadex, a selective relaxant-binding agent, is used to reverse neuromuscular blockade. This γ-cyclodextrin derivative provides rapid and effective reversal of rocuronium and vecuronium-induced neuromuscular blockade by forming stable 1:1 complexes with these agents [1]. Despite its widespread clinical acceptance and generally favorable safety profile, sugammadex has a reported incidence of severe anaphylaxis of approximately 0.005% in Japan, typically occurring within 5 min of administration [2, 3]. Recent studies report that sugammadex-induced anaphylaxis occurs in approximately 0.02–0.04% of cases, typically presenting within 5 minutes of administration with hypotension, erythema, and decreased oxygen saturation [4]. The mechanism of sugammadex-induced anaphylaxis remains incompletely understood, and it is unknown whether hypersensitivity reactions involve IgE-mediated or non-IgE mediated hypersensitivity mechanisms [3].

We report a severe anaphylactic reaction to sugammadex in a 43-year-old male patient following robot-assisted partial nephrectomy (RAPN), resulting in cardiac arrest, prolonged resuscitation, and neurological sequelae. This case emphasizes the importance of vigilant monitoring during sugammadex administration and the need for immediate and aggressive perioperative management of anaphylactic reactions. Even catastrophic cardiovascular collapse due to sugammadex-induced hypersensitivity can result in favorable neurological recovery with timely extracorporeal cardiopulmonary resuscitation (ECPR) and aggressive critical care.

Case Presentation

A 43-year-old male patient was admitted to the urology department for evaluation of a right-sided renal mass. His medical history was unremarkable and he had no known allergies to food or medications. Social history revealed consumption of one bottle of soju daily and smoking of 20 cigarettes per day. Physical examination and imaging studies revealed a 3.2 cm renal mass. The patient was thus scheduled for RAPN.

The patient was admitted to the operating room at 10:00 AM in a fasted state. No premedications were administered. Initial vital signs in supine position showed: heart rate 69 beats per minute (bpm), systolic blood pressure 171 mmHg, diastolic blood pressure 100 mmHg, oxygen saturation 99%, and body temperature 36°C.

Anesthesia was induced with 150 mg propofol and 50 mg rocuronium, followed by maintenance with remifentanil and desflurane. Tracheal intubation was performed using a 7.5 mm endotracheal tube with a McGrath laryngoscope. A 22-gauge radial arterial catheter was placed in the left radial artery for continuous hemodynamic monitoring and blood gas sampling, while an 18-gauge peripheral venous catheter was secured in the right forearm for large-bore venous access.

The patient was positioned in left lateral decubitus position with slight flexion to ensure optimal surgical exposure. Surgery commenced at 10:45 AM without complications. At 12:20 PM, 250 mL of 15% mannitol was administered for renal protection prior to renal artery clamping. Throughout the surgical procedure, the patient's hemodynamic parameters remained stably within 20% of baseline values. Oxygen saturation and core body temperature were maintained at baseline levels. Anesthesia depth monitoring produced entropy values consistently between 40–60, and end-tidal CO_2 was maintained at 32–40 mmHg. Estimated blood loss was minimal (100 mL), with a urine output of 250 mL.

Following completion of the surgical procedure, the patient was replaced into supine position. At 1:40 PM, antiemetic prophylaxis with 0.3 mg ramosetron was administered with a 4 mL loading dose of patient-controlled intravenous analgesia (PCIA). The PCIA solution contained 100 mg oxycodone, 100 mg nefopam hydrochloride, and 0.6 mg ramosetron in 76 mL normal saline, programmed to deliver 1 mL per activation with a 6-minute lockout interval and 40 mL per four-hour limit. Neuromuscular function assessment revealed incomplete recovery from muscle relaxation. Consequently, 200 mg sugammadex was administered intravenously to reverse residual neuromuscular blockade.

During preparation for transfer to the post-anesthesia care unit (PACU) at 2:05 PM, the patient suddenly developed opisthotonous convulsions and experienced cessation of spontaneous breathing. At this time, the ECG was fine. Emergency reintubation was performed immediately and continuous monitoring of vital signs was resumed.

At 2:11 PM, although spontaneous breathing briefly returned, the patient experienced sudden pulse loss and developed ventricular fibrillation. Cardiopulmonary resuscitation (CPR) was initiated immediately. Arterial blood gas analysis (ABGA) revealed severe acidosis and electrolyte disturbances: pH 7.222, PCO_2 41.4 mmHg, PO_2 306.3 mmHg, hemoglobin 16.2 g/dL, hematocrit 48%, HCO_3^- 17 mM, base excess

– 10.3, sodium 129 mmol/L, chloride 101 mmol/L, potassium 6.4 mmol/L, calcium 1.78 mmol/L, lactate 6.39 mmol/L, and glucose 172 mg/dL.

Treatment for hyperkalemia was initiated with 20 mL calcium gluconate and dextrose. Continuous infusion of dopamine and dobutamine was initiated to provide hemodynamic support. Throughout resuscitation, a total of 23 mg epinephrine was administered intravenously in 12 divided doses at 3–5 minute intervals. Chest compressions were maintained throughout nine 200-joule defibrillation attempts.

Owing to prolonged cardiac arrest and failure to achieve sustained return of spontaneous circulation (ROSC), extracorporeal membrane oxygenation (ECMO) was initiated at 2:48 PM. ROSC was eventually achieved at 3:08 PM and the patient was transferred to the intensive care unit for continued management.

At 3:20 PM, the patient experienced recurrent cardiac arrest requiring CPR resumption. Despite administration of 3 mL of lidocaine, 100 mL of 8.4% sodium bicarbonate, and 6 mg of epinephrine, along with eight 200-joule defibrillation attempts, restoration of circulation was unsuccessful. Two additional 250-joule defibrillations were applied, finally achieving ROSC at 3:40 PM.

Continuous infusion from 200 mL of 5% dextrose, sodium bicarbonate, and epinephrine was maintained, and 125 mg of methylprednisolone was administered for potential cerebral edema. Cardiology consultation and transesophageal echocardiography (TEE) revealed no significant abnormalities. By 8:00 PM, the patient was semicomatose with prompt pupillary light reflexes, and demonstrating ventilator dyssynchrony.

Laboratory investigations the following morning revealed significant abnormalities: white blood cell count 29.99 × 10^3/μL, aspartate aminotransferase (AST) 1016 U/L, alanine aminotransferase (ALT) 608 U/L, creatinine 2.01 mg/dL, calcium 7.16 mg/dL, prothrombin time INR 1.33, creatine kinase (CK) 8636 U/L, creatine kinase MB fraction (CK-MB) 483 ng/mL, high-sensitivity C-reactive protein 6.19 mg/dL, and high-sensitivity troponin I 125,000 pg/mL.

Neurological consultation and electroencephalography (EEG) suggested hypoxic brain injury. Computed tomography (CT) of the brain revealed no acute abnormalities, and magnetic resonance imaging (MRI) performed the following day revealed subtle diffusion-weighted imaging (DWI) hyperintensity in bilateral frontoparietal regions, consistent with cytotoxic cerebral edema.

ECMO was discontinued, and targeted temperature management (TTM) was initiated with a target temperature of 33°C for 24 hours, followed by controlled rewarming. The patient developed pleural effusion and pneumothorax, requiring chest tube placement. Treatment for suspected cytotoxic cerebral edema included methylprednisolone 1.5 mg/kg every 6 hours and 15–20% mannitol intravenously for 5 days. Follow-up MRI three days later revealed diffuse bilateral hyperintensity in the cerebral cortex on diffusion-weighted imaging (Fig. 1). Cardiac biomarkers gradually improved, with highly sensitive troponin I decreasing to 23,203 pg/mL, CK-MB to 219 ng/mL, and CK to 8652 U/L by the third day.

On sixth days post-incident, the patient began responding minimally to verbal commands. His mental status had improved to a stuporous state, with Richmond Agitation-Sedation Scale (RASS) scores ranging from + 1 to + 2, indicating restlessness to mild agitation. After two weeks, the patient recognized family members but remained aggressive, provided incoherent responses to questions, and demonstrated poor orientation. One month after the incident, provocation testing was performed for suspected allergens, including remifentanil, rocuronium, and sugammadex. Sugammadex caused a positive reaction, confirming a diagnosis of sugammadex-induced anaphylaxis (Fig. 2). Four months after the incident, another MRI was taken, and the hypointensity on the DWI normalized, and the cerebral edema subsided significantly, showing normal findings (Fig. 1). At 7 months after the event, the patient demonstrated substantial neurological recovery, with marked improvements in conversational ability, comprehension, repetition, and naming. Motor function had also recovered to near-baseline levels.

Discussion

Sugammadex is widely used as a selective reversal agent for steroidal neuromuscular blockers including rocuronium and vecuronium during emergence from general anesthesia. In our case, sugammadex was administered after surgery, shortly before transfer to the PACU. Within minutes, the patient experienced a fulminant allergic reaction manifesting as severe cardiac dysfunction and culminating in ventricular fibrillation. This case underscores the potential for life-threatening adverse cardiovascular events following sugammadex administration, even in patients with no history of allergies, highlighting a need for preparedness during emergence.

Sugammadex-related adverse events have been increasingly reported in recent years. Meta-analyses and large retrospective reviews have reported an approximately 0.018–0.04% incidence of perioperative hypersensitivity reactions, with hypotension and flushing as predominant manifestations. Life-threatening events comprise a significant proportion of these [4, 5]. Several case reports have documented severe bradycardia, asystole, coronary vasospasm, and ventricular fibrillation occurring within minutes of drug administration [6, 7]. Rare cases of Kounis syndrome, a coronary vasospasm associated with allergic reactions, have also been linked to sugammadex, even in the absence of cutaneous signs [8]. A recent pharmacovigilance study using the FDA Adverse Event Reporting System (FAERS) identified significant safety signals linking sugammadex to serious arrhythmias, including ventricular fibrillation, ventricular tachycardia, bradycardia, and pulseless electrical activity, with elevated reporting odds ratios for each event [9]. Liu et al. [9] suggested that these events may be mediated by hypersensitivity reactions or direct vagal stimulation, highlighting a potential proarrhythmic risk associated with sugammadex administration. The current case involving ventricular fibrillation shortly after sugammadex injection is consistent with these prior reports and adds further support to the growing body of literature indicating that sugammadex can rarely provoke critical cardiovascular complications.

The mechanisms underlying severe cardiovascular reactions to sugammadex are likely multifactorial and remain incompletely understood. One possible explanation is a type I hypersensitivity reaction in

which sugammadex acts as an allergen to trigger IgE-mediated mast cell degranulation. This leads to release of histamine, tryptase, and other inflammatory mediators, resulting in vasodilation, myocardial depression, hypotension, and, in some cases, coronary artery spasm and arrhythmias, such as ventricular fibrillation [8]. Another proposed mechanism involves direct pharmacological effects of sugammadex on the coronary vasculature independent of allergy. Angiographic studies have confirmed coronary vasospasms in the absence of classic anaphylactic features, raising the possibility of non-IgE-mediated pathways or direct smooth muscle activation [6]. Given the complexity of these mechanisms, prevention strategies must include thorough preoperative allergy screening, cautious dosing, and ensuring that full resuscitation equipment and trained personnel are immediately available at the time of administration.

Beyond sugammadex itself, a thorough differential analysis of all pharmacological and physiological factors present in this case is warranted to assess their potential contribution to the catastrophic outcome. Several concurrent agents and circumstances merit explicit consideration. First, rocuronium — the neuromuscular blocker encapsulated by sugammadex — is itself among the most common triggers of perioperative anaphylaxis in many countries [3]. Rocuronium provocation testing returned negative, therfore excluding it as the primary trigger. Second, remifentanil was administered throughout the procedure, while opioid-induced anaphylaxis is rare, it has been documented, and remifentanil provocation testing returned negative in this case, appropriately excluding it. Third, the opioid-containing PCIA solution (oxycodone and nefopam hydrochloride) was administered as a loading dose immediately before sugammadex, and while neither agent is a recognised common allergen, their temporal proximity to the event must be acknowledged as a potential, if unlikely, contributing factor. Fourth, the patient's pre-existing hypertension and significant cardiovascular risk factors (daily alcohol consumption, heavy smoking) likely increased his underlying susceptibility to severe cardiovascular decompensation once an anaphylactic mediator release was initiated. These factors may have lowered the threshold for ventricular fibrillation even in the context of a modest hypersensitivity response. Fifth, the ABGA obtained immediately after cardiac arrest revealed severe metabolic acidosis, hyperkalemia, hyponatremia, hypocalcemia, and elevated lactate. Although these findings reflect the consequence of cardiac arrest, the intraoperative administration of 250 mL of 15% mannitol may have predisposed to electrolyte shifts. Hyperkalemia and hypocalcemia are well-established precipitants of ventricular fibrillation and likely contributed to the refractoriness of the arrest. Sixth, there is a possibility of Kunis syndrome (allergic coronary artery vasospasm). Since no structural or ischemic lesions of the myocardium were found on TEE and subsequent cardiological evaluations, and coronary angiography was not performed, this mechanism was neither confirmed nor ruled out. Collectively, while sugammadex is the most strongly associated substance based on positive provocation tests, the severe outcome observed in this case can be most plausibly explained by the combined action of sugammadex-induced anaphylaxis acting on a cardiovascularly vulnerable patient amidst simultaneous drug exposure and physiological disturbances during surgery. This multifactorial perspective is consistent with the literature on perioperative anaphylaxis, suggesting that the severity of the reaction is

rarely determined by the allergen alone but is also determined by host factors and the perioperative environment. [4, 10].

Sugammadex administration timing is critical for understanding its risk profile. Sugammadex is most commonly administered during emergence, typically immediately before extubation and patient transfer to the PACU [11]. During this transition, continuous monitoring is often reduced, because patients are considered hemodynamically stable. This case emphasizes that catastrophic events can occur within minutes of administration. Several reports have shown that hypersensitivity reactions to sugammadex often manifest within 5–10 min post-injection [2, 3], which can coincide with periods of decreased clinical vigilance. Continuous cardiovascular and respiratory monitoring should thus be maintained during and after sugammadex administration, and patients should not be considered ready for PACU transfer until stability is ensured.

When severe adverse reactions, such as cardiovascular collapse, occur after sugammadex administration, early recognition and prompt intervention are critical for improving outcomes. Clinicians should maintain a high index of suspicion when sudden hypotension, bradycardia, arrhythmia, or electrocardiographic changes occur within a few minutes of drug administration [12]. Immediate management should include activation of established protocols for anaphylaxis and cardiac arrest. First-line treatment includes rapid administration of epinephrine, followed by adjunctive therapies including antihistamines, corticosteroids, and vasopressors [13]. Nitroglycerin may also be useful in cases where coronary vasospasm is suspected, such as in Kounis syndrome [8]. If arrhythmias such as ventricular fibrillation persist despite initial resuscitative efforts, advanced cardiac life support, including defibrillation and chest compressions, must be initiated immediately. In refractory cases, as observed in our patient, early consideration of ECPR may be life-saving [14].

Post-event evaluation is essential after stabilization. Serum tryptase measurement within 1–2 hours of the event can support a diagnosis of anaphylaxis. Referral for skin testing or drug challenge under specialist supervision may help identify the causative agent [15]. Cardiological evaluation, including ECG, echocardiography, and, when indicated, coronary angiography or spasm provocation testing, should be considered to rule out ischemic contributors. These assessments aid in understanding the mechanism of injury and are crucial for planning future anesthetic management. Multidisciplinary collaboration among anesthesiologists, intensivists, cardiologists, and allergists is vital to ensure patient safety.

Notably, the initial diffusion-weighted imaging demonstrated diffuse cortical hyperintensities, a finding typically associated with poor neurological prognosis. However, in this case, these abnormalities were reversible, with substantial clinical and radiological recovery observed during follow-up. This suggests that even severe diffusion abnormalities following hypoxic brain injury may not invariably indicate irreversible damage, particularly when aggressive resuscitative measures, including ECPR, and TTM, are promptly implemented.

There are several limitations to this case. First, we initially did not anticipate drug-induced anaphylaxis, so we focused cardiologic examinations and missed the timing for the serum tryptase test. Second,

coronary angiography was not performed. Third, it is the consideration of whether anaphylaxis could have been prevented, if neostigmine had been administered after waiting until the patient's muscle relaxation had subsided from moderate to mild after the surgery. The choice of sugammadex over neostigmine was driven by several specific considerations. The depth of residual neuromuscular blockade at that moment was consistent with moderate-to-deep block, a range in which neostigmine is pharmacologically limited and may fail to produce adequate reversal, particularly with deep rocuronium blockade [16]. Neostigmine, while effective for shallow block, carries its own risks in this setting, including bradycardia, bronchospasm, and increased secretions, which can complicate emergence. We acknowledge that neostigmine, at an appropriate dose for the degree of block, might have achieved adequate reversal with a lower risk of the rare but catastrophic anaphylaxis subsequently observed. However, the clinical decision to use sugammadex was consistent with current institutional practice and international guidelines for reversal of rocuronium-induced neuromuscular blockade [16].

Conclusions

Although sugammadex is a highly effective and commonly used neuromuscular reversal agent, anesthesiologists must remain vigilant for potentially life-threatening adverse reactions. This case illustrates that rare events may result in profound and lasting consequences. Continuous monitoring, readiness for immediate intervention, and heightened awareness of the early signs of cardiovascular instability are essential components of safe clinical practice during emergence from anesthesia. This case highlights that even catastrophic cardiovascular collapse due to sugammadex-induced hypersensitivity can result in favorable neurological recovery with timely ECPR and aggressive critical care.

Abbreviations

ECPR; Extracorporeal cardiopulmonary resuscitation

ECMO; Extracorporeal membrane oxygenation

RAPN; Robot-assisted partial nephrectomy

PCIA: Patient-controlled intravenous analgesia

PACU; Post-anesthesia care unit

ROSC; Return of spontaneous circulation

TEE; Transesophageal echocardiography

AST; Aspartate aminotransferase

ALT; Alanine aminotransferase

CK; Creatine kinase

CK-MB; Creatine kinase MB

EEG; Electroencephalography

CT; Computed tomography

MRI; Magnetic resonance imaging

DWI; Diffusion-weighted imaging.

TTM; Targeted temperature management

RASS; Richmond Agitation-Sedation Scale

FAERS; FDA Adverse Event Reporting System

DWI; Diffusion-weighted imaging

SI; Signal intensity

DHE; Diffuse hypoxic encephalopathy

SPT; Skin prick test

IDT; Intradermal test.

Declarations

IRB number: not applicable

Research registration number: not applicable

Ethics approval and consent to participate

Not applicable

Consent for publication

Written informed consent was obtained from the patient for the publication of this case report.

Availability of data and materials

No datasets were generated or analysed during the current study.

Competing interests

The authors declare no competing interests.

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Authors' contributions

Wangseok Do wrote the manuscript. Boo-young Hwang revised the manuscript. Jae-young Kwon reviewed the manuscript. Dowon Lee and Siyun Moon collected the patient data. Hyae Jin Kim performed a literature review.All authors read and approved the final manuscript.

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Figures

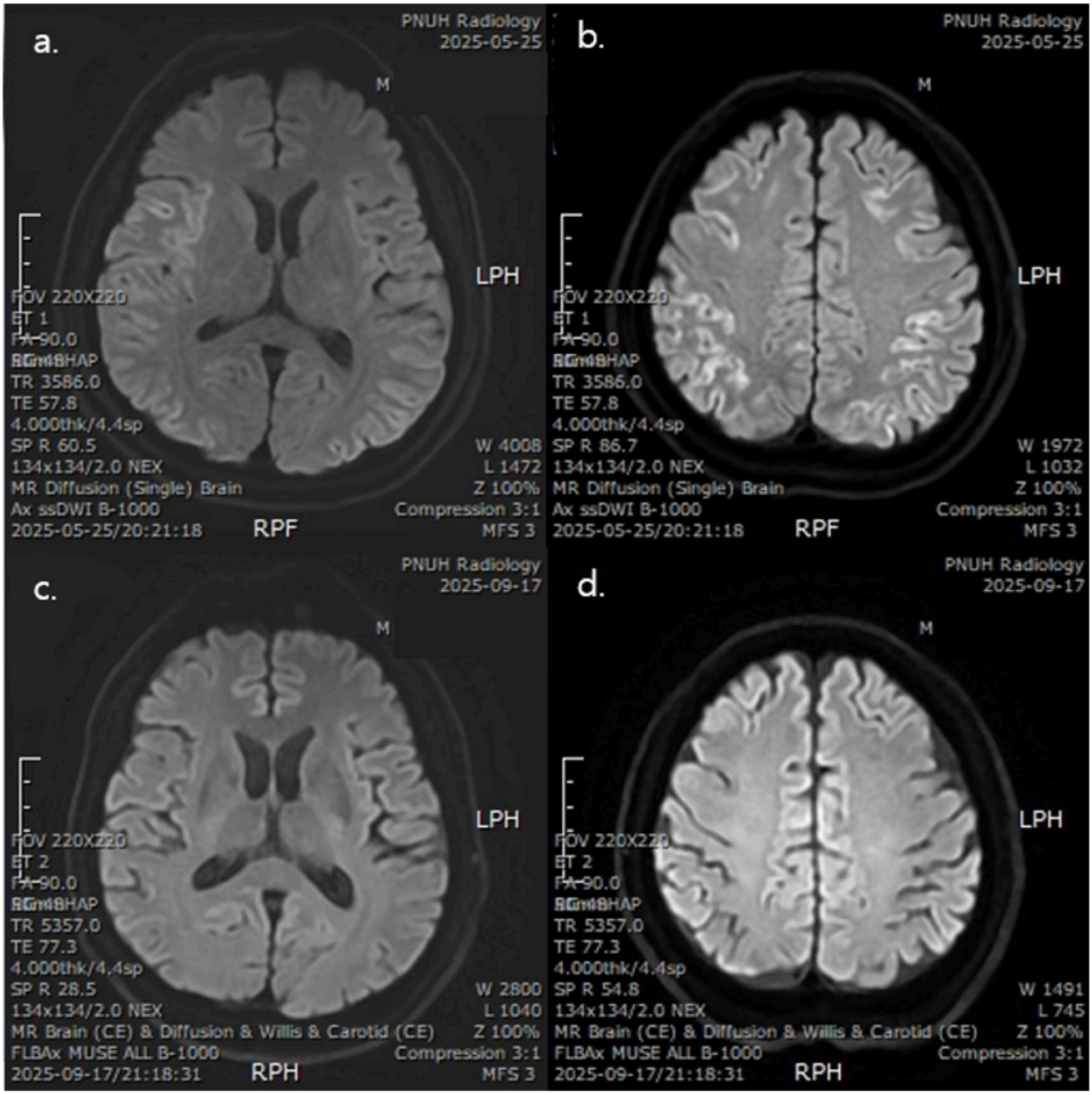


Figure 1

Diffusion-weighted magnetic resonance imaging findings following hypoxic brain injury.

Diffusion-weighted imaging (DWI) obtained 3 days after the event (a, b) demonstrates diffuse high signal intensity (SI) involving the bilateral cerebral cortex, consistent with diffuse hypoxic encephalopathy (DHE). Follow-up DWI performed 4 months later (c, d) shows complete resolution of the previously noted cortical abnormalities with no residual diffusion restriction, indicating radiologic recovery.

DWI, diffusion-weighted imaging; SI, signal intensity; DHE, diffuse hypoxic encephalopathy.

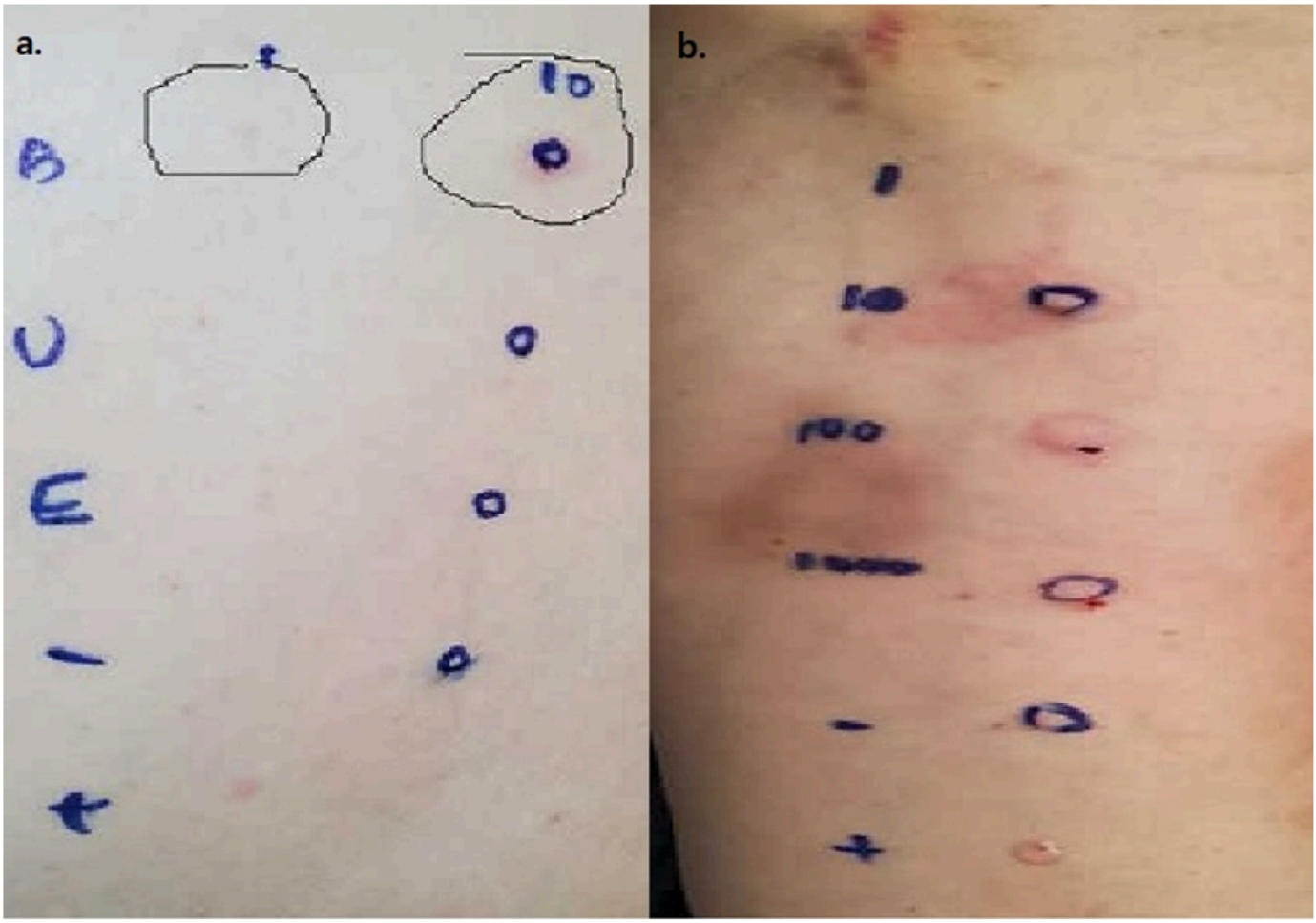


Figure 2

Skin test results

Figure 2a. Skin test results for suspected causative agents. Skin prick testing (SPT) and intradermal testing (IDT) were performed for sugammadex (B), remifentanil (U), and rocuronium (E); histamine was tested by SPT only as a positive control. Sugammadex SPT at 1 mg/mL elicited a large positive wheal-and-flare response, and IDT at 1:10 dilution also produced a clearly positive reaction, confirming true immunological hypersensitivity. In contrast, remifentanil (SPT at 0.05 mg/mL; IDT at 1:10) and rocuronium (SPT at 10 mg/mL; IDT at 1:10) produced no significant wheal response on either SPT or IDT, indicating negative results. These findings confirm sugammadex as the sole causative allergen and exclude remifentanil and rocuronium as triggers in this patient.

Figure 2b. Skin test results demonstrating hypersensitivity to sugammadex. SPT with sugammadex at 1 mg/mL elicited a clearly positive wheal-and-flare response. Subsequent IDT with serial dilutions of sugammadex (1:10, 1:100, and 1:1000) demonstrated consistent positive reactions with a concentration-dependent pattern, supporting a true immunologic response rather than a nonspecific irritant effect.

Histamine SPT, used as a positive control, showed an appropriate positive response. These findings strongly support the diagnosis of sugammadex-induced hypersensitivity.

SPT, skin prick test; IDT, intradermal test.