



Postextubation Upper-Airway Obstruction Leading to Negative-Pressure Pulmonary Edema and Hypoxic Cardiac Arrest Despite Apparent Recovery After Sugammadex Reversal: A Case Report

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Abstract

Postextubation upper-airway obstruction may cause negative-pressure pulmonary edema (NPPE) and, rarely, life-threatening cardiovascular collapse. Apparent clinical recovery after neuromuscular blockade reversal does not necessarily ensure adequate upper-airway function.

A 67-year-old woman underwent laparoscopic cholecystectomy under general anesthesia with induction of rocuronium. At the end of surgery, the train-of-four (TOF) count was 2, and sugammadex over 2 mg/kg was administered, after which four qualitative TOF responses subsequently returned. Before extubation, the patient opened her eyes and obeyed verbal commands, with breathing tidal volume of 6 mL/kg. However, thoracoabdominal asynchrony subsequently developed, and the patient was noted to have a decreased level of consciousness at the point of approximately 5 minutes after extubation. Severe hypoxemia progressed to bradycardia and pulseless electrical activity. Return of spontaneous circulation was achieved after 3 minutes of cardiopulmonary resuscitation. Post-resuscitation lung ultrasonography and chest radiography demonstrated pulmonary edema, which markedly improved within 5 hours.

This case highlights that clinically significant upper-airway obstruction may occur after apparent recovery from neuromuscular blockade with identification of qualitative TOF responses, despite administration of a clinically substantial dose of sugammadex. Such obstruction may rapidly progress to negative pressure pulmonary edema (NPPE) and hypoxic cardiac arrest.

Keywords: Airway Obstruction; Cardiac Arrest; TOF Monitoring; Neuromuscular Blockade; Negative-pressure Pulmonary Edema

Introduction

Negative-pressure pulmonary edema (NPPE) is a potentially life-threatening complication caused by forceful inspiratory efforts against an obstructed upper airway [1, 2]. Although it mostly occurs after acute postextubation airway obstruction, progression to cardiac arrest appears to be rare [3]. We report a case of severe postextubation upper-airway obstruction leading to NPPE, profound hypoxemia, and cardiac arrest despite apparently adequate recovery from neuromuscular blockade after sugammadex reversal.

The patient provided written informed consent for the publication of this case report and its accompanying images.

Case Presentation

A 67-year-old woman (height, 156 cm; weight, 55 kg) was scheduled for emergency laparoscopic cholecystectomy under general anesthesia. Her medical history included hypertension and variant angina. Preoperative electrocardiography and chest radiography showed no significant abnormalities. Preoperative B-type natriuretic peptide, troponin I, and creatine kinase-MB levels were within normal limits.

General anesthesia was induced with 10 mg of remimazolam and 50 mg of rocuronium (0.91 mg/kg) was administered to facilitate tracheal intubation. Anesthesia was maintained with sevoflurane inhalant. The operation lasted approximately 60 minutes and was uneventful, with



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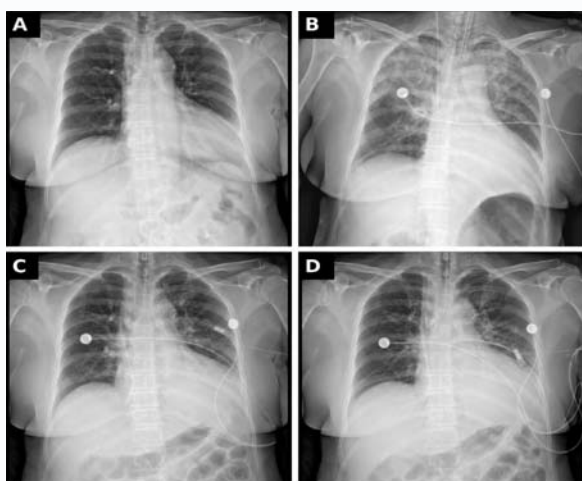


Figure 1: Serial chest radiographs obtained before and after surgery.

(A) Preoperative chest radiograph.

(B) Chest radiograph obtained immediately after surgery showing newly developed bilateral diffuse pulmonary edema.

(C) Follow-up chest radiograph obtained on postoperative day (POD) 1, approximately 5 h after completion of surgery, showing marked improvement in the bilateral pulmonary opacities.

(D) Chest radiograph obtained on POD 2 showing further resolution of the pulmonary edema.

stable intraoperative hemodynamics. Estimated blood loss was minimal (less than 50 ml), and only 100 ml of crystalloid solution was administered.

At the end of surgery, qualitative TOF monitoring showed two weak responses to TOF stimulation. Sugammadex 200 mg (3.64 mg/kg) was administered for reversal of neuromuscular blockade. Four responses to qualitative TOF stimulation subsequently returned. The patient opened her eyes with verbal stimulus and appropriately followed verbal commands. Her oxygen saturation was 100%, with spontaneous tidal volume of 6 ml/kg. Based on subjective clinical assessment, tracheal extubation was performed.

Immediately after extubation, spontaneous ventilation was present, with tidal volumes of approximately 300 ml. Subsequently, patient's breathing volume became progressively decreased and thoracoabdominal asynchrony developed, suggesting upper-airway obstruction. Despite administration of 100% oxygen with two-handed mask ventilation, adequate ventilation could not be maintained.

Approximately 5 min after extubation, progressive hypoxemia was followed by sinus bradycardia at approximately 50 beats/min, with detection of mental change and uncheckable SpO₂ value. Neither carotid nor femoral pulses were palpable, and pulseless electrical activity was diagnosed. Cardiopulmonary resuscitation was immediately initiated, and epinephrine 1 mg was administered intravenously. The patient was reintubated immediately during cardiopulmonary resuscitation. Return of spontaneous circulation was achieved approximately 3 minutes after CPR initiation.

After resuscitation, immediate lung ultrasonography demonstrated bilateral B-lines and transthoracic echocardiography showed preserved left ventricular systolic function without an apparent regional wall-motion abnormality. Post-event electrocardiography showed no ischemic changes. Cardiac biomarkers did not demonstrate a clinically significant increase, suggesting decreased possibility of cardiogenic origin.

Immediate follow-up chest radiography showed bilateral pulmonary edema, and approximately 5 h after the event, marked resolution of the pulmonary edema was identified (Figure 1). Formal transthoracic echocardiography on postoperative day 8 showed normal cardiac function. The patient was discharged on postoperative day 11 without significant cardiopulmonary or neurologic sequelae.

Discussion

The notable feature of this case was the development of severe upper-airway obstruction shortly after extubation despite apparently satisfactory recovery from neuromuscular blockade. The patient received 200 mg of sugammadex (3.64 mg/kg) when two weak responses to qualitative TOF stimulation were observed. Then, four qualitative TOF responses subsequently returned, and the patient opened her eyes and appropriately followed verbal commands before extubation. Nevertheless, effective ventilation deteriorated shortly after extubation, with thoracoabdominal asynchrony indicating inspiratory efforts against an obstructed upper airway. This was followed by acute pulmonary edema, severe hypoxemia, bradycardia, and pulseless electrical activity.

Negative pressure pulmonary edema (NPPE) results from vigorous inspiratory effort in the presence of upper-airway obstruction, which produces a substantial decrease in intrathoracic pressure. The resulting hemodynamic changes include increased venous return, an increased pulmonary capillary transmural pressure gradient, and increased left ventricular afterload, all of which favor the accumulation of fluid within the pulmonary interstitium and alveolar spaces. Studies analyzing pulmonary edema fluid in patients with NPPE have demonstrated a relatively low protein content, supporting a predominantly hydrostatic mechanism of edema formation [1, 2]. Furthermore, pulmonary edema associated with NPPE often improves rapidly after relief of the obstruction and restoration of adequate ventilation, presumably because alveolar fluid clearance remains functional [2, 3]. In the present case, the observed thoracoabdominal asynchrony indicated continued inspiratory effort despite upper-airway obstruction, supporting a mechanistic link between the postextubation obstruction and the subsequent development of pulmonary edema [4].

An important consideration in the present case is that four qualitative TOF responses were obtained after sugammadex administration, but the TOF ratio was not quantitatively measured. Because four responses TOF can be detected before the quantitative TOF ratio reaches 0.9, qualitative assessment alone could not rule out residual neuromuscular blockade in this patient [5, 6]. Similarly, in spite of clinically substantial dose administration of sugammadex, traditional signs of recovery (the ability to open the eyes, follow verbal commands, and generate an apparently adequate spontaneous tidal volume, etc.) are insufficiently reliable to exclude clinically relevant residual neuromuscular weakness [7-9].

With uncertainty of full recovery of neuromuscular blockade, upper-airway dilator muscles are particularly worthy of consideration during emergence because even minimal residual neuromuscular blockade can increase upper-airway collapsibility and attenuate the compensatory activity of the genioglossus muscle [10]. Under residual neuromuscular blockade, even minimal, there is possibility of impaired upper-airway dilator function and increased airway collapsibility despite relatively preserved respiratory timing and ventilation, potentially allowing inspiratory efforts to persist while

adequate upper-airway patency cannot be maintained [10, 11]. If inspiratory effort persists despite upper-airway closure, the resulting intrathoracic pressure gradient may promote rapid transvascular fluid movement into the pulmonary interstitium and alveoli [1, 2]. Although laryngospasm, a well-recognized cause of NPPE, could not be completely excluded [1, 2], the absence of stridor immediately after extubation, visualization of glottis opening during reintubation, and the lack of quantitative TOF monitoring favored residual neuromuscular blockade as a more likely contributing factor.

The patient's subsequent pulmonary edema after resuscitation was clinically consistent with NPPE, considering its physiologic cause and rapid resolution of disease course under restoration of airway patency. The patient's pulmonary edema likely further aggravated the already compromised gas exchange, and progressive hypoxemia was followed by sinus bradycardia and pulseless electrical activity, a sequence compatible with hypoxic cardiac arrest. Despite patient's history of variant angina and underlying disease of hypertension, no ischemic electrocardiographic changes, significant cardiac biomarker elevation, or regional wall-motion abnormalities were identified. Pulmonary thromboembolism, pneumothorax, fluid overload, and clinically apparent anaphylaxis were also not supported by the perioperative findings.

Conclusion

This case highlights that, without quantitative TOF monitoring, pharmacologic reversal with sugammadex does not necessarily ensure complete neuromuscular recovery. Also, apparent clinical recovery should not be regarded as equivalent to objective confirmation of neuromuscular recovery. Even after administration of a clinically substantial dose of sugammadex, restoration of four qualitative TOF responses, eye opening, and the ability to follow commands, clinically important upper-airway dysfunction may still occur when quantitative recovery has not been confirmed. When upper-airway obstruction develops under these circumstances, preserved inspiratory effort may generate sufficient negative intrathoracic pressure to produce NPPE, with potentially catastrophic progression to hypoxemia and cardiac arrest.

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