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Negative pressure pulmonary edema

Anju Romina Bhalotra

Department of Anesthesiology, Maulana Azad Medical College and Associated Lok Nayak Hospital, New Delhi, India

Dear Sir,

I read with interest the case report on postoperative negative pressure pulmonary edema (NPPE) that developed after the reversal of neuromuscular blockade with sugammadex by Lee et al. [1]. As the authors succinctly describe, NPPE develops when a patient makes a strong inspiratory effort against a closed upper airway/glottis, which generates a very negative intrathoracic—and hence intrapleural—pressure, leading to fluid accumulation in the alveoli and the generation of copious pink, frothy sputum. Undoubtedly, reversal of the neuromuscular blockade (NMB) with sugammadex may lead to a more rapid, complete reversal of the NMB and recovery of muscle power [2].

In the case described by the authors, at the time of the thyroidectomy, the patient was extubated only after she had regained full consciousness, spontaneous breathing, and peripheral motor power. When she suddenly complained of breathlessness despite 100% peripheral oxygen saturation, she was given a positive pressure of approximately 10 cmH_2O by the face mask and an injection of sugammadex (2 mg/kg). It is not clear whether the sugammadex was required and perhaps airway-opening maneuvers alone might have relieved her discomfort and the upper airway obstruction.

During the second surgery, anesthesia was maintained with O_2/N_2O/desflurane and a remifentanil infusion. At the end of the surgery, which lasted for about 250 minutes, the inhaled desflurane and N_2O, and intravenous infusion of remifentanil, were discontinued and seven minutes later, after she had regained consciousness and adequate spontaneous respiration and recovered her neuromuscular strength, she was given sugammadex at a dose of 2 mg/kg. At this time, she stopped breathing and began to gasp immediately following extubation, and her oxygen saturation dropped to 70%. The authors then say that "positive pressure ventilation was applied shortly by fitting a venturi mask on the airway that helped her regain self-respiration" and despite providing "continued assisted mask ventilation," the patient developed signs of frank pulmonary edema. In this second instance, the patient was very much awake and was likely to have been fighting and bucking on the endotracheal tube as the N_2O, desflurane, and remifentanil had been discontinued seven minutes earlier. All three of these agents have rapid recovery profiles. There is no mention of any other analgesia having been provided, so the patient was likely in severe pain after the rapid offset of remifentanil and was restless and agitated. The use of desflurane may have further contributed to the airway irritability and laryngospasm. The reversal of residual NMB with sugammadex also allowed this young and otherwise healthy woman to take large, strong breaths in the setting of a laryngospasm.

Further, it is not clear how the authors provided positive pressure and "continued assisted mask ventilation" with a VentiMask. The prevention of NPPE requires vigilance on the part of the conducting anesthesiologist and smooth extubation at the right time. In the second instance too, timely use of airway-opening maneuvers and application of positive pressure using 100% O_2 immediately after extubation may have prevented the development of NPPE, which might have been precipitated by laryngospasm. The role of sugammadex seems purely incidental.

In addition, as the young lady developed pulmonary edema on both occasions when she underwent surgery, it might have been advisable to have her undergo a complete cardiac evaluation before hospital discharge to exclude any cardiac cause of pulmonary edema.

Corresponding author: Anju Romina Bhalotra, M.D.
Department of Anesthesiology, Maulana Azad Medical College and Associated Lok Nayak Hospital, A-1/59 Safdarjang Enclave, New Delhi 110029, India
Tel: 91-01126183684, Fax: 91-01126183684
Email: drakgk@yahoo.co.in
ORCID: https://orcid.org/0000-0003-0700-2941

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