Negative pressure pulmonary edema following laryngospasm

Sir,

Negative pressure pulmonary edema (NPPE) is a potentially fatal condition with a multifactorial pathogenesis. NPPE is commonly encountered after upper airway obstruction with a large negative intrathoracic pressure generated by forced inspiration against an obstructed airway. The negative pressure leads to an increase in pulmonary vascular volume and capillary transmural pressure, damaging the alveolar–capillary membrane leading to a transudate of fluid into the pulmonary interstitium.[1,2] Type I NPPE typically occurs immediately after acute airway obstruction such as in laryngospasm or epiglottitis.[3,4] Type II NPPE typically occurs after relief of a chronic upper airway obstruction such as in adenotonsillar hypertrophy.[3] It is critical that anesthesiologists are aware of this uncommon condition as early detection of clinical signs is vital to the treatment and to patient outcome.

We describe a case of a 40-year-old male with a medical history of hypertension, diabetes, and end-stage renal disease who underwent surgery for arteriovenous fistula creation under general anesthesia. He had a reassuring airway and normal cardiac and pulmonary status on physical examination. He underwent an uneventful induction and intubation. His maintenance phase was also uneventful. During his emergence from anesthesia, he was ventilating spontaneously with good tidal volumes. His oropharynx was suctioned and he was extubated after opening his eyes. Following extubation, an oxygen mask was placed and there was no mask fogging noted by the anesthesiologist. In addition, there was no end-tidal CO_2 noted on monitors despite a good mask seal. The patient began to desaturate and became increasingly hypertensive and disoriented. Laryngospasm was suspected and an oral airway was placed along with administration of continuous positive airway pressure with minimal improvement using significant effort. Succinylcholine was given to relieve the laryngospasm which improved the ability to bag mask. However, the patient could not maintain oxygen saturation above 90% without significant assistance so the decision was made to reintubate. Upon reintubation, there was noted to be significant airway secretions that took considerable time to suction. The endotracheal tube (ETT) was passed and frothy pink secretions were noted to be filling the ETT requiring suctioning. The patient had secretions suctioned and was transported to the intensive care unit intubated for further management. The patient's chest X-ray demonstrated diffuse pulmonary infiltrates and pulmonary edema. He was managed with lung protective ventilation with PEEP, supplemental oxygen, and diuretics. He was extubated on postoperative day 3 without complications.

There are no specific interventions proven to prevent NPPE, but avoiding laryngeal irritation is likely to reduce the occurrence by preventing laryngospasm. With prompt diagnosis and treatment, NPPE may resolve within 24–48 h.[5] When diagnosis is delayed, these complications may be fatal. It is crucial for the anesthesiologist suspect this condition if patients suffer from an event of airway obstruction on extubation. If NPPE is suspected, the airway should be secured through ETT, supplemental oxygen should be administered, and positive pressure support should be given to enhance oxygenation. Low tidal volume ventilation is recommended. More aggressive rescue therapies for refractory hypoxemia include neuromuscular blockade, prone positioning, and extracorporeal membrane oxygenation.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. There is no identifying information used in this article.

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Conflicts of interest

There are no conflicts of interest.

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