Introduction

Negative Pressure Pulmonary Edema (NPPE) occurs due to high negative inspiratory pressure during upper airway obstruction causing a high pressure gradient for fluid to move into the interstitial and alveolar space. Rales, wheezing, desaturations, and copious pink frothy secretions are the classic signs.

Case Report

17 yo 66 kg female who suffered a nasal fracture after a cheerleading accident presented to a surgical center for closed reduction. PMH consisted of AV canal defect repaired at 5 months of age and seasonal asthma. Holter monitor one year prior showed intermittent 1st degree AV block & isolated PVCs. An echo 2 weeks prior to her nasal reduction revealed mild MR/TR with normal systolic function. Induction, insertion of LMA and maintenance of anesthesia were uneventful. Awake LMA removal resulted in respiratory distress with patient trying to take three enormous breaths against a presumed closed glottis. Positive pressure via mask with 100% oxygen was given but the patient became hypoxic requiring intubation. Patient was intubated but there was no ETCO2 or breath sounds. After reintubation 400 ml of pink, blood tinged secretions was suctioned. The patient went into cardiovascular collapse and ACLS was initiated. Pink frothy fluid continued to fill her ETT. She was given multiple doses of epinephrine, defibrillations and compressions for 30 minutes at the satellite surgery center. After return of spontaneous circulation she was transported to a tertiary pediatric hospital but arrested again in the ambulance and while in the ED. CXR showed worsening pulmonary edema, TEE revealed worsening LV function, and her ABG showed significant metabolic acidosis. The patient required hand ventilation with high airway pressures but was unable to keep O2 saturations above 90%. She continued to be hemodynamically unstable with declining respiratory status so the decision was made to initiate VA ECMO. She underwent balloon atrial septostomy on POD #1 due to left heart distention. After decannulation from EMCO on POD #3, she continued to require vasopressors until extubation on POD #5. She had fevers, tachypnea, tachycardia and HTN due to post-ECMO SIRS along with hallucinations that resolved before discharge on POD #16.

Discussion

Our patient suffered from NPPE due to high negative airway pressure against an obstructed upper airway (Muller maneuver) secondary to presumed laryngospasm. High negative airway pressure causes a disturbance in the hydrostatic and oncotic pressure in the pulmonary vasculature. Pulmonary edema fluid usually has a low protein concentration supporting a hydrostatic mechanism for NPPE [1]. Although this patient had an AV canal repair in infancy, there did not appear to be any predisposing factors from a cardiac standpoint. Her severe, yet temporary, cardiac dysfunction was likely Takotsabu cardiomyopathy secondary to the massive epinephrine doses that she received throughout the resuscitation. Our patient suffered refractory hypoxemia, cardiomyopathy, and hemodynamic instability despite aggressive treatment so ECMO was the only remaining option. This sequela of NPPE raises interesting points of discussion including early recognition of laryngospasm, importance of positive pressure ventilation, aggressive resuscitation, and early transport to a tertiary level facility. It also highlights the importance of the care team model and crisis resource management training.

References: