Post-laryngospasm pulmonary edema

Lt. Col. LEON F. DEISERING, CRNA, MSN, USA, AN
Maj. DALE A. DOUGLASS, CRNA, MS, USA, AN
Fort Hood, Texas

Post-laryngospasm pulmonary edema can be directly related to anesthesia in two types of situations.

Two case studies are presented concerning these situations and their causative factors. It is postulated that laryngospasm-induced pulmonary edema may be the result of hypoxia, intrathoracic pressure changes or intravascular fluid shifts. While this phenomenon is rare, early assessment and rapid intervention are essential preventative measures.

Pulmonary edema has multiple causes, two of which can be related directly to anesthesia. One is acute upper airway obstruction, and the other is an adverse reaction following the administration of naloxone for reversal of analgesia. In the following two case presentations, laryngospasm during the emergence phase of general anesthesia occurred and was complicated by the development of pulmonary edema. The patient in case I also had received naloxone for reversal of analgesia.

Case I

A 20-year-old black male was admitted for elective repair of a right inguinal hernia. Based on a negative medical history and normal laboratory findings, including chest x-ray and a 45.5 hematocrit, an ASA I rating was assessed.

After an inadequate subarachnoid block was performed, the patient was prepared for general anesthesia with 0.4 mg of intravenous atropine and 100% oxygen by mask. Intubation was accomplished without difficulty, utilizing 400 mg of thiopental for induction and 30 mg of intravenous succinylcholine for relaxation.

Anesthesia was maintained with 200 µg of intravenous fentanyl and 66% nitrous oxide, 33% oxygen and 0.5% isoflurane via a semiclosed circle system and mechanical ventilation. Breath sounds were bilaterally clear and equal before and after the patient was placed on the ventilator.

The surgical procedure lasted 40 minutes, with an estimated blood loss of 50 ml and a total of 1200 cc crystalloid intravenous replacement. During emergence, 0.2 mg of naloxone were given intravenously, resulting in a combative, difficult-to-control patient.

Oral suctioning and extubation were performed, followed by an immediate laryngospasm and glottic closure. Vigorous inspiratory efforts were demonstrated with substernal and intercostal retraction, while 100% oxygen via continuous positive airway pressure (CPAP) mask was applied. Without additional intervention, the apneic episode resolved, and the patient was transferred to the recovery area breathing spontaneously.

Approximately 90 minutes later, the patient became restless and exhibited tachypnea and tachycardia, with a preference for the sitting position. Bilateral rales were present by auscultation. Arterial blood gases on 35% mask oxygen were pH 7.26, pCO 2 60.8, pO 2 41.6 and 74% oxygen satura-
tion. The chest x-ray report indicated "fluid overload—pulmonary edema."

Following nasal intubation, approximately 200 ml of frothy, pink sputum was suctioned from the trachea. The patient was given morphine and furosemide, maintained on supplemental oxygen, positive end-expiratory pressure (PEEP) and mechanical ventilation overnight in the intensive care unit. The blood gases and chest x-ray were markedly improved on the first postoperative morning, allowing for ventilator weaning and extubation. The patient was discharged on the second postoperative day in satisfactory condition and without evidence of sequelae.

**Case II**

A 10-month-old infant male (ASA I, 5 kg) was admitted to the same-day surgical center for an elective examination under anesthesia by the ophthalmology service. On arrival, the child's lungs were clear and laboratory results were normal. Anesthesia was maintained with halothane, nitrous oxide and oxygen via an endotracheal tube. A 50 ml dose of crystalloid intravenous solution was administered during the 35-minute eye examination.

During emergence, robust movement of extremities, crying and coughing efforts were evident. After the infant was suctioned and extubated, the vigorous crying continued for about 30 seconds, and then respirations became diminished. Breathholding followed, and pulse rate dropped from 150 to 50 beats per minute.

Despite positive pressure oxygen by mask, the infant's color became dusky, and atropine 0.2 mg, followed by intravenous succinylcholine 15 mg, were given. His dusky color continued and, during laryngoscopy, blood-tinged froth was observed around his vocal cords. After intubation, the infant's bilateral breath sounds were equal, but diminished. Approximately 15 ml of frothy secretions were suctioned from his trachea.

While respirations were controlled with 100% oxygen, an arterial blood gas was drawn (results: pH 7.16, pCO$_2$ 78, pO$_2$ 52.2, oxygen saturation 75.7). The infant's pulse rate returned to a range of 140-150 beats per minute, and a chest x-ray was ordered that revealed apparent pulmonary edema.

**Figure 1**

Possible mechanisms in the etiology of pulmonary edema associated with upper airway obstruction.

Adapted with permission from Travis KW, Todres ID, Shannon DC: Pulmonary edema associated with croup and epiglottitis. *Pediatrics.* 59:695-698.
The infant was transferred to the intensive care unit for overnight ventilation and medical management for pulmonary edema.

Discussion

The development of pulmonary edema during acute upper respiratory obstruction has been reported both in children and adults. There are several mechanisms involved (Figure 1), all related to altered gas exchange and changes in intrathoracic pressures.

Hypoxia causes pulmonary arteriolar constriction. This, along with severe hypoxemia and anxiety, can result in a massive increase in the levels of circulating catecholamines, as evidenced by a systemic vasconstriction and a shift of the blood volume to the pulmonary system. The net result is increased pulmonary perfusion pressures, which favor the formation of edema across a membrane that has increased permeability.

The extreme negative pressure created by attempts to breathe against a closed glottis (Mueller maneuver) overcomes the ability of the lymphatics to move fluid and may result in destruction of the anatomical integrity of the capillary walls of the pulmonary microvasculature. This extreme negative pressure also enhances venous return to the right side of the heart, increasing preload and pulmonary hydrostatic pressure, resulting in pulmonary hyperemia and edema.

There have been numerous reports of adverse effects following naloxone administration. These include hypertension, pulmonary edema, ventricular arrhythmias and cardiac arrest. Adverse reactions to naloxone may be related to the reversal of analgesia and profound stimulation of the central nervous system. There is an outpouring of adrenal catecholamines which can lead to pulmonary vasconstriction, pulmonary hypertension and possibly increased pulmonary vascular permeability.

Naloxone will inhibit further the endogenous pain suppression pathway and may permit unopposed noradrenergic transmission from medullary centers that can precipitate pulmonary edema.

In the first case, both suspected factors for pulmonary edema were present. The patient was given intravenous naloxone 0.2 mg to reverse analgesia, resulting in a combative, difficult-to-control patient who developed airway obstruction postextubation.

In the second case, the infant’s robust movements, coughing and vigorous crying may have increased the levels of catecholamines substantially enough to create systemic vasconstriction and a shift of blood volume to the pulmonary system, with the immediate result of pulmonary edema.

Summary

Both the assessment of laryngospasm during emergence from general anesthesia and the intervention should be rapid. Any obvious precipitating causes, such as blood, secretions or other matter should be removed from the oral cavity in and around the larynx. The mandible should be pulled upward and forward, while 100% oxygen is being administered by mask with gentle CPAP. Should these measures fail and cyanosis develop, the patient should be given a rapid-acting muscle relaxant, and reintubation should be considered.

REFERENCES


AUTHORS

Lt. Col. Leon F. Deisering, CRNA, MSN, a graduate of the Anesthesiology for Army Nurse Corps Officers Course at William Beaumont Army Medical Center in El Paso, Texas, received his BSN from Marymount College of Kansas in Salina and his MSN from The Catholic University of America, Washington, D.C. Having retired from active duty, Lt. Col. Deisering is currently director of Mercy Hospital School of Anesthesia, Portland, Maine.

Maj. Dale A. Douglass, CRNA, MS, graduated from the Anesthesiology for Army Nurse Corps Officers Course at Walter Reed Army Medical Center in Washington, D.C. He received a BS degree in business administration from Tarleton State University in Stephenville, Texas, a BSN from Texas Christian University in Fort Worth, Texas, and an MS in anesthesia nursing from the State University of New York at Buffalo. Maj. Douglass is currently director of the Phase II Anesthesia Program at Darnall Army Community Hospital, Fort Hood, Texas.

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