Laryngospasm is one of the more common forms of airway obstruction encountered by an anesthetist. Therapy usually is straightforward, with resolution of the obstruction normally occurring within minutes. In some cases, however, the patient’s vigorous inspiratory efforts may lead to a rapidly deteriorating form of pulmonary edema.

Two cases are presented that are quite typical of the development of this complication. The etiology, recognition and management of this form of non-cardiac pulmonary edema is discussed.

Pulmonary edema is an infrequent, but dramatic, complication of airway obstruction. Two cases are presented that are quite typical of the development of this complication. Both patients, who were young and in good health (ASA I)—developed pulmonary edema associated with upper airway obstruction (laryngospasm) during general anesthesia. Both quickly developed the typical clinical signs of pulmonary edema—tachypnea, hypoxia, pink frothy sputum, bilateral interstitial infiltrates, etc.—that necessitated ventilatory support with supplemental oxygenation and positive end-expiratory pressure (PEEP).

Vigorous inspiratory efforts against a totally obstructed upper airway may result in acute non-cardiogenic pulmonary edema (NCPE). Inspiration against the closed glottis generates large negative intrathoracic pressures across the alveolar-capillary membrane, altering membrane permeability. Interstitial water accumulates, leading to pulmonary edema. Laryngospasm, a well-recognized anesthetic complication, can produce total airway obstruction leading to pulmonary edema.

Treatment of NCPE includes ventilatory support with supplemental oxygenation and PEEP, continuous positive air pressure (CPAP), fluid restriction, diuretics and possibly steroids. Arterial blood gases (ABG) and chest films (CXR) must be monitored.

Pulmonary edema is a frequent cause of respiratory failure. Since etiology and treatment differ between cardiogenic and non-cardiogenic pulmonary edema, it is important to distinguish the cause quickly. Pulmonary edema that occurs subsequent to an episode of airway obstruction should alert the anesthetist to this non-cardiogenic source.

Anesthetists frequently are called upon to deal with different forms of airway obstruction—laryngospasm being among the most common. One of the complications of this condition is a form of pulmonary edema—a relatively rare occurrence, but a potentially catastrophic one. Two cases are presented that document the development and treatment of this anesthetic complication.

Case one
A 19-year-old active duty Marine PFC was admitted for excision of a left-sided gynecomastia. Preanesthetic evaluation revealed a young, healthy 72 kg ASA I male. Preoperative laboratory tests, complete blood count (CBC) and urinalysis (UA) were within normal limits.
General anesthesia was induced by mask with oxygen, nitrous oxide and halothane. Airway management proved difficult, with the patient coughing initially, then going into laryngospasm, which was refractory to positive pressure ventilation. The patient was given pentothal and succinylcholine and intubated. Anesthesia was maintained with oxygen, nitrous oxide, halothane and fentanyl (Sublimaze®), and the surgery was completed uneventfully.

Upon admission to the recovery room, the patient was awake and oriented, but tachypneic and dyspneic. His arterial blood gases (ABGs) on 100% oxygen via mask were pH 7.21, \( pO_2 \) 61, \( pCO_2 \) 65, base excess (BE) -2.3. A CXR revealed bilateral interstitial infiltrates consistent with pulmonary edema. The patient was taken to the intensive care unit (ICU), where he was maintained on CPAP and oxygen.

By the following morning his respiratory status had improved greatly, as demonstrated by ABG analysis—pH 7.47, \( pO_2 \) 138, \( pCO_2 \) 32 on intermittent mandatory ventilation (IMV) 0, \( FiO_2 \) 28% and a CPAP of 2.5 cm H2O. His breath sounds were clear, and the repeat CXR showed marked improvement. The patient was extubated with a total ventilatory support time of 18 hours. The patient's preoperative course proceeded uneventfully, and he was discharged on the fourth postoperative day.

**Case two**

A 23-year-old active duty Navy corpsman was admitted through the emergency room with a 24-hour history of right lower quadrant pain, diagnosed as appendicitis. Preoperative laboratory tests (CBC, UA, Lytes) were within normal limits, and a preoperative anesthetic evaluation revealed a 75 kg ASA 1E male. The patient was scheduled for appendectomy under general anesthesia.

Anesthesia and surgery commenced without incident. Upon extubation, the patient developed laryngospasm refractory to positive pressure ventilation and subsequently was reintubated after the use of succinylcholine. He was ventilated with 100% oxygen for approximately 15 minutes, after which he began breathing spontaneously with adequate minute ventilation. The patient was extubated when stable and awake and taken to the ICU for recovery.

On arrival in the ICU, the patient was tachypneic and began producing a frothy pink sputum. ABGs on 60% oxygen were pH 7.96, \( pO_2 \) 62, \( pCO_2 \) 48. A CXR revealed bilateral interstitial infiltrates consistent with pulmonary edema. Auscultation of lung fields revealed bilateral rales and rhonchi.

Oxygen was increased to 100% by mask, furosemide and morphine were administered and ABGs were monitored. Since the patient's \( pO_2 \)s remained in the 70s, he was reintubated and placed on ventilatory support (40% oxygen, PEEP of 5 cm H2O), with subsequent improvement of his ABGs to pH 7.45, \( pO_2 \) 89, \( pCO_2 \) 37. A Swan-Ganz catheter revealed a normal hemodynamic profile.

Within 12 hours, the patient's respiratory status improved markedly. Upon an IMV of 4, 40% oxygen and 5 cm PEEP, his ABGs were pH 7.38, \( pO_2 \) 145, \( pCO_2 \) 44. A CXR revealed greatly reduced infiltrates. The patient was gradually weaned and extubated that day, transferred to the ward the next morning and discharged home on the fourth postoperative day.

**Discussion**

Pulmonary edema is a frequent cause of acute respiratory failure. The knowledge that it can be produced secondary to an episode of airway obstruction is relatively new.

Acute airway obstruction was suggested as the probable cause of a form of non-cardiogenic pulmonary edema (NCPE) in 1977, when three cases involving strangulation, interrupted hanging and laryngeal tumor went on to develop pulmonary edema. Prior to 1977, NCPE had been documented in bowel infarction, gram negative sepsis, head injury, pulmonary hypertension and vagotomy, but never in relation to airway obstruction. Since this initial observation, a number of case reports have been submitted in which acute upper airway obstruction has been discussed as the etiology behind the rapid development of NCPE. Upper airway obstruction as a result of an acute infectious process (epiglottitis, laryngotracheitis, spasmodic croup) has been presented as a cause of pediatric NCPE. Acute asthmatic episodes also have been implicated.

As can be seen in Table I, a variety of pathologic states have been implicated in the production of NCPE and are of concern to the anesthetist.

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**Table I**

<table>
<thead>
<tr>
<th>Causes of non-cardiogenic pulmonary edema</th>
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<tbody>
<tr>
<td>Bowel infarction</td>
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<td>Gram negative sepsis</td>
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<td>Head injury</td>
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<td>Pulmonary hypertension</td>
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<td>Vagotomy</td>
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<td>Airway obstruction:</td>
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<td>Strangulation</td>
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<td>Laryngospasm</td>
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<td>Epiglottitis</td>
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<td>Foreign bodies</td>
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<td>Obstructive neoplasm</td>
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<td>Asthma</td>
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Laryngospasm-induced pulmonary edema

Laryngospasm is a well-recognized anesthetic complication. The bronchial and glottic spasms produced can cause complete airway obstruction.

A patient who attempts inspiration against an obstructed airway can generate large (greater than 80 torr) negative intrathoracic pressures. These negative pressures generate a large pressure difference across the alveolar-capillary membrane. According to the Starling equation, a low interstitial-alveolar pressure compared with intravascular capillary pressure favors transudation of fluid from the interstitium and, eventually, into the alveolus. Capillary wall integrity is disrupted as the high negative pressures are transmitted to peribronchial and perivascular spaces, leading to interstitial water accumulation. With relief of the airway obstruction, the resultant rise in pleural pressure forces this interstitial water into the alveolus causing pulmonary edema.

Diagnosis and treatment

The pulmonary edema resulting from acute airway obstruction usually is immediate, but can be delayed for hours after the initial insult. The clinical picture is one of immediate or delayed respiratory distress with tachypnea, shortness of breath and production of pink frothy sputum. Auscultation reveals rales and rhonchi, and the CXR shows diffuse interstitial and alveolar infiltration. The ABGs demonstrate hypoxia and hypercarbia.

Initial treatment is aimed at relieving the cause of the airway obstruction. With laryngospasm, this includes positive pressure ventilation with supplemental oxygenation, alleviating the stimulus (secretions, light anesthesia) and succinylcholine, if positive pressure ventilation fails to relieve the obstruction. At this point, the patient may or may not require, intubation and followup ventilatory support.

With manifestations of respiratory distress and diagnosis of NCPE, ventilatory support with supplemental oxygenation and PEEP/CPAP usually is necessary. Fluid restrictions, diuretics and possibly steroids are supplemental therapeutic interventions. ABGs and serial chest x-rays should be monitored continually, and the patient weaned from ventilatory support as the edema resolves. This type of pulmonary edema commonly shows rapid resolution, often within 24 hours with appropriate treatment.

Conclusion

Laryngospasm, such as that resulting from airway manipulation during anesthesia, can lead to airway obstruction. The large negative pressures produced during the patient’s inspiratory attempts against the closed glottis can alter pulmonary alveolar and capillary permeability, causing pulmonary edema.

With proper diagnosis and appropriate treatment, this form of pulmonary edema shows rapid resolution.

These facts should alert the anesthetist and focus attention on this non-cardiac source when an episode of airway obstruction is followed by pulmonary edema.

REFERENCES


AUTHORS

Christopher C. Ferguson, CRNA, received his BSN from the University of Utah in 1976. He was a 1981 graduate of the BSNA program, George Washington University, Washington, DC. At the time this paper was written, he was on active duty at the Navy Hospital, Camp Pendleton, California. He is now a senior nurse anesthetist at Fergusson Specialty Hospital, Grand Rapids, Michigan.

Carol Mann, CRNA, received her BS in nursing from the University of Bridgeport, Connecticut, and her BS in anesthesia from George Washington University, Washington, DC. She currently is a lieutenant in the Navy Nurse Corps, Naval Hospital, Camp Pendleton, California.

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