CLINICAL VIGNETTE

Respiratory Insufficiency After Surgery: A Case of Postobstructive Pulmonary Edema

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Postobstructive pulmonary edema (POPE) is a well-recognized but relatively rare complication of upper airway obstruction. It typically develops rapidly and can be life-threatening if not diagnosed promptly. Diverse causes of upper airway obstruction leading to POPE have been described. Postanesthetic laryngospasm has been implicated as the most common cause of this syndrome in adults. The pathogenesis of POPE is multifactorial and the clinical features are well defined. Patients generally recover rapidly with mainly supportive care. This syndrome should be included in the differential diagnosis of perioperative respiratory insufficiency after an episode of airway obstruction. We present a case of POPE due to laryngospasm after an uncomplicated rotator cuff repair.

Case Report

A 62-year-old man reported right shoulder pain and disability for several years. Magnetic resonance imaging (MRI) revealed a full thickness tear of the supraspinatous and infraspinatous tendons and the patient was scheduled for arthroscopic surgical repair. Past medical history included hypertension, acid reflux, obesity, and hyperlipidemia. There was no history of previous cardiovascular or pulmonary disease. There was no history of obstructive sleep apnea. The patient had undergone several previous surgeries without any surgical or anesthetic complications. There were no known drug allergies and his current medications included lisinopril, aspirin (discontinued a week prior to surgery), and omeprazole.

Preoperative vital signs were significant for a blood pressure (BP) of 162/97 mmHg, pulse of 96 beats/min, and respiratory rate (RR) of 12 breaths/min. His oxygen saturation (O_2 sat) was 97% on room air. The remainder of the preoperative physical examination was normal. The patient underwent arthroscopic right rotator cuff surgery under general anesthesia with a right interscalene block. Intubation was without incident and the surgery lasted approximately three hours. The patient had stable vital signs throughout the procedure. He received a total of 2.6 liters of intravenous fluids during the procedure and the estimated blood loss was 100 mL. No intraoperative complications were noted. Following the surgery, the patient was successful extubated by the Anesthesiologist.

Shortly thereafter, the patient was noted to have respiratory distress with significant accessory respiratory muscle use and according to notes from the operating room, was "attempting to breath against a closed airway". The Anesthesiologist suspected laryngospasm and performed a jaw thrust and the patient's "airway appeared open". There was no emesis and no evidence of gastric contents on oral suctioning. The patient's respiratory distress resolved rapidly and he did not require reintubation and was transferred to the recovery room. Once in the recovery room, hypoxia was noted with an O_2 sat around 80% on 2L nasal cannula O_2 and a facemask was placed with 15L O_2 and the O_2 sat improved to 90%. Initial vital signs in the recovery room were significant for elevated BP of 220/110 mmHg. The patient was given intravenous labetolol with improvement of his blood pressure.

On assessment several minutes later, the patient complained of right shoulder pain but denied having any chest pain, shortness of breath, or cough. He appeared comfortable and had no apparent respiratory distress. Examination showed normal vital signs (BP 110/70 mm Hg, pulse 68 beats/min, temperature 97° F, and RR 16 breaths/min) except for an O_2 sat of 80% on room air. With 15 L face mask O_2, the O_2 sat was 100%. Pulmonary examination was notable for bilateral basilar rales and diminished breath sounds at the right lung base. Cardiac examination was normal and there was no peripheral edema. Laboratory evaluation showed white blood cell count of 17,000/μL, B-naturetic peptide (BNP) level of < 20 pg/mL, and negative troponin I of < 0.04 ng/mL. Basic metabolic panel was normal. Arterial blood gas (ABG) done on room air revealed a pH of 7.36, pCO_2 40.8 mmHg, pO_2 42.2 mmHg, and O_2 sat of 72.8% consistent with significant hypoxia. Electrocardiogram (EKG) was normal without ischemic changes. Chest radiograph (CXR) showed vascular congestion and mild interstitial edema, an elevated right hemidiaghram, and an opacity of the right lung base consistent with atelectasis versus pneumonia (**Figure 1a**). Echocardiogram was normal with an estimated ejection fraction of greater than 75% and no evidence of valvular abnormalities or diastolic dysfunction.

The patient was diagnosed with acute postobstructive pulmonary edema (POPE) as a result of postanesthetic laryngospasm and was treated with supplemental O_2 and intravenous furosemide. Initially, empiric antibiotics for

possible aspiration pneumonia were given but discontinued after 24 hours given the patient's rapid clinical and radiographic improvement. The elevation of the right hemidiaphragm was most likely related to the nerve block and resolved rapidly. The patient's hypoxia improved rapidly and within 48 hours his lung examination, O_2 sat, and CXR normalized (**Figure 1b**). The patient was discharged home and remained well at a follow-up appointment several weeks later.

Discussion

Pulmonary edema in association with upper airway obstruction was first described in the literature in1927, when it was observed that prolonged inspiration against a fixed resistance resulted in pulmonary edema in an experimental dog model[1]. Subsequently, POPE was first described in humans in the 1960s as an autopsy finding in victims of suicidal hanging[2]. The first case series in adults was published in 1977 reporting three patients who developed POPE after acute airway obstruction from a laryngeal tumor, strangulation, and interrupted hanging[3]. Since then, a variety of causes have been reported, with postanesthetic laryngospasm being the most common[4]. Several possible pathogenic mechanisms contributing to POPE have also been described. The clinical features of POPE are well defined and treatment is mainly supportive.

The rapid onset of pulmonary edema following upper airway obstruction should alert physicians to the possible diagnosis of POPE. POPE which occurs after an acute event causing airway obstruction, such as laryngospasm, is often termed Type I. Pulmonary edema which follows the relief of chronic upper airway obstruction, such as after tonsillectomy or removal of upper airway tumors, is often called Type II[5]. Predisposing factors for POPE have been identified and include conditions that make upper airway obstruction more likely such as: (1) anatomically difficult intubation; (2) nasal, oral, or pharyngeal surgical site or pathology; and (3) obesity with obstructive apnea[6]. Numerous causes of POPE have been described, with postanesthetic laryngospasm being the most frequent cause in adults (>40% of cases) (**Table 1**)[4,7]. Among pediatric patients, acute upper airway obstruction from epiglottitis and croup comprise greater than 75% of reported cases of POPE[8].

The true incidence of POPE is uncertain and varies depending on the etiology. Furthermore, the incidence is likely underestimated because many cases may not be identified clinically or misdiagnosed[5,9]. The incidence of laryngospasm with general anesthesia is estimated to be 9 in 1,000 patients in general population[10]. In patients with laryngospasm, it is believed as many as 10% develop subsequent pulmonary edema[11]. Thus, the overall incidence of POPE may be as much as 1 per1000 in adults undergoing general anesthesia. Some studies, in fact, have reported incidence rates of POPE in this population from 0.05-1%[9,12]. In children who often develop acute airway obstruction from croup, epiglottitis, or postextubation subglottic edema, studies have reported an incidence of POPE of up to 9.6 percent[13].

The pathogenesis of pulmonary edema formation following relief of upper airway obstruction is likely multifactorial[5,9,14]. It is generally agreed upon that the principal mechanism is related to the markedly negative intrathoracic pressures generated acutely as a result of attempted inspiration against a closed glottis (obstructed airway). For this reason the syndrome is often referred to as "negative pressure pulmonary edema" and is often seen in young athletic patients. This negative pressure presumably increases venous return from the systemic circulation and increases pulmonary blood volume. The increased blood volume in the right ventricle creates a bowing effect on the left ventricle that decreases the compliance of the left ventricle with a resultant decrease in stroke volume. The end result of this cascade of events increases pulmonary hydrostatic pressure leading transudation of fluid from capillary bed to the interstitium with resultant pulmonary edema[11,14].

Support for the hypothesis that hydrostatic mechanisms are the cause of pulmonary edema in POPE comes from a study of pulmonary edema fluid/plasma protein ratios[7]. Based upon previous studies, a ratio of <0.65 was consistent with hydrostatic pulmonary edema, while a ratio of >0.75 was consistent with high-permeability pulmonary edema (from various types of acute lung injury). Patients with a ratio between 0.65 and 0.75 were thought to have a mixed etiology or have early absorption of the edema fluid. Of 341 total patients in one study of pulmonary edema, POPE was diagnosed to be the cause in ten patients. Fluid analysis of these ten patients showed mean edema fluid/plasma protein ratio of 0.54±0.15. The authors concluded that this ratio proved the hydrostatic mechanism for edema fluid formation in cases of postobstructive pulmonary edema[7].

Other factors have also been postulated to explain why pulmonary edema often occurs after relief of the airway obstruction rather than during the actual episode of obstruction. It has been suggested that in addition to forceful inspiratory efforts, expiratory efforts against a closed airway create an auto-PEEP (positive end expiratory pressure) effect leading to some degree of increased intrathoracic pressure as well. The increased intrathoracic pressure prevents venous return, increased pulmonary blood volume, and may actually protect against the formation of pulmonary edema during the obstructing episode.[14] After the obstruction is relieved, the auto-PEEP resolves rapidly. There is a subsequent rush of fluid to the pulmonary vasculature and a decrease in alveoli pressure creating a pressure gradient for transudation of fluid leading to pulmonary edema[9,11].

Other authors believe other mechanisms are important and have proposed that widespread disruption of capillary

membranes due to hypoxia and/or mechanical stress also contributes to POPE[11,14]. In support of this theory, evidence of hemorrhagic lesions on the mucosal linings of the trachea and large bronchi has been reported during bronchoscopy in some patients with POPE. Investigators have concluded that this finding indicates disruption of the pulmonary capillaries and argues against the hydrostatic pressure theory.[15] These findings may explain why many patients with POPE are observed to have pink frothy sputum. In addition, contradictory to the findings reported above, some studies have found elevated edema fluid/plasma protein ratios (>0.75) in patients diagnosed with POPE indicating increased vascular permeability[16]. However, it has been argued that delayed fluid collection resulted in erroneous results in these studies as the fluid clearance rate of intact alveoli can be as high as 14% per hour.[7]

A final possible contributing mechanism for POPE involves the massive release of catecholamines that is triggered by hypoxia and stress following airway obstruction.[9] The hyperadrenergic response causes constriction of blood vessels peripherally shifting fluid to the pulmonary vasculature. In addition, increased adrenergic tone also induces pulmonary vasoconstriction leading to increased pulmonary capillary wedge pressure favoring the development of pulmonary edema[14, 17]. Regardless of which mechanism predominates in an individual patient, the combination of increased hydrostatic pressure, increased vascular permeability, and increased adrenergic tone are believed to form the pathogenesis of POPE.

The diagnosis of POPE is based on clinical features and radiological findings. The onset of pulmonary edema is typically recognized with minutes of the relief of the airway obstruction[4]. However, some case reports have documented a delayed onset of several hours (up to 4-6 hours) and authors have cautioned against premature discharge of surgical outpatients who have had an episode of perioperative airway obstruction[9, 14]. The main clinical findings include shortness of breath and increased work of breathing. Examination often reveals hypoxia, rales, and pink frothy sputum or hemoptysis. Chest radiograph typically shows bilateral pulmonary interstitial infiltrates consistent with pulmonary edema[5, 8, 11].

The main differential diagnosis includes aspiration of gastric contents, myocardial ischemia and cardiogenic pulmonary edema, pulmonary embolism, and anaphylaxis. In a patient with no cardiac history, aspiration pneumonia is often the main competing diagnosis. The rapid resolution of symptoms and radiographic findings in patients with POPE are useful in distinguishing this syndrome from aspiration. In aspiration, the rapid onset and resolution of radiographic findings does not typically occur and radiological changes often lag behind clinical status for several days[11].

Treatment of POPE is mainly supportive and full and rapid recovery can be expected[5,9]. Many patients need only supportive therapy. Some patients have significant hypoxemia and may require continuous positive airway pressure (CPAP) and/or temporary intubation[7,8]. Furosemide is frequently given to promote diuresis, but its efficacy in the management of POPE remains unclear. Corticosteroids have been used but their role is controversial[14]. Regardless of the particular treatment modalities, patients typically show rapid improvement and resolution is usually complete within 36 hours[8,9,14]. The key factors in successful management of patients with POPE appear to be immediate reestablishment and maintenance of a patent airway and oxygen supplementation. Rarely, death has occurred from severe hypoxia leading to brain damage or from progression to acute respiratory distress syndrome (ARDS)[5].

In conclusion, POPE is a well-described, relatively uncommon, and often unrecognized complication following an episode of upper airway obstruction. Airway obstruction from a variety of causes can lead to POPE, but postanesthetic laryngospasm is by far the most common etiology in adults. The diagnosis is typically straightforward but other causes of respiratory insufficiency should be considered. The pathogenesis of POPE is multifactorial and treatment is largely empiric. Most patients recovery quickly without significant sequelae. This syndrome should be included in the differential diagnosis of perioperative respiratory insufficiency after a possible episode of airway obstruction. Heightened physician awareness of POPE is important to ensure prompt recognition and proper treatment.

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FIGURE LEGEND:

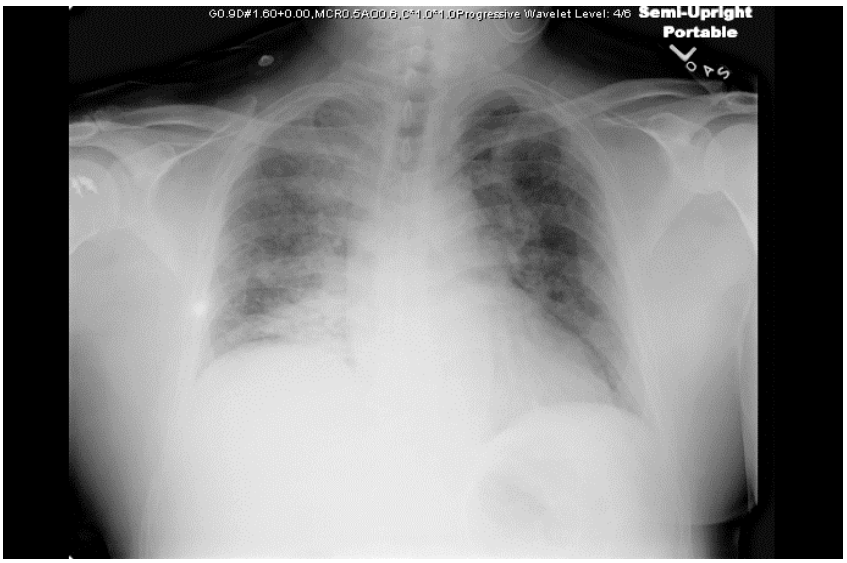

Figure 1a

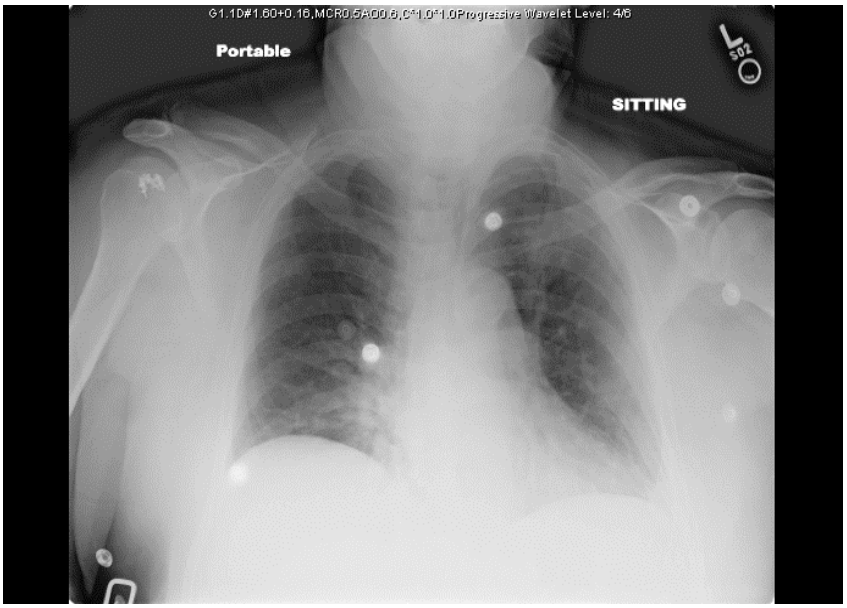

Figure 1b

Figure 1: a. Chest x-ray showed interstitial infiltrate, right lower lobe atelectasis/infiltrate, and elevated right hemidiaghram. b. Chest x-ray showed resolution of interstitial infiltrate and right lower lobe atelectasis/infiltrate and improved right elevated hemidiaghram two days later.

Table 1 . Selected Causes of Postobstructive Pulmonary Edema[5]

A. Type I (after acute airway obstruction)
   a. Postextubation laryngospasm
   b. Strangulation
   c. Epiglottitis / croup
   d. Choking / foreign-body aspiration
   e. Endotracheal tube obstruction (eg. biting of the endotracheal tube)
   f. Upper airway tumors

B. Type II (after surgical relief of chronic upper airway obstruction)
   a. After tonsillectomy/adenoidectomy
   b. After upper airway tumor removal
   c. Hypertrophic redundant uvula