Negative Pressure Post-Tracheal Extubation Alveolar Hemorrhage

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Negative pressure pulmonary edema is an uncommon complication of extubation of the trachea (≅0.1%) mostly caused by laryngospasm (1). Upper airway obstruction from glottis closure leads to marked inspiratory efforts, which generate very negative intrathoracic pressure. This may cause pulmonary edema (1,2) and, rarely, hemoptysis (3,4). This report is the first to document both bronchoscopic and computed tomography (CT) findings consistent with alveolar hemorrhage and capillary failure in this setting.

Case Report

A healthy 21-yr-old man had elective surgery (a nerve graft for a traumatic spinal nerve injury). His medical history and physical examination before surgery were unremarkable except for symptoms and signs related to right trapezius muscle palsy. Sampling of the saphenous external nerve and grafting of the spinal nerve were performed under general anesthesia (IV fentanyl and propofol). The tracheal intubation, anesthesia, mechanical ventilation, and surgical procedure were uneventful (the patient had stable blood pressure and oxygen saturation). Over the operative period (≅3.5 h), 2000 mL of lactated Ringer's solution was given, and subsequently the trachea was extubated. Immediately thereafter, the patient developed marked respiratory distress, followed by frank hemoptysis and decreased arterial oxygen saturation. Whether stridor was present is unknown. The chest radiograph showed bilateral pulmonary infiltrates. The patient was transferred to our intensive care unit. On admission, temperature, heart rate, and arterial blood pressure were 37.7°C, 80 bpm, and 140/60 mm Hg, respectively. Heart sounds were physiologic and neck veins flat. Breathing was labored (30 breaths/min). Bilateral inspiratory rales were present dorsally and ventrally up to the apex of the lungs. No stridor was heard. Oxygen saturation was 100% (rebreathing mask; F_{IO_2} ≅ 80%).

The hematocrit and white blood cell count were 43% and 19,000 per microliter, respectively. Platelet count, prothrombin, and activated partial thromboplastin times were normal. The arterial blood gases (F_{IO_2} ≅ 80%) showed pH_a 7.36, Pa_{O_2} 190 mm Hg, Pa_{CO_2} 44 mm Hg, and HCO_3^- 24 mEq/L. The chest radiograph (Fig. 1A) showed diffuse bilateral pulmonary infiltrate, without enlargement of the heart or of the vascular pedicle. A bronchoscopy with bronchoalveolar lavage showed no mucosal abnormalities and minimal blood in the central airways. The bronchoalveolar lavage fluid (right middle lobe: 90 mL instilled, 60 mL recovered) was characteristic of alveolar hemorrhage, with an increasing amount of bloody fluid recovered as the instilled fluid was being suctioned and a large amount of red and white blood cells (3.48×10^6 per milliliter; polymorphonuclear leukocytes 92.5%, and 0% hemosiderophage). A diagnosis of postextubation pulmonary edema complicated by alveolar hemorrhage was made. The patient improved rapidly over a few hours and agreed to have a high-resolution chest CT the next day (Fig. 1B) to document the distribution of this unusual cause of the alveolar hemorrhage. Two days later, the chest radiograph showed a complete resolution of the pulmonary infiltrate.

Discussion

Many reports of pulmonary edema secondary to acute upper airway obstruction after tracheal extubation have been published (2,5,6). During episodes of upper airway obstruction, inspiratory efforts against a closed glottis (modified Mueller maneuver) may result in markedly negative pleural pressures (>-100 cm H_2O), which tend to increase left ventricular preload and afterload. In addition, altered pulmonary vascular resistance by hypoxia and increased adrenergic state may result in right ventricle dilation, interventricular septum shift to the left, and left ventricular diastolic dysfunction. These changes associated with increased left heart loading conditions (which both enhance microvascular intramural hydrostatic pressure), together with negative pleural pressure transmission to lung interstitium (7) (which decreases extramural hydrostatic pressure), may result in a marked increase in transmural pressure, fluid filtration into

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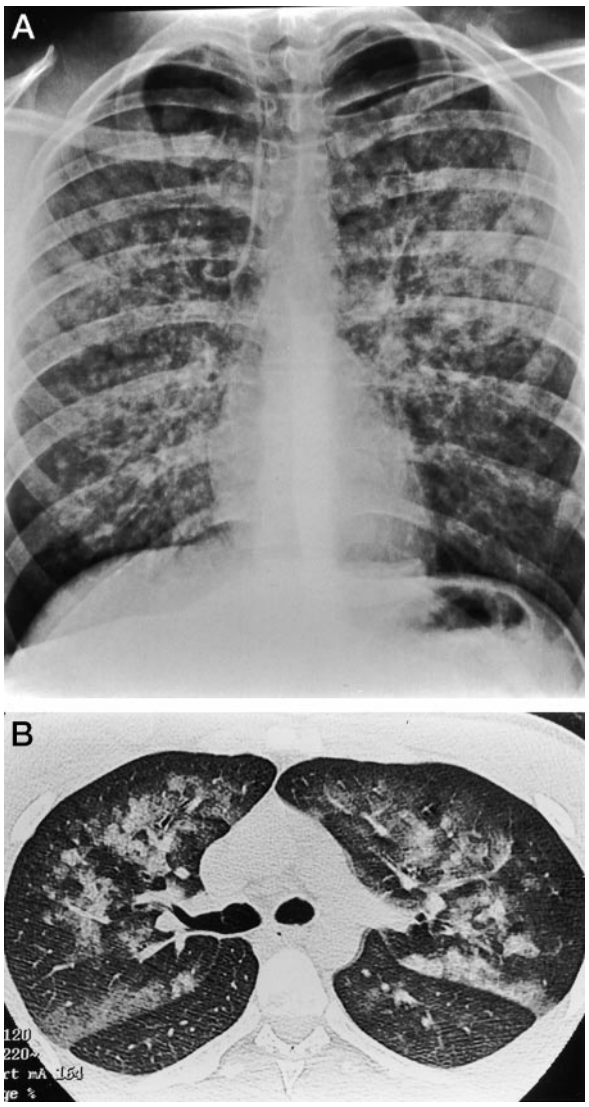

Figure 1. 1A, A 21-yr-old patient admitted for postextubation pulmonary edema. The admission erect chest radiograph displays a diffuse alveolar pattern of pulmonary edema that predominates in the middle lung zones. The lung cortex is relatively spared. No Kerley's line, peribronchial cuffing, or enlarged hilum can be observed. The mediastinal vascular pedicle and heart size are remarkably reduced. The chest film obtained 48 h after admission returned to normal. 1B, High-resolution computed tomography (CT) section obtained at the level of the carina (24 h after admission) displays bilateral and symmetric patchy areas of ground-glass attenuation, which predominates in the upper lobes and spares the lung cortex. No Kerley's line can be detected. Bronchial walls are not thickened, and pulmonary veins and arteries are sharply demonstrated. Pleural spaces are free of fluid. These CT findings are consistent with negative pressure pulmonary edema.

the lung (Starling equation), and the development of pulmonary edema (2,4) and capillary failure (8), as documented in animals (9).

Very few reports specifically addressed the issue of hemoptysis in the setting of negative pressure pulmonary edema (3,4,8). Alveolar hemorrhage, the hallmark of capillary failure (8), has been reported only once (10). In that report and in this one, alveolar hemorrhage was documented by bronchoscopy and bronchoalveolar lavage. This strongly suggests that large intrathoracic negative pressure swings may also cause capillary failure in humans (8). In another report, diffuse punctuate hemorrhages throughout the tracheobronchial tree were visualized, but no bronchoalveolar lavage was performed, suggesting that the systemic bronchial circulation may also be affected (4).

Although the radiographic findings associated with postextubation pulmonary edema have been reviewed (6), how postextubation edema distributes within the lungs has not been assessed by thoracic CT. Unlike with other forms of pulmonary edema, CT sections displayed a striking preferential central and nondependent distribution of ground-glass attenuation (edema/hemorrhage) that parallels the pleural and interstitial pressure gradients. Both pressures tend to be more negative in the central and nondependent regions than in the dependent and peripheral lung regions, respectively, and those regional pressure differences tend to increase with inflation and inspiratory effort (7). As a result, the interstitial and, therefore, perivascular pressures tend to decrease the most in the central and nondependent regions, and the transmural vascular pressure changes and capillary stress should be maximal in those regions. This could explain the striking distribution of lung edema and suggests that extramural pressure changes are instrumental in the development of pulmonary edema and capillary failure. If confirmed by other reports, this distribution of edema may be of diagnostic value. Usually, however, the diagnosis is not difficult, especially if rib retraction with poor air movement, laryngospasm, stridor, or all three are recognized.

In conclusion, negative pressure pulmonary edema should be recognized as one of the conditions that may manifest as alveolar hemorrhage likely caused by capillary failure (8).

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