Case Report

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Re-expansion Pulmonary Edema after Drainage of Pleural Effusion in a Pediatric Patient with a Large Anterior Mediastinal Mass

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Abstract

Clinical case presentation of a 13 year old male with a newly diagnosed anterior mediastinal mass who developed rapid respiratory distress after drainage of a pleural effusion. We include a discussion of the incidence, natural history, and peri-operative management of children with re-expansion pulmonary edema.

Keywords: Re-expansion pulmonary edema, Anterior Mediastinal Mass, Pleural Effusion, Pediatric Oncology, Pediatric Anesthesia.

Introduction

Re-expansion pulmonary edema (RPE) was first described as a complication of thoracentesis by Pinault in 1853 [1]. It is a rare phenomenon that is important to clinicians because of its rapid presentation and high morbidity and mortality. Here we present the case of a 13-year-old male who developed RPE after drainage of a pleural effusion.

A 13-year-old, 40 kg, male with a past medical history of attention deficit-hyperactivity disorder presented to his pediatrician's office with a history of ten days of cough and difficulty breathing. On physical exam, the patient was noted to have decreased breath sounds in the left upper lobe. He was diagnosed with presumptive pneumonia and prescribed a five-day course of azithromycin 250 mg daily. According to the patient's mother, his symptoms continued to worsen, and she began to notice an asymmetric fullness of the left chest. She returned one day later to his pediatrician who then requested a chest x-ray revealing complete opacification of the patient's left lung field accompanied by mediastinal shift. He was transferred to the Emergency Department where lung auscultation showed decreased left-sided breath sounds and anterior chest fullness with prominent veins but no lymphadenopathy. On review of systems, the patient endorsed an 11 kg unintentional weight loss, but denied chest pain, orthopnea, sleep disturbance, fevers, or night sweats.

A chest computed tomography (Figure 1) was obtained which revealed a large anterior mediastinal mass measuring up to 12.8 x 11.1 cm in anterior-posterior dimension and 16.2 cm in cranio-caudad axis. The mass caused rightward mediastinal shift and attenuation of the great vessels and left mainstem bronchus with nearly complete collapse of the left lung with minimal residual aeration of the left upper lobe apical segments and left lower lobe superior segments. He was also noted to have small pleural and pericardial effusions. The radiologist suspected a neoplasm, likely lymphoma and recommended a tissue biopsy be performed in interventional radiology for confirmation.

On the day of surgery, the patient was pre-medicated with midazolam 2 mg IV. General anesthesia was induced with ketamine 80 mg IV and was maintained with a propofol infusion at 300 mcg/kg/min. The patient also received additional IV fentanyl boluses for analgesia. Given the patient's preoperative clinical stability and tolerance of sleeping supine, the procedure was performed with a natural airway and avoidance of muscle relaxants. Supplemental oxygen was provided by a rebreathing facemask, and end-tidal capnography used to monitor ventilation. The patient remained hemodynamically stable throughout the procedure, and there were no intraoperative complications.

After the interventional radiologist performed anterior mediastinal mass biopsy under ultrasound guidance, a pigtail catheter was placed in the left pleural space and suctioned at a pressure of -120 cm H_20. After a total of 1.64L of straw-colored fluid was drained, the pigtail catheter was removed. There was immediate, visible improvement in chest wall excursion. However, the patient's oxygen saturation (SPO_2) decreased from 100% to 92% despite supplemental oxygen via facemask. At the conclusion of the procedure, the patient was transported to the post-anesthesia care unit (PACU). During his first hour in the PACU, the patient became agitated, tachypneic, and hypoxic. His SPO_2 decreased further to 82%. A chest x-ray was obtained (Figure 2). Given the size of his mass, the left lung field was difficult to visualize but there was no evidence of pneumothorax. The patient's respiratory support

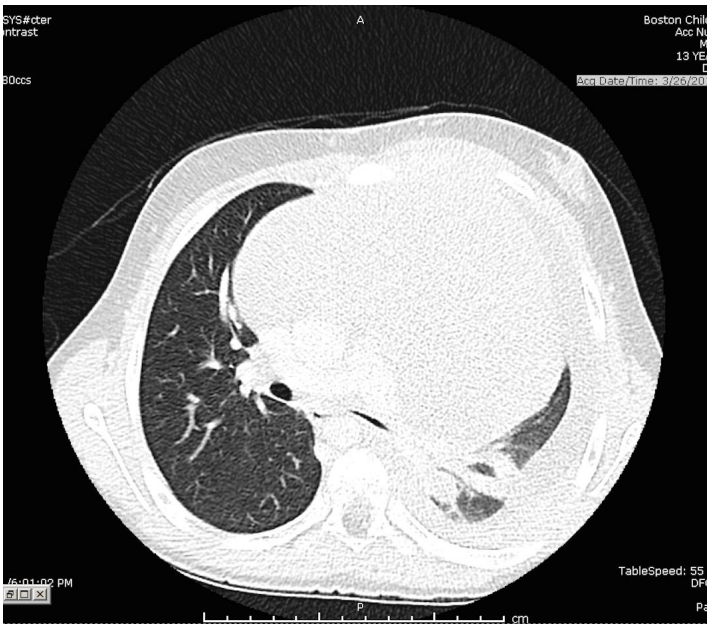

Figure 1: Chest CT; Transverse section of Chest CT showing large anterior mediastinal mass, rightward mediastinal shift, and compression of the left lung and left mainstem bronchus.

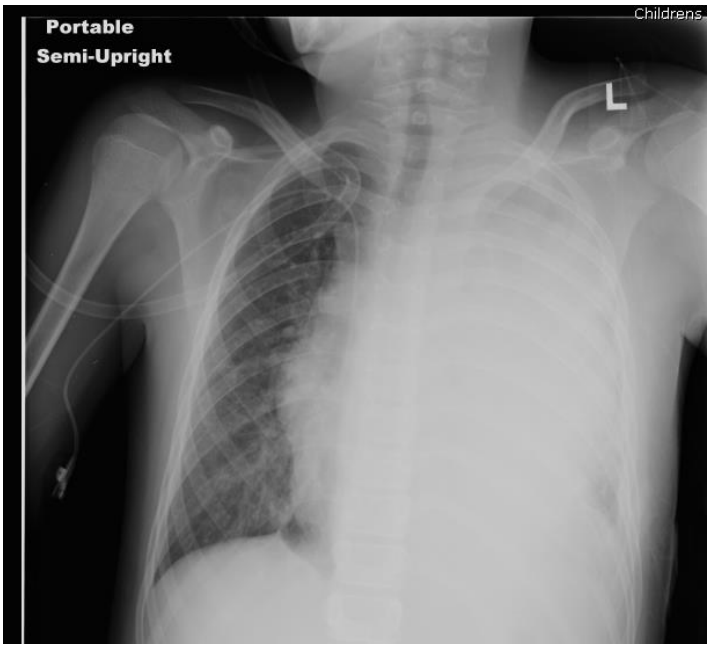

Figure 2: CXR; Portable AP chest xray taken immediately post-operatively.

was escalated to a non-rebreathing facemask, and he was admitted to the intensive care unit (ICU) for concern of re-expansion pulmonary edema.

In the ICU, the patient was placed on bi-level positive airway pressure (BiPAP) with pressures of 12/5 cm H_2O with dramatic improvement of his ventilation. He did not experience any hemodynamic instability or require the additional use of diuretics. His clinical status improved, and his respiratory support was weaned to nasal cannula on post-operative day (POD) 1 and to room air by POD 2. On POD 3, the patient was transferred from the ICU to the Oncology service where he began induction chemotherapy with a rapid and dramatic improvement in the size of his tumor.

Discussion

The anesthetic considerations of anterior mediastinal masses have been discussed extensively in the literature [2-4]. Briefly, the primary concerns are of catastrophic collapse of either the great vessels or the airway if there is a loss of chest wall tone or relaxation of bronchial smooth muscles. The goal then is to preserve spontaneous ventilation throughout the procedure and avoid muscle relaxation with depolarizing or non-depolarizing paralytics. In the event of airway collapse, the obstruction is often below the level of the carina and below the level of an optimally placed endotracheal tube. The anesthesiologist should be prepared to rescue ventilation through a rigid bronchoscope. In the event of circulatory collapse, the patient may have to be emergently placed on cardio-pulmonary bypass (CPB) or extra-corporeal membrane oxygenation (ECMO). Given these significant risks, this procedure is preferentially performed under local anesthesia infiltration with or without mild sedation.

Though many patients experience immediate improvement in oxygenation, ventilation, and ventilation/perfusion (V/Q) matching after drainage of a pleural effusion, considerable risk remains to the patient throughout the post-operative period. A rare but serious complication of pleural drainage or lung re-expansion is re-expansion pulmonary edema. Current understanding of RPE is limited and based primarily on case reports and anecdotal evidence. It is a rare disease with an incidence of 0.2% to 1% reported in most studies [5-10]. It is characterized by pulmonary edema after the re-expansion of a lung that has had prolonged collapse such as with pneumothorax, pleural effusion, surgical positioning, or mediastinal tumor. Initially, it was believed that RPE only occurred in lungs collapsed for at least three days; however, certain authors have suggested that it can occur with shorter time frames of minutes to hours [8-9]. RPE usually develops within one hour of drainage but can occur as late as 24 hours [10]. Its presentation can vary widely from purely radiographic evidence without symptoms to cough, tachypnea, pinkish sputum, hypoxia, or respiratory failure. Younger patients seem to be at greatest risk for developing it [5]. It is a serious complication with mortality reported as high as 20% [11].

The mechanism of RPE remains unknown. Importantly, it is not the result of cardiogenic sources or ventricular failure. Hypotheses that have been proposed include excessive negative pleural pressures [11-14], an acute inflammatory response, an increase in hydrostatic forces failed lymphatic drainage, and impaired pulmonary surfactant [13]. Animal models have shown an up-regulation of mRNA expression of pro-inflammatory cytokines leading to a SIRS-like response characterized by edema, neutrophil recruitment, and increased permeability of the pulmonary alveolar-capillary membrane [15,16]. Other authors have suggested hydrostatic forces caused by an increased trans-alveolar gradient

from rapid re-expansion or prolonged lateral decubitus positioning causes RPE [17].

The method used to expand the collapsed lung (e.g. needle aspiration, chest tube suctioning, hi-frequency jet ventilation, or re-inflation with positive pressure ventilation) does not seem to influence the development of RPE. It is still unclear whether the speed, level of suction, pleural pressure, or total volume drained influences the incidence of RPE [18]. Still many recommendations have been made about preventing RPE, but most are based on class C evidence (expert opinion). The British Thoracic Society suggests delaying the application of suction to chest tubes and using low-pressure, high-volume suction systems with an optimal suction of -10 to -20 cm H_20. It has also been suggested that thoracentesis of pleural effusions should be restricted to 1L at a time in adult patients [19]. By these standards, the thoracentesis performed by the radiologist in our case was clearly aggressive. Some authors feel that pleural pressures are the best guide for degree of suction and suggest that the procedure should be stopped if the pleural pressure drops below -20 cm H_20 [12]. However, Feller-Kopman et al. found no difference in opening or closing pressures and pleural pressures in patients who developed RPE and controls. He also points out that many patients have had greater than one litre removed without incident, and many patients require drainage of more than a litre for symptomatic relief. He concludes that "the development of RPE is not related to the volume of fluid removed, pleural pressures, and pleural elastance [20]. Other measures to prevent RPE that have been suggested are limiting crystalloid infusions and administering dexamethasone [21].

Given the variety of preventative measures and the lack of clinical data to support any of them, a high index of suspicion and initiation of prompt therapy may presently be the best defense against RPE. Post-operative patients should have vital signs monitored in an appropriate recovery settings with nurses trained to identify rapid onset dyspnea, increased work of breathing, crackles on auscultation, or a new oxygen requirement. Treatment is supportive and dictated by the clinical severity of the disease. Most cases can be treated with close observation and supplementary O_2. Lateral decubitus position on the affected side is recommended to reduce intrapulmonary shunting and improve V/Q matching [22]. If patients require ventilatory support, they should first receive non-invasive continuous or bi-level positive airway pressure via a facemask; however patients who meet the criteria for respiratory failure may require intubation. Diuretics and steroid use have not shown a clear benefit [23].

Since its initial description over 150 years ago, great progress has been made in characterizing RPE. However, it is still unclear which patient, disease, and procedural factors determine who will develop RPE. The patient presented here had a relatively slow growing tumor causing a prolonged period of collapse. His lung re-expansion was performed rapidly at high-wall suction. While pleural pressures were not measured, with a wall suction of -120 cm H_20, the pleural pressures were certainly less than -20 cm H_20 for the majority of the procedure. Additionally, he had a large volume (1.64 L) drained during the procedure. Since the occurrence of this case, our institution has adopted a policy of draining pleural effusions by hand aspiration, gravity, or low-wall suction. The total effusion drained is left to the discretion of the proceduralist, but practitioners are advised that close monitoring of pulmonary compliance, gas exchange, work of breathing is warranted intra- and post-operatively. While current literature supports conservative, "gentle" drainage of pleural effusion to prevent RPE, further studies are needed to elucidate the pathophysiologic mechanisms, risk factors, and best therapeutics. Thus far no guidelines have been published regarding the diagnosis and treatment of RPE, and the mainstay of treatment has been largely supportive.

References

1. Pinault HA (1853) Considérations cliniques sur la thoracentèse. These de Paris (in French).
2. Slinger P, Karsli C (2007) Management of the patient with a large anterior mediastinal mass: recurring myths. Curr Opin Anaesthesiol 20: 1-3.
3. Béchard P, Létourneau L, Lacasse Y, Côté D, Bussières JS (2004) Perioperative cardiorespiratory complications in adults with mediastinal mass: incidence and risk factors. Anesthesiology 100: 826-834.
4. Slinger P (2010) Anesthesia for Thoracic Surgery. Miller's Anesthesia (7th edn.) 1819-1888.
5. Trachiotis GD, Vricella LA, Aaron BL, Hix WR (1997) As originally published in 1988: Reexpansion pulmonary edema. Updated in 1997. Ann Thorac Surg 63: 1206-1207.
6. Matsuura Y, Nomimura T, Murakami H, Matsushima T, Kakehashi M, et al. (1991) Clinical analysis of reexpansion pulmonary edema. Chest 100: 1562-1566.
7. Jones PW, Moyers JP, Rogers JT, Rodriguez RM, Lee YC, et al. (2003) Ultrasound-guided thoracentesis: is it a safer method? Chest 123: 418-423.
8. Ravin CE, Dahmash NS (1980) Re-expansion pulmonary edema. Chest 77: 709-710.
9. Yanagidate F, Dohi S, Hamaya Y, Tsujito T (2001) Reexpansion pulmonary edema after thoracoscopic mediastinal tumor resection. Anesth Analg 92: 1416-1417.
10. Kira S (2014) Reexpansion pulmonary edema: review of pediatric cases. Paediatr Anaesth 24: 249-256.
11. Mahfood S, Hix WR, Aaron BL, Blaes P, Watson DC (1988) Reexpansion pulmonary edema. Ann Thorac Surg 45: 340-345.
12. Light RW, Jenkinson SG, Minh VD, George RB (1980) Observations on pleural fluid pressures as fluid is withdrawn during thoracentesis. Am Rev Respir Dis 121: 799-804.
13. Pavlin J, Cheney FW Jr (1979) Unilateral pulmonary edema in rabbits after reexpansion of collapsed lung. J Appl Physiol Respir Environ Exerc Physiol 46: 31-35.
14. Miller WC, Toon R, Palat H, Lacroix J (1973) Experimental pulmonary edema following re-expansion of pneumothorax. Am Rev Respir Dis 108: 654-656.
15. Suzuki S, Tanita T, Koike K, Fujimura S (1992) Evidence of acute inflammatory response in reexpansion pulmonary edema. Chest 101: 275-276.

16. Sprung CL, Loewenherz JW, Baier H, Hauser MJ (1981) Evidence for increased permeability in reexpansion pulmonary edema. Am J Med 71: 497-500.
17. Sue RD, Matthay MA, Ware LB (2004) Hydrostatic mechanisms may contribute to the pathogenesis of human re-expansion pulmonary edema. Intensive Care Med 30: 1921-1926.
18. Villena V, López-Encuentra A, Pozo F, De-Pablo A, Martín-Escribano P (2000) Measurement of pleural pressure during therapeutic thoracentesis. Am J Respir Crit Care Med 162: 1534-1538.
19. Mac Duff A, Arnold A, Harvey J (2010) Management of spontaneous pneumothorax: British Thoracic Society pleural disease guideline 2010. Thorax 65: ii18–ii31.
20. Feller-Kopman D1, Berkowitz D, Boiselle P, Ernst A (2007) Large-volume thoracentesis and the risk of reexpansion pulmonary edema. Ann Thorac Surg 84: 1656-1661.
21. Zhan X, Wang WD, Wang JJ (2007) Surgical treatment of mediastinal neoplasm in children. World J Pediatr 3: 1.
22. Dias OM, Teixeira LR, Vargas FS (2010) Reexpansion pulmonary edema after therapeutic thoracentesis. Clinics (Sao Paulo) 65: 1387-1389.
23. Echevarria C, Twomey D, Dunning J, Chanda B (2008) Does re-expansion pulmonary oedema exist? Interact Cardiovasc Thorac Surg 7: 485-489.