Reexpansion Pulmonary Edema in Pediatrics

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Abstract: Reexpansion pulmonary edema is a rare complication that may occur after drainage of pneumothorax or pleural effusion. A number of factors have been identified that increase the risk of developing reexpansion pulmonary edema, and pathophysiologic mechanisms have been postulated. Patients may present with radiographic findings alone or may have signs or symptoms that prompt evaluation and diagnosis. Clinical presentations range from mild cough to respiratory failure and hemodynamic compromise. Treatment strategies are supportive, and should be tailored to match the severity of the condition.

Key Words: pulmonary edema, pneumothorax, pleural effusion, thoracostomy, procedural complication

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TARGET AUDIENCE

This CME article is intended for pediatric emergency medicine physicians, emergency medicine physicians, pediatricians, nurse practitioners, nurses, physician assistants, and any other medical personnel involved in the care of children that may require procedural drainage of a pneumothorax or pleural effusion.

LEARNING OBJECTIVES

After completion of this CME article, readers should have improved their knowledge of and enhanced their competence to:

1. List risk factors associated with reexpansion pulmonary edema (REPE)
2. Recognize signs and symptoms of REPE when it occurs
3. Initiate appropriate preventative and management strategies that correlate with severity of presentation

CASE

A 12-year-old girl presents to the emergency department with 2 days of back pain and shortness of breath. She went to see the school nurse, who noted hypoxemia and decreased breath sounds on her left side. On arrival at the emergency department, she was anxious and complaining of mild back pain and shortness of breath. Her vital signs were the following: temperature, 36.8°C; heart rate, 123; respiratory rate, 22; blood pressure, 132/84; and oxygen saturation, 100% on room air. She was 5' 7" tall and weighed 48 kg. Her breaths sounds were significantly decreased on the left. The remainder of her examination was unremarkable. An initial radiograph showed a large left-sided pneumothorax (Fig. 1A). A 12F pigtail catheter was placed, with immediate coughing that quickly resolved. A postprocedure radiograph showed significant lung reexpansion with a small residual apical pneumothorax (Fig. 1B). Approximately 30 minutes later, she began coughing again and her oxygen saturation dropped to 92% on room air. Crackles were appreciated in her left lung fields. A repeat chest film showed interval opacification of the mid-left hemithorax consistent with pulmonary edema and possibly pleural effusion (Fig. 1C). The patient was placed on supplemental oxygen and admitted to the hospital. Serial radiographs during her hospitalization showed resolution of the pneumothorax and the radiographic opacities in the left lung fields, and she was discharged home.

Reexpansion pulmonary edema (REPE) is an uncommon complication after reinflation of a collapsed lung. It is most commonly described with lung reexpansion after treatment of pneumothorax or pleural effusion, although other etiologies (eg, mediastinal tumor resection[1]) have also been reported. Although uncommon, REPE is not a new discovery. A single case after drainage of a pleural effusion was published in 1853,[2] and a case series of 42 adult patients with "albuminous expectoration" after thoracenteses was published in 1905.[3] More than 50 years later, Carlson et al[4] reported the same complication after thoracostomy for pneumothorax. Reports of REPE in children were first published in the 1980s.[5,6] Since that time, cases have been described throughout the entire age range of pediatrics, with the youngest involving a preterm infant.[7] Only 1 pediatric case has been reported in the emergency medicine literature.[8] Given the paucity of publications describing REPE in pediatric practice, much of the current understanding described in this review reflects experience and data from the adult population.

The true incidence of REPE is unclear. Current rates are thought to be less than 1%,[9–11] although an incidence as high as 30% has been reported.[12] There are likely a number of reasons for such discrepant rates. Early case series likely reflected publication biases capturing higher than expected rates. More recent publications may also be difficult to generalize, given differences in inclusion criteria and study design. In addition, different definitions of REPE are used. Reports that include only symptomatic cases will result in lower rates than those that use a radiographic definition regardless of symptomatology. For example, the highest reported rate (~30%) comes from a study that prospectively performed computed tomography (CT) scans on all enrolled patients. Patients found to have these very sensitive CT-based findings were considered to have REPE; however only 2 of the 25 patients were clinically symptomatic.[13] There are no available data on rates of REPE specifically in children, with current publications limited to case reports and literature reviews. One study showed that *younger* patients (defined as < 40 years) are at greater risk of developing REPE.[14] However, given the minimum age for inclusion was 20 years, it is unclear how this translates to the pediatric population.

RISK FACTORS

A number of risk factors have been identified to help predict which patients are most likely to encounter REPE. The following

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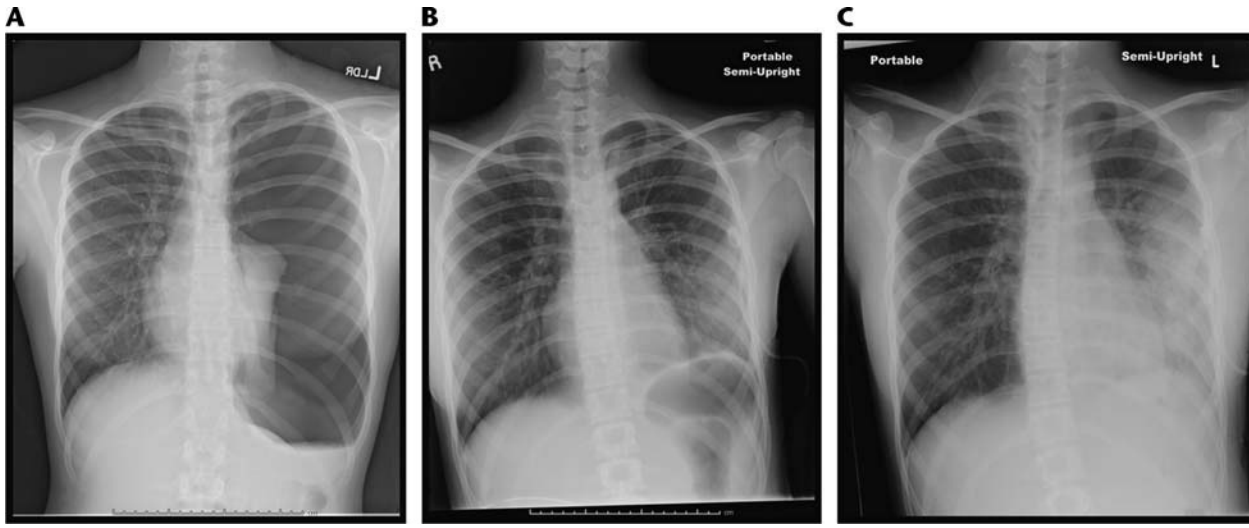

FIGURE 1. 1A, Radiograph of large left side pneumothorax. 1B, Postprocedure film with lung reexpansion after pigtail placement, with residual apical pneumothorax. 1C, Follow-up film with new left-sided reexpansion pulmonary edema.

variables have all been investigated as potential contributors: underlying etiology, age, rapidity of reinflation, use of (excessive) negative pressure, chronicity of lung collapse, size of the pneumothorax, and volume of fluid drained. Although these risk factors are cited throughout the literature, the supporting data are sometimes conflicting.

Underlying Etiologies

Lung collapse can occur from associated pneumothorax, pleural effusion, or atalectesis (which can be secondary to compressing mass, patient positioning, thoracic surgery, or single-lung ventilation).[15–18] Although reports of REPE have been described with each of these etiologies, reexpansion after treatment of spontaneous pneumothorax seems to carry the greatest risk of REPE.[15]

Age

Younger age is frequently cited as a risk factor for REPE. This stems from a retrospective study of 164 adult patients with spontaneous pneumothorax that found a higher incidence of REPE in the age group 20 to 39 years than in those older than 40 years. Interestingly, although the sample size was small (n = 23), the rate of REPE in patients aged 0 to 19 years was lower than that in the 20 to 39 age group.[14] Given the paucity of further data in children, it is not clear that "younger age" is also a risk factor in the pediatric population.

Use of Negative Pressure

Rapid lung reexpansion after application of negative pressure has been reported to increase the risk of REPE in both animal and clinical studies.[19,20] The most recent British Thoracic Society Guidelines on management of pneumothorax have suggested that suction should not be routinely applied. When used, recommended pressures should be between −10 and −20 cm H_20.[21] However, it is important to note that avoiding use of negative pressure is not necessarily protective, and cases of REPE have been reported in both children and adults after pleural drainage is connected to water seal alone.[8,22]

Chronicity of Lung Collapse

Many of the original descriptions of patients with REPE had symptoms suggesting chronically collapsed lung.[4] In an animal study, REPE only developed in the group with lung collapse for more than 3 days.[19] Clinical studies and reviews of the literature have supported this assertion, suggesting increased risk with longer durations of symptoms.[4,12,20,23–25] However, REPE has also been described with relatively short duration of symptoms, including less than 2 hours in a pediatric case report.[8]

Size of the Pneumothorax

When lung collapse is secondary to a pneumothorax, the volume of air in the pleural space has been associated with risk of REPE. Multiple studies have suggested that drainage of large pneumothoraces are more likely to be complicated by REPE.[12,23] One study defined pneumothorax size as small (<1/3 hemithorax), moderate (>1/3 hemithorax), and severe (complete lung collapse). The rate of REPE in these groups was 0%, 7%, and 17%, respectively, and occurred in nearly one half of cases described as tension pneumothorax.[14]

Amount of Fluid Drained

A similar principle has been described when lung collapse is secondary to pleural fluid rather than air. A number of citations report that removal of larger volume of fluid increases the risk of REPE.[10,14,25] Ault et al performed a prospective cohort study evaluating complications from nearly 10,000 thoracentesis in adults. They found an association with the volume of fluid removed and the likelihood of REPE; specifically, no cases of REPE when no fluid was removed, 4 cases (0.05%) when 1 to 1500 mL was removed, and 6 cases (0.75%) when more than 1500 mL was removed.[26] Using their data, they calculated that there was a 0.18% increased risk of REPE for every 1 ml of fluid removed.[26] Interestingly, 2 prospective studies in adults showed a very low rate of clinical REPE (0.2%–0.5%) with no association with volume of fluid removed or pleural pressures.[9,11] Importantly, both studies used development of symptoms as an indication for temporary cessation of fluid removal, which may be protective. The British Thoracic Society Guidelines suggest that no more than 1.5 L be drained initially, although with an acknowledgement that "there is no evidence for actual amounts" and no recommendations for the pediatric population.[27]

PATHOPHYSIOLOGY

The exact pathophysiology of REPE is unknown, although a number of mechanisms have been postulated. The etiology is likely multifactorial in nature. The leading hypotheses include the following: increased vascular permeability, changes in lymphatic flow, decreased surfactant, and changes in hydrostatic pressure in pulmonary vasculature during reexpansion.

Alveolar-capillary membrane disruption is thought to occur after ischemia reperfusion–mediated injury with reexpansion of the lung.[13,28,29] Compressed lung parenchyma becomes ischemic. Subsequent reexpansion results in reperfusion, which is believed to result in the production of reactive oxygen species, which may exacerbate endothelial damage.[9,16,30] The damaged capillaries subsequently become permeable to protein and fluid, resulting in fluid accumulation in the lungs. Animal studies have confirmed this increased permeability and identified key contributing inflammatory components in the process, including interleukin 8 (IL-8), monocyte chemoattractant protein 1 (MCP1), leukotriene B4, and xanthine oxidase.[31,32]

A number of other potential pathophysiologic mechanisms have been proposed as well. Reduction in lymph flow with decreased lymphatic clearance may also contribute to the generation of excessive fluid in the lung.[4,17,19,33,34] A reduction in surfactant may also contribute to the fluid accumulation during REPE. It is believed that surfactant is lost over time in collapsed alveoli.[4,34,35] Surfactant lowers alveolar surface tension, which counteracts fluid influx into the alveoli. Therefore, loss of surfactant in collapsed lung tissue more easily allows for fluid accumulation during reexpansion. Finally, higher perfusion in areas of high negative pressure or pulmonary venous constriction may lead to hydrostatic edema. Negative intrapleural pressure during reexpansion of the lung may result in vascular flooding. The resultant increase in pulmonary capillary pressures increases hydrostatic forces, which allows transudation of fluid into the alveoli. Animal models have supported this mechanism as well.[8,34,36]

CLINICAL PRESENTATION AND DIAGNOSIS

There is a great deal of variability in the presentation of REPE. Patients can be asymptomatic and diagnosed only on routine postprocedure plain film or CT when performed. Alternatively, a range of symptoms and signs have been described in both pediatric and adult patients. These most commonly occur immediately or within the first few hours postprocedure but may evolve even up to 24 hours later.[8,20,37,38] Reported symptoms include the following: tachypnea, rapidly progressive dyspnea or chest tightness/pain, and cough, which is sometimes productive of pink, frothy secretions. Given that coughing is described with lung expansion without edema, cough that is severe, persistent, or starts temporally remotely from the procedure may be more suggestive of REPE.[39] Nausea and vomiting have also been described.

Signs of REPE on physical examination include the following: new hypoxemia, which may result in cyanosis if profound. The degree of hypoxemia is likely influenced by the extent of ventilation/perfusion mismatching, volume of fluid in the airspaces, and reduction in lung compliance.[20] Crackles are commonly appreciated on the affected side. New serous or serosanguineous fluid may begin to drain from the chest tube. In profound cases, dramatic onset of respiratory failure and/or hemodynamic instability can occur. Hypotension may result from significant fluid shifts into the lung, or myocardial depression after the evacuation of the pneumothorax.[34]

Radiographically, REPE can involve part of the lung or the entire hemithorax. Descriptions of findings on radiographs include the following: pulmonary vascular congestion, alveolar and interstitial edema, and fluffy, patchy or diffuse infiltrates.[8,20,36,38] Such findings in a newly expanded lung can sometimes be difficult to distinguish from underlying pulmonary disease initially masked by lung collapse.[40] Although it is not routine to obtain a CT in these patients, reported findings in children and adults describe confluent alveolar opacities, often with a ground glass appearance consistent with pulmonary edema.[8,9,15,41]

Nearly all findings of REPE occur on the ipsilateral side as the initial lung collapse, although bilateral involvement has been reported.[17,20] One proposed pathophysiologic mechanism for contralateral involvement invokes compression atalectesis of the *unaffected* side secondary to mediastinal shift.[32]

MANAGEMENT

There are 2 strategies related to the management of lung reexpansion and REPE. The first is to proactively take appropriate measures to mitigate the risks of the development of REPE.[42] The second focuses on optimal strategies to support patients in whom REPE has occurred (Table 1).

Given the concern that REPE may be more likely to occur when the lung is rapidly reexpanded or when large volumes are evacuated, slower and limited evacuations may be prudent.[43] Using water seal drainage rather than negative pressure, or limiting the amount of pressure to less than −20 cm H_20 may be helpful.[21,44,45] Alternatively, intermittently clamping the tubing during drainage has also been suggested as a means to allow slower reexpansion.[46] Importantly, REPE has been identified even with the avoidance of suction; therefore, use of water seal by itself should not be expected to be completely protective.[12] Although there is some controversy around the efficacy, consensus opinion from the British Thoracic Society and the American College of Chest Physicians suggest limiting the removal of fluid or air to 1 to 1.5 L in adults.[21,47] No equivalent volumes have been proposed in children. Of note, studies have shown the safe removal of larger volumes of fluid in adults with no increased rate of REPE, when using patient symptomatology as a criteria for cessation of drainage.[9,39] Therefore, an alternative preventative strategy might be to

TABLE 1. Summary of Management Strategies for Reexpansion Pulmonary Edema

Clinical Presentation	Therapy
Radiographic-only REPE	Observation
Hypoxemia/mild increased respiratory effort	Supplemental oxygen
Moderate to severe increased work of breathing	Positive pressure with PEEP
Hypovolemia and hypotension	Volume repletion and inotropic support
Hypervolemia and volume overload	Diuretics (avoid overdiuresis)
Refractory respiratory failure or hypotension	ECMO

PEEP indicates positive-end expiratory pressure.

avoid rapid expansion as previously mentioned and prepare for pausing drainage when a patient begins coughing or becomes otherwise symptomatic. There have also been suggestions to use antioxidants and supplemental oxygen to prevent reactive oxygen species formation, although these strategies have not been well tested.[41]

Even with careful attention to currently recognized preventative strategies, REPE may still occur in both children and adults. Radiographic-only REPE may require only observation. Fortunately, REPE is typically a self-limited condition, and the majority of symptomatic cases can be managed with conservative supportive care alone. A number of therapies have been proposed, including supplemental oxygen, steroids, aggressive fluid resuscitation, and inotropic agents as needed.[13,48,49] The most common clinical presentation involves hypoxemia and/or increased respiratory effort. In these patients, supplemental oxygen should be provided. For more severe cases, positive pressure with PEEP may be required. Noninvasive positive pressure ventilation can be used when appropriate for institutional resources and provider experience.[50,51] Alternatively, when there is concern for impending respiratory failure or profound hypoxemia, endotracheal intubation and mechanical ventilation are considered the mainstay of therapy.[1] Positive pressure ventilation works by reexpanding collapsed alveoli, increasing functional residual capacity, and reducing shunting.[52] Differential lung ventilation has been proposed as an option to manage the differential pulmonary dynamics often found in REPE, although this technique is rarely required, with only 2 case reports in the pediatric literature.[16,53] Diuretics have been proposed,[13,48,49] although some authors caution against their use and believe they can contribute to deleterious hypovolemia.[8,41] When fluid shifts are large, hemodynamic compromise may require intravascular volume repletion and possible inotropic support. The successful use of extracorporeal membrane oxygenation (ECMO) as a salvage therapy has also been described.[53] Additional rescue therapies are currently under investigation, including "rapid pleural space reexpansion," which reintroduces drained pleural fluid back into the patient's pleural space; however, further studies need to be performed before recommending this as a potential therapy.[54]

OUTCOMES

Fortunately, the clinical outcome in patients with REPE is very favorable. Symptoms often progress for 1 to 2 days, but almost always fully resolve shortly after with no residual effects. Although 1 adult study reported a fatality rate of 20%, this may reflect publication bias of the most dramatic cases at the time.[20] Only 1 pediatric mortality after REPE has been reported and 1 other case requiring ECMO stabilization ultimately with a complete recovery.[53,55] However, with the literature limited to only case reports and series in pediatrics, it is difficult to know what the denominator of number of pleural procedures performed, to estimate morbidity or mortality figures. Fortunately, anecdotal experience and a paucity of reported cases suggest that such profound outcomes are rare in pediatric practice.

CONCLUSIONS

Reexpansion pulmonary edema is a rare complication after procedural reexpansion of the lung, most commonly after collapse from pneumothorax or pleural effusion. Although the incidence in pediatrics is low enough that published literature is limited to case reports and series, practitioners should be aware of this potential complication. Current recommendations are to avoid rapid and large volume reexpansion, although precise volumes are not known in pediatrics, nor are these strategies certain to be effective. Treatment is supportive and titrated to the patients' clinical presentation. Fortunately, the majority of patients do very well with no long-term sequelae.

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