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Introduction

Reexpansion pulmonary edema (REPE) is a rare, high-risk complication of lung reexpansion after prolonged compression. While there are reports in the adult literature and in adolescent patients, which suggest 20% mortality, there are very few cases reported in the preadolescent age group.

Case Description

EP is an otherwise healthy, 19-month old female who presented to her primary care physician with a chronic dry cough. Physical exam revealed minimal breath sounds over her left chest. Chest x-ray showed a completely opaque left hemithorax, with tracheal and cardiac deviation. Chest CT revealed a 10.4 x 8.6 x 7.4 cm predominantly fat containing mass occupying the majority of the left hemithorax. Following needle biopsy suggesting a benign giant lipoma, she was taken to the operating room for thoracotomy and tumor removal. One-lung ventilation was achieved utilizing intentional right-mainstem intubation. Anesthesia was maintained using propofol and remifentanyl infusion, with thoracic epidural using 0.2% ropivacaine infusion. The tumor was removed in its entirety. After gradual reexpansion of her left lung, the patient developed profound hypotension, increased peak pulmonary pressures, and pink froth in her endotracheal tube, consistent with symptoms of REPE syndrome. She was aggressively resuscitated with fluids (10cc/kg PRBC, 25cc/kg 5% albumin), and inotropic therapy (epinephrine 0.4 mcg/kg/min and norepinephrine 0.4mcg/kg/min). She was taken to ICU intubated, and weaned off of inotropic support within hours of surgery and extubated the following day. She was discharged to home on post-op day 4.

Discussion

REPE may be isolated to reperfusion edema with an increased fluid requirement, or it may take the form of a more severe, potentially lethal syndrome characterized by hypotension, low cardiac output^{1,2}. It has been reported following lung reexpansion for many reasons (pneumothorax, tumor removal, effusion drainage). Adult literature reports mortality rate of approximately 20%^{1,2}, many in otherwise healthy, younger adults, and despite massive fluid resuscitation and high-dose inotropic therapy. It is theorized that there are two components to the development of this syndrome: abnormal thickened pulmonary capillaries from chronic compression, and sheer stress to the capillaries upon rapid reexpansion¹. As a result of capillary damage, prostacyclin (PGI₂) and other inflammatory mediators are released², causing systemic vasodilatation. This can cause significantly decreased blood pressure and systemic venous return³. Treatment is based on the severity of disease and can range from fluid restriction and diuretics, positive pressure ventilation with positive end-expiratory pressure, to ECMO in rare extreme cases⁴. Treatment is the same in both children and adults⁴. Due to the rarity of the process and lack of reports in the pediatric literature, many practitioners are likely to be unaware of this potentially lethal complication from reexpansion of a chronically compressed lung.

1. Sohara, Y. *Ann Thorac Cardiovasc Surg* 2008. 14(4): 205-209

2. Sherman, S. *J of Emer Med* 2003. 24(1): 23-27

3. Mehta et al. *Am Heart Journal* 1981. 102(5): 835-840

4. Kira, S. *Paediatr Anaesth*. Online publication Nov 14, 2013
