The Heart's Halo: Caring for Pediatric Pneumopericardium

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J Pediatr Intensive Care 2018;7:213–215.

Abstract

Pediatric pneumomediastinum and pneumopericardium (PPC) are rare complications, which can arise from various etiologies. We report a case of pediatric PPC secondary to an asthma exacerbation and discuss relevant diagnostic and management principles. Physicians must be aware of PPC and its implications due to the high mortality rate. PPC patients require close observation with continuous cardiorespiratory and telemetry monitoring, and providers experienced in the management of cardiac tamponade at a center capable of providing cardiothoracic surgical intervention. Time to resolution is multifactorial, but can be achieved promptly with supportive care and treatment of underlying condition.

Keywords

- pneumopericardium
- pediatric
- critical care

Introduction

Pediatric pneumomediastinum (PM) and pneumopericardium (PPC), defined by the presence of air within the mediastinal connective tissue and the pericardial sac, respectively, are rare complications observed in cases of repeated elevation in intra-alveolar pressures.¹ Various etiologies have been identified, including blunt trauma, barotrauma, fistulas with air-filled structures, gas-producing bacterial infections, and iatrogenic causes.^{2–6} These air-leak syndromes have also been associated with lung pathologies prone to generate elevated intra-alveolar pressures, including asthma, cystic fibrosis, and interstitial diseases.^{7,8} PPC is less common, yet potentially more fatal in pediatrics due to the risk of cardiac tamponade (also known as tension PPC), requiring increased awareness, prompt recognition, and understanding of complications to guide management.⁹ We report a case of pediatric PPC secondary to asthma exacerbation and discuss relevant diagnostic and management principles.

Case Description

A 9-year-old previously healthy male without history of trauma presented to the emergency department with a

2-day history of cough, rhinorrhea, and tactile fevers. A chest X-ray (CXR) demonstrated peribronchial thickening without focal consolidations. He had symptomatic improvement after a breathing treatment with a β -adrenergic bronchodilator for presumed asthma exacerbation and was discharged home; however, he returned later that evening with worsening cough, increased work of breathing, and new-onset chest pain. Computed tomography of the chest revealed air in the mediastinum and cardiac sac, consistent with PM and PPC (→Fig. 1). The patient was transported to the pediatric intensive care unit (PICU), with close monitoring for signs of cardiac tamponade and consultation with cardiothoracic surgery.

On arrival, the patient was hemodynamically stable, requiring 6 L of oxygen per minute via simple face mask. A repeat CXR confirmed PPC and cervical subcutaneous emphysema. An electrocardiogram (ECG) was significant for sinus tachycardia. A respiratory viral panel was positive for *Rhinovirus*. His asthma exacerbation required standard medical therapy of bronchodilators and glucocorticoids. Otolaryngology (ear, nose, throat [ENT]) was consulted to assess for fistulous tracts as a source of PM and PPC. He was closely monitored for development of tamponade physiology with continuous telemetry and pulse oximetry. Serial CXRs were obtained, demonstrating resolution of PPC after

received

February 23, 2018

accepted after revision

April 15, 2018

published online

May 16, 2018

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Verlag KG, Stuttgart · New York

DOI <https://doi.org/10.1055/s-0038-1653981>.
ISSN 2146-4618.

Fig. 1 Sagittal (left) and axial (right) views of a noncontrast chest computed tomography study of a previously healthy 9-year-old male patient presenting with cough and chest pain. The patient was found to have pneumopericardium (black arrows) and pneumomediastinum (white arrows).

24 hours, and he was transferred out of the PICU (→**Fig. 2**). No further evaluation for fistulous tracts was completed as the patient's PPC was believed to be secondary to undiagnosed asthma. The patient was discharged home from hospital on day 3 in stable condition.

Discussion

In this case, a 9-year-old male was diagnosed with PM and PPC presumably caused by an asthma exacerbation triggered by *Rhinovirus* infection. One of the first pathophysiologic mechanisms leading to PM and PPC was described in 1939 and is now known as the “Macklin effect.”¹ This postulated

that repeated elevation in intra-alveolar pressure leads to rupture of alveolar walls, and subsequent air leakage into the pulmonary interstitium. From there, air traverses peribronchial and perivascular sheaths to the hilum of the lung, the mediastinum, and toward discontinuous pericardial connective tissue, resulting in air within the pericardial space. Pediatric PPC is rare and to our knowledge the incidence rate is not well described in the literature. However, the incidence of all air-leak syndromes (such as pulmonary interstitial emphysema, pneumothorax, PM, PPC) in newborns has been reported at 1 to 2%, with an incidence as high as 40% in mechanically ventilated infants.¹⁰ Historically, case series of pediatric PPC were reported with virtually universal

Fig. 2 Chest X-ray series of patient demonstrating resolution of pneumomediastinum (white arrows) and pneumopericardium (black arrows). The left image was obtained on day of admission. The follow-up image on the right was obtained on the second day of admission and demonstrates reabsorption of pneumomediastinum and pneumopericardium.

mortality,¹¹ but the severity of outcome has been reduced over the years with prompt diagnosis, risk reduction, and intervention when indicated. Still, physicians must be aware of PPC and its implications in the pediatric population due to persistently high mortality rates (57%).⁹

Patients with PM and PPC typically present with retrosternal chest pain (80–90%), dyspnea (50%), and occasionally a positive Hamman's sign: a crunching sound heard on auscultation, reflecting the air between the anterior parietal pericardium and the thoracic cavity.¹² Severe cases may also present with cyanosis, pulsus paradoxus, hemodynamic instability, and signs of cardiac tamponade, including hypotension, distant heart sounds, and elevated jugular venous pressure. Other physical exam findings may include a "Bruit de Moulin" murmur, a splash sound caused by air and fluid meeting in the pericardial space.¹³ ECG may reveal nonspecific, unreliable changes such as sinus tachycardia, low-voltage, ST-segment changes, and T-wave inversion.^{9,14} Echocardiogram findings in PPC may vary from cardiac cycle-associated spontaneous pericardial echoes seen as a "comet-tail" artifact¹⁵ to complete obscuration of the heart by air,¹⁶ thus limiting its usefulness in diagnosis. Consideration of bedside echocardiography is warranted with understanding of its limitations in this clinical context. PPC is often confirmed on imaging with the heart partially or completely encircled by an inner layer of air and outer layer of soft tissue representing the pericardial sac, known as the "halo sign."¹⁷ Once diagnosed, monitoring for PPC complications while awaiting air absorption is imperative. Cardiac tamponade, or tension PPC, is one of the most serious complications and occurs in 37% of the cases.¹⁸ Tension PPC requires emergent decompression, either percutaneously via pericardiocentesis or with open drainage.¹⁹

Management of PPC should include serial imaging with CXRs to demonstrate resolution by tissue reabsorption of the misplaced air. Telemetry and ECGs should be monitored for changes consistent with cardiac tamponade, including sinus tachycardia, pulsus alternans, and low QRS-voltage. If the source of PPC is unknown, further investigation is required to assess for fistulous tracts or other underlying disease with potential work-up including airway evaluation by ENT, Gastrografin swallow study, or esophagogastroduodenoscopy. Assessing for potential risk factors (history of trauma, recent operation, mechanical ventilation) or undiagnosed pulmonary disease (asthma, pulmonary hypoplasia, cystic fibrosis) should be completed to help risk stratify each patient.^{20,21} Patients with PPC require close observation and providers experienced in the management of cardiac tamponade at a center capable of providing pediatric cardiothoracic surgical intervention. Time to resolution is likely multifactorial, but can be achieved promptly with supportive care and treatment of the underlying condition.

Funding
None.

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