Case Report

Spontaneous tension pneumomediastinum in young child complicating bronchopneumonia successfully managed with high frequency ventilator

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ABSTRACT

Spontaneous pneumomediastinum means air in mediastinum is usually associated with a sudden formation of pressure gradient between the alveolus and surrounding tissue, resulting from either an over inflation of the alveoli or a reduction of interstitial pressure. Spontaneous mediastinum is predisposed to asthma, respiratory tract infections, or situations reproducing the valsala maneuver, uncommon in young children but potentially life threatening.

Keywords: High frequency ventilator, Spontaneous tension pneumomediastinum

INTRODUCTION

Spontaneous mediastinum following pneumonia is an uncommon entity in paediatric practice. Chalumeau reported prevalence of SPM between 1 in 800 and 1 in 42000, 70 % of cases of pneumomediastinum in children are due to bronchospasm or respiratory tract infection.3,9 We describe a case of spontaneous pneumomediastinum in child who is one year and five month old boy which developed during an episode of pneumonia without any history of bronchial asthma, trauma, or foreign body aspiration. He developed tension pneumomediastinum requiring placement of intercostal drainage tube with high frequency ventilator followed by conventional ventilator, successfully resolved after two months and fifteen days of Picu course without mediastinotomy.

CASE REPORT

One year and five months old male admitted because of fever for seven days, increase in breathing with inconsolable crying for seven days. He was well seven days before, above symptoms started by prodromal symptoms of common cold, fever which was high grade approaches to 39 C° continuous but relieved by paracetamol. Fever was not associated with rigors nor with skin rash. Next day of fever his breathing rate increased without cynosis or intercostals retraction. Mother brought the child in ER took ventolin nebulization and oral antibiotic in early phase of illness. Patient admitted on floor as a case of bronchophneumonia due to detoriation his condition. Patient has no previous admission or any episode of bronchospasm apart from at the age of two months got bronchiolitis treated as out-patient. He was born by LSCS with uneventful pregnancy with full term gestation, delivery was uncomplicated. His birth weight was 3 kg. He has one older male sibling 4 years old with h/o hyperactive airway disease, parents are without any chronic or allergic disease.

He has no allergy to food or drugs or skin allergy. He achieved growth mile stones at appropriate age. They are living with their grand-parents with no chronic illness.
No pets or smoking at home. He had contact with relative children who had viral infection. He is well grown child with height and weight on 50th percentile. He was febrile look sick, irritated easy to crying. No palor or cyanosis, he was tachypneic but hemodynamically stable. O₂ sat 88-91% at room air, bilateral equal chest air entry with expiratory wheeze and crepitations. No intercostal retractions, other systemic examination normal. His initial CBC and biochemistry and VBG were normal. We started treatment with antibiotic ceftriaxone and Azithromycin antibronchospasm nebulization with corticosteroid 1/v. He was little improved for two days, 3rd day he developed more respiratory distressed though his blood gas was acceptable. X-ray chest showed bronchopneumonia, patient was being shifted to pediatric ICU.

**Figure 1: X-ray during admission 1.**

**Figure 2: X-ray during admission 2**

**Figure 3: X-ray during CXR during PICU stay.**

**Figure 4: X-ray during 2nd week.**

**Figure 5: X-ray 3rd week.**
He was little improved on third day, on same day evening, developed sudden subcutaneous emphysema over neck with respiratory distress, need more O₂ to keep saturation >92 %. X-ray chest done no pneumothorax found. Venous blood gas showed pH 7.45 PCO₂ 29 PO₂ 47 HCO₃ 20, no improvement with increasing difficulty in breathing. Parents were informed regarding condition and intubated with ETT size 4.5 with conventional mechanical ventilator with FiO₂ 100 % Rate 25 PIP 20 PEEP 5. Blood gas was acceptable. Next few hours need to increase ventilator setting, X-ray chest revealed bilateral pneumothorax and pneumopericardium. Chest tube was placed bilaterally, no improvement were found in O₂ saturation which was 75 % to 80 % with FiO₂ 100%. Patient was supported with ionotropes and patient was fully sedated. Patient was diagnosed as air leak syndrome with ARDS. Persistent lower O₂ sat need to increase ventilator setting later on shifted to HFOV with map 26 amplitude 47 FiO₂ 100 % FR 10. His subcutaneous emphysema increasing in size approaching chest to face. Computed tomography scan chest confirmed pneumomediastinum. Patient was on ventilator for 2 months and 15 days. During this prolong course patient developed infection. Frequent Blood culture, urine culture and ETT tip secretion, wound swab C/S for chest tube. Central venous line, Blood c/s found Pseudomonas aerogenosa, coagulase-ve staph aureus and gram-ve bacilli at different time, treated with appropriate antibiotic sensitive to organism.

Patient was improved with resolving ARDS and air leak syndrome finally extubated successfully with supportive treatment and family support patient shifted to floor with sequel of prolong critical care. Daily physiotherapy and gradually weaned O₂, patient gained weight and finally discharge home with planned followup.

**DISCUSSION**

Spontaneous pneumomediastinum is rare and usually self-limited disease most commonly seen in young men and parturient women. However in young children SPM found mostly with asthma and lower respiratory tract infection.

The pathophysiology was demonstrated by Macklein in animal studies. An increased pressure gradient between the intralveolar and interstitial spaces enhances air leakage from small alveolar openings and ruptured alveoli in to the perivascular sheath yielding interstitial emphysema. The pressure gradient favour air direction along the vascular sheaths toward the hilum.

Pneumothorax may occur if the mediastinum pressure rises abruptly, air usually reach in to the neck as visceral layers of deep cervical fascia are contagious with mediastinum. Dissection of the free air in to the pericardial space is a common complication of barotraumas in neonates and into esophagus to the retro peritoneal tissue, abdominal wall and lower limb.

It may also escaped in to the subcutaneous or deep tissues of the neck and track down through the diaphragm around the aorta. The most common cause of Pneumomediastinum is alveolar rupture. Alveolar rupture occurs in the presence of elevated intra-alveolar pressure or damage to alveolar walls. Causes of elevated alveolar pressure include air way obstruction (e.g. by mucous plugging in an asthma person or by a foreign body), mechanical ventilation (particularly with a large ventilatory volume or high end expiration pressure), blunt trauma, coughing emesis or vasalva maneuver (e.g. during parturition). Cause of damage to alveolar walls include Pneumonitis, emphysema, lung fibrosis and acute respiratory distress syndrome. Other causes of Pneumomediastinum include trauma, gas producing infections in head, neck and abdomen and surgery in the upper GIT as well dental surgery.

Diagnoses of mediastinal emphysema is not difficult providing it is kept in mind if subcutaneous air in the neck and over the abdomen. The diagnoses is confirmed by a chest radiography illustrating multiple thin, lucent streaks outlining mediastinal structure elevating the mediastinal pleura and often extending in to the neck or chest wall. Lateral view of chest X-ray also give retrosternal air if PA view not illustrating, computed tomography is usually reserved where finding of plain radiography are inconclusive. Ultra sound has been used to diagnose pneumomediastinum as bed side procedure especially in critically ill patients. Majority of patients with SPM resolve spontaneously with prompt treatment of the underlying cause (pneumonia in our case) and by measure to reduce the intra alveolar pressure by oxygen, steroids and broncho dilator, rarely SPM may be extensive convert into tension pneumomediastinum interferes with the circulation and lung expansion would require intercostals chest drain, ventilator and may also require cervical mediastinotomy as lifesaving. We used high frequency ventilator in this case to avoid volume trauma and we treated recurrent infection promptly.
CONCLUSION

Spontaneous pneumomediastinum in children though known complication of pneumonia is not commonly seen. A high index of suspicion is required to diagnose the entity clinically. Chest radiograph and computed tomography confirmed the diagnoses. Ultrasonography can be used in critically ill patient.3,5

The outcome of pneumomediastinum varies from a benign course to a malignant and progressive one. It can be fatal if mediastinum pressure exceed with the circulation and lung expansion. Early recognition and prompt management with high frequency ventilator as in our case, inotropes, antibiotics, corticosteroid along with good supportive care help in resolution of pneumomediastinum. Role of high frequency ventilator in pneumomediastinum still limited need more clinical trials and evidence, we found it more helpful than conventional.

Patient need to minimize the activities associated with development of pneumomediastinum, medical conditions like asthma, chest infection, gastro esophageal reflux disease should be treated aggressively, children at risk of pneumomediastinum should be vaccinated fully including pertussis and influenza.2

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REFERENCES


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