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Pneumopericardium, pneumomediastinum, and pneumorrachis complicating acute respiratory syncytial virus bronchiolitis in children

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Abstract. – OBJECTIVE: We report 2 children with Respiratory Syncytial Virus (RSV) infection complicated with spontaneous pneumopericardium (PP) and pneumomediastinum (PM), one also associated with pneumorrhachis (PR).

PATIENTS AND METHODS: Two previously healthy children presented with fever, violent dry cough, dyspnea, and tachypnea. Chest X-ray and CT scans showed sizeable PP and PM in both patients. One of them also presented PR. Children were initially treated with intravenous antibiotics, antipyretics, and a cough sedative. Because of worsening of respiratory distress syndrome, children underwent helmet-delivered CPAP with oxygen supplementation. The patients' clinical conditions quickly improved and they were discharged in good conditions.

RESULTS: Pathogenetic mechanism of spontaneous PP and PM complicating RSV infection could be related to the cough, causing intrathoracic pressure increase and rupture of alveoli near the mediastinal pleura. Nevertheless, RSV seems to play a role in facilitating such complications, attenuating the cough threshold in infected children.

CONCLUSIONS: RSV bronchiolitis can lead respiratory and systemic consequences, so their prompt recognition is essential to establish a fast and adequate therapy, especially control of cough and respiratory distress syndrome treatment.

Key Words:
Respiratory syncytial virus, Bronchiolitis, Pneumopericardium, Pneumorrhachis, Pneumomediastinum.

Introduction

Respiratory syncytial virus (RSV) is the most common trigger of bronchiolitis and viral pneumonia constituting a clinical problem worldwide[1]. RSV infection is the second largest cause of mortality, after malaria, in infants outside the neonatal period and is the leading cause of hospitalization in infancy in developed countries[2]. It is estimated to cause 34 million of lower respiratory tract (LRT) infections, 3,4 million hospitalizations and up to 199,000 deaths per year in children younger than 5 years of age[1]. This constitutes a relevant clinical and social problem, representing a huge healthcare burden and associated cost.

It has been suggested that infants may be more liable to catch an RSV infection presenting with fever, violent cough, dyspnoea, and bilateral patchy pulmonary shadows at the chest X-ray[3].

Early age, prematurity, chronic lung disease of prematurity, congenital heart or lung disease, neuromuscular disorders, immunodeficiency, cystic fibrosis, Down's syndrome, represent risk factors for developing severe RSV disease, complications and hospitalization[4]. Unfortunately, also previously healthy children lacking any risk factor could experience severe RSV infection[1]. More common complications included pneumonia, bacterial super-infection and exacerbation of underlying medical conditions[5], more rarely pulmonary hemorrhage[6]. For those reasons, pediatricians should be aware of both risk factors of severity and complications which can occur in order to promptly start a proper respiratory and supportive treatment.

To our knowledge, no cases of children with RSV bronchiolitis presented with spontaneous pneumomediastinum (PM), pneumopericardium (PP) and pneumorrachis (PR) have been described. Here we report about 2 cases of spontaneous PP and PM, in one case also associated with PR, in 2 previously healthy children affected by RSV infection.

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Case Reports

Case 1

A previously healthy 3-year-old boy presented to our emergency room with one-day lasting fever and cough. His physical examination showed fever (38°C), dry cough, dyspnea, tachypnea and subcutaneous emphysema (SE) on the left side of his neck and upper chest. Chest auscultation revealed decreased breath sounds and moist crackling rales in the left lower lobe. Blood pressure (BP) was 130/80 mmHg; heart rate was 130 beats/min. Arterial blood gas analysis (AGA) showed pH 7.47, PO2 82.4 torr, PCO_2 29.6 torr; white blood cell count (WBC) revealed lymphopenia (lymphocyte count 580/ml); C-reactive protein (CRP) was increased (54 ng/ml, normal values < 5 ng/ml). Chest X-ray documented PM and widespread SE, suspected apical pneumothorax and bilateral increase of lung density. A subsequent chest CT scan showed a sizeable PM with SE, associated with PP and PR (Figure 1A). Since his clinical conditions were deteriorating and his dyspnoea was worsening (percutaneous oxygen saturation – SO_2 – 80% breathing room air), the child was referred to our Pediatric Intensive Care Unit (PICU) where he underwent helmet-delivered CPAP with oxygen supplementation (50% FiO_2). CPAP was delivered by the noninvasive ventilator (CF 800; Drager, Lubeck, Germany) set to 6 cmH_2O. The helmet (CaStar; Starmed, Mirandola, Italy) is a hood made of transparent, latex-free polyvinyl chloride. The patient was initially treated by intravenous Amoxicillin-Clavulanate and Clarithromicin, antipyretics, a cough sedative (cloperastine fendizoate) and intravenous fluids. The following day, the diagnosis of RSV infection was made by RT-PCR on a nasopharyngeal swab. After two days the patient's clinical conditions improved with progressive decrease of oxygen need and he was discharged from the PICU 4 days later. A control chest CT scan showed a partial resolution of both PM and PR after 5 days from the patient's admission (Figure 1B).

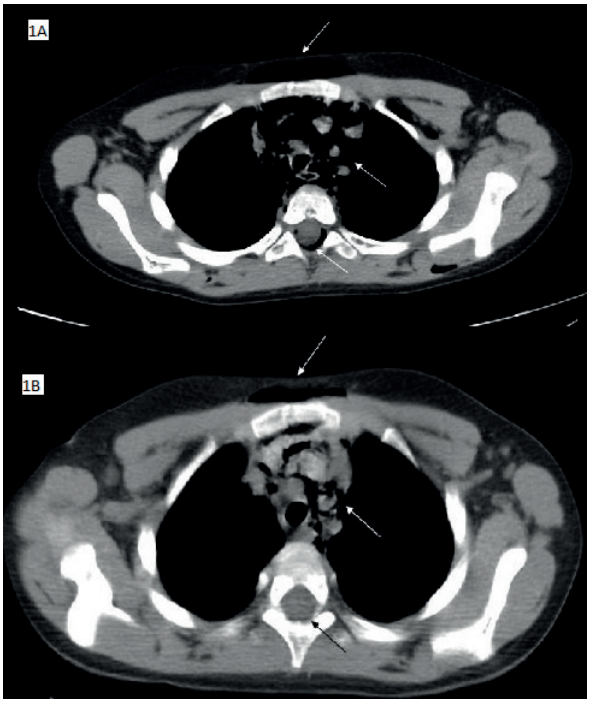

Figure 1. Chest axial CT images showing: ***A,*** pneumomediastinum, pneumopericardium, pneumorrachis and subcutaneous air (*see arrows*) ***B,*** partial resolution of pneumomediastinum, pneumopericardium and complete resolution of pneumorrachis after 5 days from patient's admission.

Case 2

A 2-year-old boy was admitted to our emergency room complaining of non-productive cough and dyspnea since 24 hours. On clinical examination, he presented dyspnea, tachypnea, fever and a violent cough. Chest auscultation revealed decreased breath sounds and crackles in the right lower lobe. BP was 81/49 mmHg, heart rate 144 beats/min. AGA showed pH 7.37, PO_2 76.1 torr, PCO_2 39.5 torr; WBC was increased (TLC 14350/ml) with high neutrophil count (11810/ml); CRP was increased (27 ng/ml). Chest radiography showed an air-space consolidation involving the right middle and lower lobes. In a few hours the patient's clinical conditions get worse, his SO_2 was 82%, he complained of chest pain, and his clinical examination revealed extensive SE involving his neck. A new chest X-ray confirmed the presence of SE and showed PM and PP (Figure 2A). For the worsening of his clinical conditions, he was referred to our PICU where started helmet-delivered CPAP (6 cmH_2O, FiO_2 50%). At the admission, the patient was initially given broad-spectrum antibiotics, a cough sedative (cloperastine fendizoate) and intravenous fluids. The day after, RSV was identified by RT-PCR in the nasopharyngeal swab. After two days he rapidly improved showing no more need for oxygen supply and then he was discharged from the PICU. New chest X-ray showed a partial resolution of both PM and PP after 3 days from his admission to the PICU (Figure 2B).

Discussion

Bronchiolitis is a worldwide respiratory infection caused by an inflammation of the small

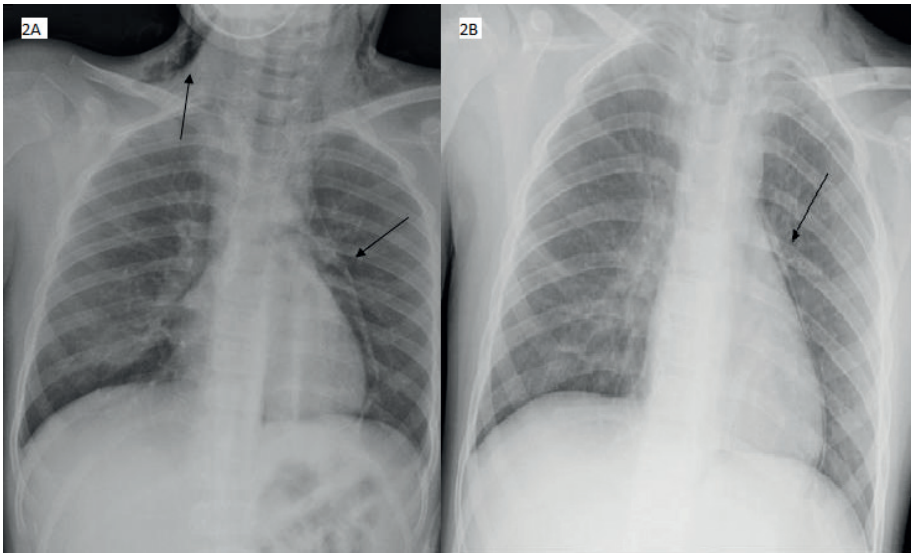

Figure 2. Chest X ray showing: ***A,*** pneumomedistinum, pneumopericardium and subcutaneous emphysema (*see arrows*) ***B,*** partial resolution of both pneumomediastinum and pneumopericardium after 3 days from patient's admission.

airways, the bronchioles. RSV first infects the upper respiratory tract, leading to mild coryzal symptoms. Within 1-3 days, RSV infection spreads to the LRT[2], infecting the ciliated epithelial cells of the mucosa of the bronchioles and pneumocytes in the alveoli[3] and causing cough, dyspnea, and cyanosis[2].

Severe LRT disease likely is the result of both uncontrolled virus infection, causing syncytial cell death and epithelial barrier breakdown, and tissue damage caused by a dysregulated immune response. A high viral load is associated with a high release of pro-inflammatory immune mediators such as cytokines, which are important for lung cell proliferation, activation, and differentiation, and chemokines, which orchestrate immune cells infiltration into the lungs, recruiting and activating leukocytes and inducing an excessive immune response[1,7]**.**

Cellular infiltration of the peribronchiolar tissue, edema, increased mucous secretion, sloughing of infected epithelial cells and impaired ciliary beating cause varying degrees of intraluminal obstruction. During inspiration, a negative intrapleural pressure is generated and air flow past the obstruction. The positive pressure of expiration further narrows the airways, producing greater obstruction, which causes wheezing[4]. Physical examination reveals prolonged expiration, crackles, wheezing; in severe disease, respiratory distress, oxygen desaturation, significantly reduced feeding and dehydration appear[2].

Children with underlying chronic diseases more likely develop complications. Nevertheless, also previously healthy children can face up to severe disease. Based on these data, it is essential to keep in mind that severe complications, such as cardiovascular and pulmonary disorders, should be excluded.

To our best knowledge, our study is the first report of spontaneous PP and PM associated with PR complicating the clinical course of RSV infection in healthy children.

The most common complication of RSV infection is pneumonia. Other reported complications are dehydration, encephalopathy, and exacerbation of underlying chronic disease[5].

Spontaneous PM and PR are uncommon entities *per se* indeed[8,9]. They often result from rupture of pulmonary alveoles bordering bronchioles or pulmonary vessels and might be triggered by asthma, respiratory infections and several circumstances involving a Valsalva maneuver, such as coughing or vomiting[9,10]. PR represents an exceptional imaging finding caused by various etiologies, mainly traumatic and iatrogenic[11]. Almost exceptionally, PR is found in combination with air distribution in other compartments and cavities of the body, particularly with pneumothorax[12], PM[13,14] or SE[11,15]. Also spontaneous PM following RSV infection is extremely rare, and there are only some reports in the literature. Only a few cases of PR associated with PM have been described, caused by violent coughing due to bronchial asthma or acute bron-

chitis[15], by trauma[13] or exceptionally in diabetic ketoacidosis[14]. Our cases suggest a possible higher prevalence of spontaneous PM in RSV-infected children which may reflect some pathologic findings in the respiratory airways.

It has been reported about a child with influenza virus bronchiolitis complicated by PM and SE[16] and also that influenza A virus infection attenuates the cough threshold of children, mainly in children with asthma. The enhanced cough response following influenza A virus infection is probably mediated by damage to the airways epithelium[17]. The association of these airways pathologic findings due to the action of RSV with a violent cough may explain the relatively high incidence of rupture of the respiratory tract in previously healthy children. On the other hand, even if spontaneous, PM and PR may recover by their own, patients must be strictly monitored and treated with oxygen supplementation, as in the present report, to avoid desaturation, dehydration and other severe complications.

Conclusions

Our cases of spontaneous PP, PM, and PR due to the RSV infection may represent a diagnostic and therapeutic challenge for pediatricians. Although these conditions usually are self-limiting and without further respiratory consequences, their prompt recognition in children affected by this infection is essential to establish fast and adequate therapy, mainly related to the control of the cough and the initiation of a proper respiratory distress syndrome treatment.

Conflict of interest

The authors declare no conflicts of interest.

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