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CASE REPORT

Spontaneous Pneumothorax as an Early Manifestation of Influenza A (H1N1) in a Pediatric Patient: A Case Report

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Abstract

Objective: To report on a case of a child with influenza subtype A H1N1 who exhibited a spontaneous pneumothorax as an initial manifestation of the illness. **Case Description:** A 8-year-old male patient experienced a dry cough in the 24 hours prior developed dyspnea, productive cough, tachypnea, tachycardia, and subcutaneous emphysema. Upon admission to the emergency ward, the patient was tachycardic with bilaterally palpable subcutaneous emphysema in the thoracic wall, was reliant on an oxygen mask, and experienced drops in arterial oxygen saturation. Chest and lung examinations revealed coarse crackles, expiratory wheezing, and suprasternal retraction. Patient received a corticosteroid, a bronchodilator, and an antibiotic. Imaging exams revealed bilateral pneumothoraces and bilateral subcutaneous emphysema. Patient was hospitalized in the Pediatric Intensive Care Unit (ICU). Bilateral chest drainage was installed; intubation and mechanical ventilation were initiated. On the sixth day after admission, the test for A/H1N1 came back positive. Patient was released from the Pediatric ICU on the ninth day and was released from the hospital the following day without symptoms. **Comments:** A pneumothorax as an early manifestation of A/H1N1 is a rare occurrence in children. This case reflects the need for broader differential diagnostic options in children with spontaneous pneumothoraces.

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INTRODUCTION

Influenza is a communicable and highly contagious acute viral infection of the respiratory tract that is distributed worldwide. The species *Influenza A virus* possesses a subtype known as H1N1. Patients with an H1N1 infection may quickly develop a lower respiratory infection, which can progress to acute respiratory distress syndrome (ARDS).[1] These clinical cases are characterized by fever with signs of upper airway impairment such as rhinorrhea, sore throat, dysphonia, and cough; these impairments are associated with at least one sign of systemic impairment involving malaise, chills, headache, and/or myalgia.[2] Among pediatric patients, the disease is frequently mild or oligosymptomatic and exhibits more self-limiting progression than in adults, though 1% to 8% of patients die, most frequently as a result of respiratory failure produced by airway immaturity or a lack of immunity.[3] Clinical differentiation between influenza A subtype H1N1 (A/H1N1) and other respiratory infections is hindered by the non-specificity of symptoms.[4] This difficulty in distinguishing between clinical symptoms affects reports on the true incidence of H1N1 cases and also delays both laboratory diagnosis of A/H1N1 and the type of care necessary.[5]

A pneumothorax is defined as the presence of air in the pleural space, which can lead to respiratory and hemodynamic conditions, and, in some cases, death. The occurrence of a spontaneous pneumothorax in childhood is uncommon (2.6 cases in 100,000), and it is known that pre-existing conditions such as lung infection and cystic fibrosis increase the risk of pneumothoraces.[6] Manifestations differ depending on the amount of lung tissue involved.[7] Resolution is spontaneous and occurs within a few days in cases with few symptoms. In cases of respiratory failure, chest drainage must be employed until the lungs are fully expanded once more. In persistent cases, surgical treatment is recommended.[8]

CASE REPORT

The guardians of an 8-year-old male living in an urban area in southeastern Brazil sought medical attention for his dry cough that had lasted for the 24 hours prior. They reported using ambroxol hydrochloride with no improvement. The first urgent care facility that the patient visited administered fenoterol hydrobromide and ipratropium bromide, as well as dexamethasone (the medical report from this facility did not list the dosages). The patient's guardians also reported their personal decision to administer oral doses of amoxicillin/clavulanic acid with no medical recommendation to do so. In the hours following his urgent care visit, the patient worsened clinically and developed dyspnea and productive cough (yellow mucus with blood). He was sent to a secondary public hospital where he was found to have tachypnea , a respiratory rate (RR) of 40 breaths per minute, tachycardia , a heart rate (HR) of 121 beats per minute, and subcutaneous emphysema. He received 1 g ceftriaxone and 400 mg clindamycin and was referred to a tertiary public hospital in northwestern São Paulo State.

Upon admission into the emergency department of the tertiary referral hospital, the patient was conscious, hydrated, afebrile, and exhibited a normal skin color; RR was 32 breaths per minute with a non-rebreather mask and oxygen saturation was 91%; he exhibited palpable subcutaneous emphysema in the face, neck, and chest. A respiratory examination revealed an audible vesicular breath sounds only in the lung base, as well as diffuse coarse crackles, symmetric expansibility, normal abdominal respiration with suprasternal retraction and no intercostal retractions. The patient also exhibited expiratory wheezing.

In the emergency department, the patient was received a loading dose of methylprednisolone, inhalation treatment with salbutamol, oxygen therapy using a non-rebreather mask, and 90 mg/kg/day of amoxicillin/clavulanic acid administered intravenously. A chest X-ray was performed (Figure 1) and revealed bilateral pneumothoraces, bilateral subcutaneous emphysema in the cervical region and in the left lateral thoracic wall, and diffuse opacities representing lesions in the air space. A chest CT (Figure 2) revealed lung parenchyma: bilateral pneumothoraces, the collapse of the lung parenchyma at the apices, ground-glass opacities in the inferior lobes (particularly on the right side) and with consolidation representative of infection. The CT also showed pneumomediastinum and diffuse subcutaneous emphysema in the thoracic wall. Because of these findings, the patient was hospitalized in the pediatric ICU three hours after admission into the emergency ward.

The patient's physical exam upon admission into the ICU revealed worsened dyspnea (RR 37 breaths per minute, nasal flaring, and subcostal and intercostal retraction without wheezing). An arterial-blood gas test (ABG) was

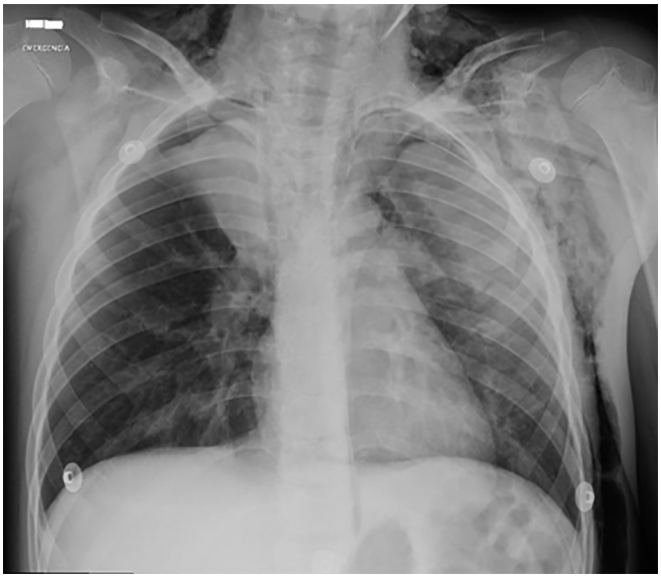


Figure 1. Chest X-Ray. Bilateral pneumothoraces, bilateral subcutaneous emphysema in the cervical region and in the left lateral thoracic wall, diffuse opacities representing lesions in the air space.

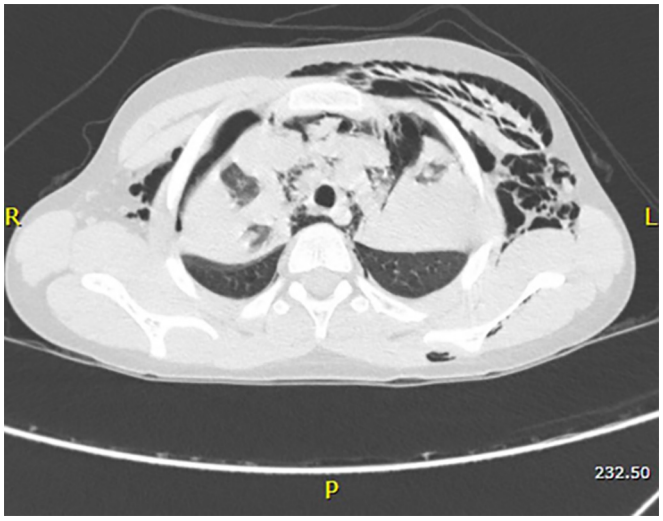


Figure 2. Chest CT. Lung parenchyma: bilateral pneumothoraces, collapse of the lung parenchyma at the apex, ground-glass opacities in the inferior lobes (particularly on the right side), and consolidation representative of infection. Mediastinum: pneumomediastinum. Thoracic wall: diffuse subcutaneous emphysema in the thoracic wall.

performed and found a pH of 7.36, PaO2 of 80.1 mmHg, pCO2 of 42.7 mmHg, a bicarbonate concentration of 23.7 mEq/L, a base excess of -1.72, 96% SaO2, and a lactate concentration of 1.2 mEq/L. The thoracic surgery team performed a bilateral chest drainage. A pigtail drain was installed on the right side and a conventional pediatric drain was installed on the left.

After the chest drainage procedure, an additional chest X-ray was performed. Though subcutaneous emphysema persisted, the pneumothoraces were found to have been resolved; atelectasis was present in the right apex. Amoxicillin/clavulanic acid was suspended and 100 mg/kg/day of ceftriaxone + 200 mg/kg/day of oxacillin was introduced. Food was suspended, and IV hydration was introduced. Over the next few hours, non-invasive mechanical ventilation was used; however, 12 hours after the chest drainage, intubation for mechanical ventilation was required. Approximately 12 hours after the start of mechanical ventilation via an endotracheal cannula, the ABG revealed respiratory acidosis: a pH of 7.17, a PaO2 of 106 mmHg, a pCO2 of 86 mmHg, a bicarbonate concentration of 30.9 mEq/L, a base excess of 0.19, 97% SaO2, and a lactate concentration < 1.0 mEq/L. Terbutaline, the β_2 adrenergic receptor agonist, was introduced at a dose of 0.2 mg/kg/min. Methylprednisolone was also introduced, and sedation was optimized with midazolam and fentanyl combined with cisatracurium besilate, a neuromuscular-blocking drug. Material was collected to test for the influenza virus, and the patient was also given both the antiviral medication oseltamivir at a dosage of 120 mg every 12 hours and the antibiotic clarithromycin at a dosage of 15 mg/kg/day. Over the six hours following these changes, the patient's condition improved and his ABG results began to normalize.

After four days on mechanical ventilation, the patient was extubated without complications. On the fifth day of hospitalization, all of the cultures were found to be negative, and both ceftriaxone and oxacillin were suspended. On the sixth day of hospitalization, the A/H1N1 results came back positive. The infectious disease team chose to maintain oseltamivir for 10 days at the same dosage. On the eighth day of hospitalization, both chest drains were removed without complications. Bilateral subcutaneous emphysema persisted, though without pneumothoraces. The patient was released from the pediatric ICU on the ninth day of hospitalization and was sent to the general pediatric inpatient ward. He was released from the hospital on the following day with no symptoms. Because he had been referred to our hospital from his hometown, the patient did not receive follow-up care with our team and we were unable to obtain follow-up data.

DISCUSSION

The H1N1 subtype of the influenza A virus of the family Orthomyxoviridae is known worldwide and has been the cause of pandemics.[9] It is crucial that the imaging results seen most commonly in A/H1N1 patients be identified in order to aid in both differential diagnoses and our understanding of disease progression, as well as to reveal possible complications, particularly in more severe cases.[10]

A study that reviewed chest X-rays of A/H1N1 patients found that 92.6% exhibited some type of lung abnormality, while only 7.4% had normal imaging results. The most common manifestations found were peribronchial cuffing, nodular opacities, ground-glass opacities, and reticulonodular opacities, all of which are likely to be bilateral.[10-13] There are few reports on spontaneous pneumothorax cases, since it is a very rare occurrence in cases of A/H1N1 infection unless the patient's infection is complicated by a respiratory distress syndrome requiring mechanical ventilation or by a concurrent lung disease.[14] The patient in this case report exhibited both the more common radiological findings (such as ground-glass opacity in the inferior lobes with consolidation on the right side) and the uncommon symptom of bilateral pneumothoraces with subcutaneous emphysema. In a review of the literature for the purposes of this publication, we found only one publication about spontaneous pneumothorax in a patient with A/H1N1, and it described a case of an adult woman.[9]

CONCLUSION

A pneumothorax as an early manifestation in children with influenza subtype A H1N1 (A/H1N1) is a rare occurrence. The physiopathology involved is not yet clear; the most common theory is that pneumothorax is simply a complication in disease progression or of the respiratory support mechanisms used, though these explanations are not useful in this case. This case reflects the need for broader differential diagnostic options in children with spontaneous pneumothoraces.

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