Case Report

# Severe Adenovirus Pneumonia Complicated by Subcutaneous Emphysema in A 3-Year-Old Child: A Case Report

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## Abstract

**Background:** Adenovirus is a common cause of respiratory infection in children and usually produces a self-limited illness. However, lower respiratory tract infection can occasionally progress to severe pneumonia with significant respiratory distress and complications. Subcutaneous emphysema is an uncommon complication during acute respiratory infection and may create additional diagnostic and management challenges. **Case presentation:** A 3-year-old girl presented with 2 days of high-grade fever and rhinitis, followed by cough and acute breathing difficulty. On admission she had tachypnea, subcostal retractions and nasal flaring. She was admitted to the pediatric intensive care unit because of respiratory distress and was treated with nebulised adrenaline, levalbuterol and budesonide along with systemic medications. On the first day of admission, respiratory distress worsened and high-flow nasal cannula support was started. During the hospital course, she developed subcutaneous emphysema and required closer respiratory and radiological monitoring. A nasopharyngeal respiratory syndromic panel detected adenovirus, while the other tested respiratory viruses and bacteria were not detected. Serial chest radiographs showed increasing subcutaneous emphysema during the period of respiratory distress. She gradually improved with supportive treatment and respiratory care; high-flow support was discontinued after four days, the emphysema resolved, and she was discharged clinically stable. **Conclusion:** Severe adenovirus pneumonia can progress rapidly in young children and may be associated with unusual air-leak manifestations such as subcutaneous emphysema. Early recognition of respiratory deterioration, appropriate respiratory support and serial clinical and radiological assessment are important for a favourable outcome.

**Keywords:** adenovirus, viral pneumonia, child, respiratory distress, subcutaneous emphysema, high-flow nasal cannula.

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## Introduction

Human adenoviruses are important respiratory pathogens in children and can cause a wide spectrum of illness ranging from uncomplicated upper respiratory infection to bronchiolitis and pneumonia. Young children are particularly affected, and adenovirus has been identified in a meaningful proportion of paediatric acute lower respiratory tract infections. In a large study from Beijing, adenovirus was detected in 5.8% of children evaluated for acute lower respiratory tract infection, with most infected children being younger than 5 years.[1]

Most adenovirus infections are self-limited, but severe pneumonia can occur and may be associated with prolonged respiratory symptoms, intensive care admission and long-term pulmonary sequelae.[2,3] Clinical presentation is often nonspecific, with fever, cough, rhinorrhea and respiratory distress being common features. Therefore, laboratory confirmation is useful when severe disease is suspected. Recent reviews also emphasise that treatment of severe adenovirus pneumonia is mainly supportive, with respiratory support being an important part of management because there is no routinely established specific antiviral therapy for otherwise healthy children.[4,5]

Air-leak complications such as pneumomediastinum and subcutaneous emphysema are uncommon in children with respiratory infections. They may occur when increased intrathoracic pressure associated with forceful coughing or severe airway disease leads to leakage of air from the alveolar or airway spaces.[6] Although subcutaneous emphysema has been described with several viral respiratory infections, its occurrence during severe adenovirus pneumonia is clinically noteworthy. We report the clinical course of a 3-year-old girl with laboratory-confirmed adenovirus pneumonia who developed subcutaneous emphysema during respiratory deterioration and subsequently recovered with supportive management.

## Case Presentation

**Patient information and presenting complaints:** A 3-year-old girl, weighing 11.6 kg, was brought to the hospital with fever for 2 days, rhinitis for 2 days, cough for 1 day and breathing difficulty beginning on the day of admission. The fever was described as high grade and was associated with rhinitis. The

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cough was predominantly present for one day. On the day of presentation, the child developed increasing breathing difficulty with chest retractions and noisy breathing. She had initially been taken to an outside hospital, where nebulisation was given, following which she was referred for further management.

There was no history of poor activity, vomiting or poor oral intake. There was no previous similar episode. Antenatal, natal and postnatal histories were uneventful. The child was immunised according to the national immunisation schedule and had achieved age-appropriate developmental milestones.

**Clinical deterioration:** During worsening respiratory distress on hospital day 1, the respiratory rate was reported as 60 breaths/min and $SpO_2$ was 90–92% on room air. Intercostal retractions, bilateral wheeze and crepitations were described at this stage. HFNC was initiated because of increased work of breathing and worsening oxygenation.

**Examination**

| Parameter | At admission | At the time of deterioration |
|---|---|---|
| Temperature | 100.2 °F | 99.2 °F |
| Pulse rate | 140/min | 150/min |
| Respiratory rate | 50 breaths/min | 60 breaths/min |
| $SpO_2$ | 95% on room air | 90-92% on room air |
| Capillary refill time | <2 seconds | <2 seconds |
| Weight | 11.6 kg | 11.6 kg |
| Respiratory examination | Subcostal retractions, nasal flaring and stridor/noisy breathing; no crepitations or wheeze | Subcostal retraction, intercostal retractions, bilateral wheeze and crepitations |
| Cardiovascular examination | S1 and S2 heard normally | S1 and S2 heard normally |
| Abdomen | Soft and non-tender | Soft and non tender |
| Neurological examination | GCS 15; no focal neurological deficit | GCS 15;no focal neurological deficit |

## Investigations

**Laboratory investigations:** The available outside record documents a C-reactive protein (CRP) value of 6.04 (reference interval of 2–6mg/dl). The complete blood count showed haemoglobin 11.5 g/dL, haematocrit 34.7%, total white blood cell count 11,440/µL and platelet count 3.56 lakh/µL. Differential count: neutrophils 85%, lymphocytes 12%, eosinophils 1%, monocytes 2% and basophils 0%.

The Respiratory Syndromic Panel (FilmArray – BIOFIRE), performed on a nasal swab, detected adenovirus. Coronavirus 229E, HKU1, NL63 and OC43, SARS-CoV-2, human metapneumovirus, rhinovirus/enterovirus, influenza A and B, parainfluenza viruses 1–4, respiratory syncytial virus, Bordetella pertussis, Bordetella parapertussis, Chlamydia pneumoniae and Mycoplasma pneumoniae were not detected.

The available biochemical report showed total bilirubin 0.4 mg/dL, direct bilirubin 0.3 mg/dL, indirect bilirubin 0.1 mg/dL, alkaline phosphatase 139 U/L, ALT 15 U/L, AST 45 U/L, total protein 7.0 g/dL, albumin 4.3 g/dL and globulin 2.7 g/dL. Renal/electrolyte results were: blood urea 24 mg/dL, creatinine 0.4 mg/dL, eGFR >90 mL/min/1.73 $m^2$, sodium 137 mmol/L, potassium 5.1 mmol/L and chloride 106 mmol/L. These results do not indicate a clear major renal or hepatic biochemical impairment in the available sample, although direct bilirubin and alkaline phosphatase were above the stated reference values.

Venous blood gas analysis at admission revealed a pH of 7.43, $pCO_2$ of 35 mmHg, bicarbonate of 23.2 mmol/L, and lactate of 1.1 mmol/L, indicating no significant acid–base disturbance. Following clinical deterioration and initiation of high-flow nasal cannula (HFNC) support, repeat VBG showed a pH of 7.41, $pCO_2$ of 34 mmHg, bicarbonate of 21.6 mmol/L, and lactate of 0.8 mmol/L. The serial blood gas analyses demonstrated preserved pH and lactate levels without significant hypercapnia or lactic acidosis.

**Radiological findings:** Serial chest radiographs were obtained during admission. Figure 1A shows the admission chest radiograph, before the documented major deterioration. Figure 1B shows the radiograph obtained during respiratory distress, when subcutaneous emphysema was identified. Figure 1C shows the repeat radiograph documenting increased subcutaneous emphysema during the period of distress. Figure 1D shows the final radiograph before discharge, with substantial improvement.

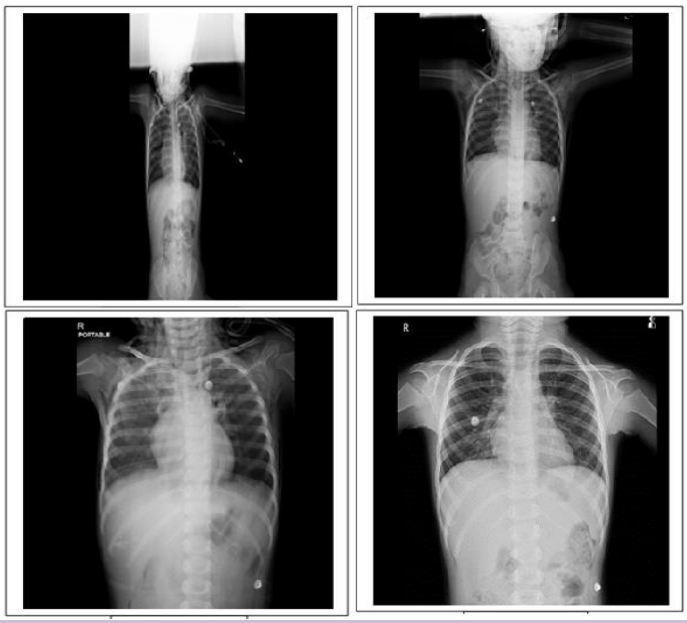


**Figure 1A: admission chest radiograph on day 1 of admission; Figure 1B, radiograph obtained during increased respiratory distress on day 1 of admission; Figure 1C, repeat radiograph showing increased subcutaneous emphysema on day 2 of admission; Figure 1D, final pre-discharge radiograph showing interval improvement on day of discharge.**

**Treatment and clinical course:** Because of respiratory distress with subcostal retractions and stridor/noisy breathing, the child was admitted to the PICU. Initial treatment included nebulised adrenaline, levalbuterol and budesonide, dexamethasone and supportive medications. Paracetamol, pantoprazole, montelukast with levocetirizine and oral azithromycin were also documented. On hospital day 1, worsening respiratory distress and increased work of breathing, with respiratory rate reported at 50–60 breaths/min and $SpO_2$ 90–92% on room air, prompted initiation of high-flow nasal cannula (HFNC). The inspired oxygen requirement subsequently increased from 21% to 30%, and nebulisation frequency was increased.

Subcutaneous emphysema was first noted approximately 6–8 hours after HFNC initiation. While high positive distending pressure from HFNC can theoretically contribute to air leak dynamics, the temporal onset closely coincided with severe coughing and forceful respiratory distress, pointing to the underlying severe small airway inflammation and high work of breathing as the primary drivers. Serial chest radiographs showed the appearance and subsequent increase of subcutaneous emphysema during the period of respiratory distress. Pulmonology consultation was obtained. Intravenous ceftriaxone was given for the documented lower respiratory tract infection/severe respiratory illness, alongside antipyretics, methylprednisolone and continued nebulisation. Intravenous magnesium sulfate was documented as a second line adjunctive bronchodilator; an allergic reaction to it was also recorded.

The child remained on high-flow nasal cannula support and medical treatment for approximately four days. Her respiratory distress gradually settled, after which high-flow support was weaned and stopped. She was transferred out of the PICU, nebulisation was continued with decreasing frequency, and intravenous antibiotics were completed for seven days. The subcutaneous emphysema resolved clinically and radiologically. The child became active and alert, tolerated oral feeds well and maintained stable vital signs.

At discharge, she was prescribed a short course of cefpodoxime, montelukast/levocetirizine, vitamin supplementation and vitamin D. Influenza vaccination was advised. In the event of recurrent respiratory infection, HRCT, genetic evaluation and immunodeficiency work-up were advised.

## Discussion

This case highlights the potentially severe respiratory course of adenovirus infection in a young child. The child initially had a short history of fever, rhinitis and cough, but respiratory distress developed rapidly. The diagnosis was supported by a multiplex respiratory panel that detected adenovirus and did not detect the other respiratory pathogens included in the panel. Adenovirus is well recognised as a cause of paediatric respiratory infection, particularly among younger children, and can occasionally produce severe lower respiratory tract disease.[1,2]

The clinical deterioration in this child is consistent with the spectrum described in severe adenovirus pneumonia. Severe cases may present with marked respiratory distress and can require intensive respiratory support. Reviews of pediatric adenovirus pneumonia emphasize the importance of early recognition of worsening respiratory effort and timely escalation of supportive respiratory care.[4] The present child required PICU care and high-flow nasal cannula support, but did not require invasive mechanical ventilation.

An important feature of this case was the development of subcutaneous emphysema during the period of respiratory deterioration. Subcutaneous emphysema represents the presence of air within the subcutaneous tissues and may occur in association with pneumomediastinum or other air-leak syndromes. In children, viral respiratory infections and vigorous coughing have been reported as triggers for pneumomediastinum and subcutaneous emphysema.[6,7] The mechanism is thought to involve increased intrathoracic pressure and alveolar rupture, with air dissecting along tissue planes. In the present case, the available radiographs clearly documented increasing subcutaneous emphysema, but pneumomediastinum was not explicitly reported in the source record; therefore, it would be inappropriate to label the case as pneumomediastinum without further imaging confirmation.

The patient's course also illustrates the role of serial assessment. The initial radiograph was followed by repeat imaging when respiratory distress worsened, which demonstrated progression of the subcutaneous emphysema. Later imaging documented improvement. This temporal relationship, together with clinical improvement following respiratory support and medical treatment, supports careful observation and treatment of the underlying respiratory illness when the child remains clinically stable. Published paediatric reports similarly describe conservative management and close monitoring for viral-associated air-leak syndromes in clinically stable children.[6,7]

Another clinically relevant point is the absence of documented previous respiratory disease. The child had no history of similar episodes, had normal development and was immunized according to schedule. However, an immunodeficiency work-up was advised only if further respiratory infections occurred, so the present record does not establish a formal immunological evaluation. Likewise, the adenovirus serotype was not determined. These limitations should be acknowledged because both host factors and viral type can influence the severity and complications of adenovirus infection.[3,5]

The child improved without invasive ventilation and was discharged in stable condition. Nevertheless, severe adenovirus pneumonia can be followed by persistent respiratory complications in some children, including post-infectious bronchiolitis obliterans and other chronic airway abnormalities.[3,4] For this reason, appropriate follow-up is important, particularly if recurrent or persistent respiratory symptoms develop.

**Limitations:** HRCT was advised by the pulmonology team but was not documented as completed during this admission. Consequently, the precise anatomical extent of the air leak could not be further characterised by CT imaging.

## Conclusion

Severe adenovirus pneumonia may present with rapid respiratory deterioration in a young child and can be accompanied by unusual complications such as subcutaneous emphysema. In this case, early PICU admission, high-flow nasal cannula support, appropriate medical treatment and serial radiological assessment were followed by complete clinical improvement. The case emphasises the need for clinicians to remain alert to air-leak complications during severe viral respiratory illness and to individualise follow-up according to the subsequent respiratory course.


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## Conflicts of interest

There are no conflicts of interest.

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