Case Series

Not All Infantile Air Leaks Tell the Same Story: A Case Series from South India

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ABSTRACT

Spontaneous pneumothorax (SP) in infancy is uncommon and often secondary to underlying infectious, congenital, or neoplastic lung pathology. Clinical presentation may be subtle, and pediatric management strategies are largely extrapolated from adult guidelines. In this case series, five infants aged 2–8 months with spontaneous pneumothorax are presented. Etiologies included viral lower respiratory tract infection in three infants, congenital bullous disease in one, and pleuropulmonary blastoma in the last infant. Two infants were managed successfully with intercostal chest drainage. Three of them required surgical intervention due to persistent air leak or structural lung abnormality, including bullectomy and lobectomy. One infant was diagnosed with Type I pleuropulmonary blastoma on histopathology. From these cases, it can be inferred that infantile spontaneous pneumothorax represents a heterogeneous clinical entity. Persistent or recurrent air leak warrants advanced imaging and early surgical evaluation to identify underlying structural or neoplastic causes.

Key words: Pneumothorax, Infant, Thoracoscopy, Pleuropulmonary Blastoma

Spontaneous pneumothorax (SP) is defined as the presence of air within the pleural space in the absence of trauma or invasive procedures [1]. It is categorized as primary spontaneous pneumothorax (PSP), occurring without underlying lung disease, and secondary spontaneous pneumothorax (SSP), associated with identifiable pulmonary pathology [2]. While PSP predominates in adolescents, SP in infancy is rare and more frequently secondary in nature [3]. The estimated annual incidence of pediatric spontaneous pneumothorax is approximately 4 per 100,000 males and 1.1 per 100,000 females, with significantly lower rates in infancy [4]. Clinical presentation in infants may be nonspecific, including tachypnea, hypoxemia, feeding difficulty, and decreased breath sounds [5].

Reported etiologies include viral lower respiratory tract infections, necrotizing pneumonia, congenital pulmonary airway malformation (CPAM), congenital lobar emphysema, congenital bullous disease, and, rarely, pleuropulmonary blastoma (PPB) [6–8]. Pathophysiologically, increased intrathoracic pressure from coughing or crying, combined with structurally vulnerable lung parenchyma, may lead to alveolar rupture and air leak [9]. Diagnosis is primarily radiographic, with chest radiography serving as the first-line modality. Computed tomography (CT) aids in identifying structural abnormalities and guiding surgical planning [3,6]. Pediatric-specific management guidelines remain limited, and treatment decisions are often individualized [10]. Five infants with spontaneous pneumothorax, illustrating diverse etiologies and management strategies are presented here.

CASE PRESENTATION

Case 1

An 8-month-old female, born at term with no antenatal or perinatal complications, presented with sudden respiratory distress and feeding difficulty. There was no history of trauma, previous lung disease, or family history of pulmonary disorders.

On examination, she was tachypneic and hypoxic with absent breath sounds over the right hemithorax. Chest radiograph revealed a large right-sided pneumothorax with mediastinal shift. Emergency needle decompression followed by intercostal chest drainage was performed. Persistent air leak continued beyond 72 hours despite appropriate drainage.

CT thorax demonstrated multiple cystic lesions in the right middle and lower lobes (Figure 1). Thoracoscopic exploration

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revealed a large bulla with smaller adjacent bullae. Bullectomy was performed using Video-Assisted Thoracoscopic Surgery (VATS). Histopathology confirmed Type I pleuropulmonary blastoma (PPB). Postoperative recovery was uneventful. During the 6-month follow-up, the infant remained asymptomatic without recurrence.

Figure 1. CT image for Case 1.

Case 2

A 2-month-old male, born at term and previously healthy, presented with cough, wheeze, and increased work of breathing for four days. Polymerase Chain Reaction (PCR) confirmed Respiratory Syncytial Virus (RSV) infection. The initial radiograph showed bilateral infiltrates (Figure 2).

On day three of hospitalization, worsening respiratory distress prompted repeat imaging, which revealed a left lower lobe pneumatocele with loculated pneumothorax. Intercostal chest drainage was performed. The infant improved clinically and radiologically within five days and was discharged without recurrence.

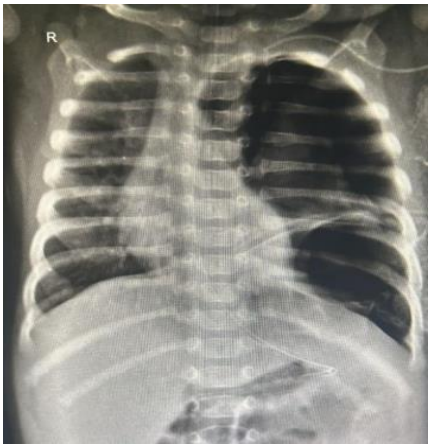

Figure 2. Radiographic image for Case 2.

Case 3

A 7-month-old female presented with fever and acute respiratory distress. PCR confirmed influenza A infection. Arterial blood gas showed respiratory acidosis. Chest radiograph demonstrated a moderate right-sided pneumothorax.

Intercostal drainage resulted in rapid clinical improvement. CT imaging showed no structural abnormality. The pneumothorax resolved completely, and follow-up was uneventful.

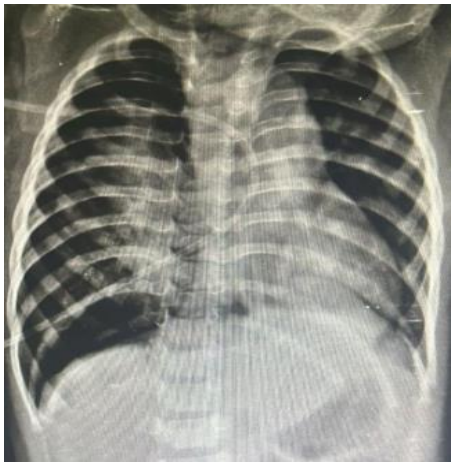

Figure 3. Radiographic image for Case 3.

Case 4

A 6-month-old female presented with acute respiratory distress. She was born in term with normal antenatal scans. Examination revealed decreased breath sounds on the right side. Radiograph confirmed right pneumothorax.

CT thorax suggested congenital bullous disease. Thoracoscopy identified a large, isolated bulla without bronchial communication. Bullectomy was performed by Video-Assisted Thoracoscopic Surgery (VATS). Histopathology showed normal lung tissue. The child recovered well.

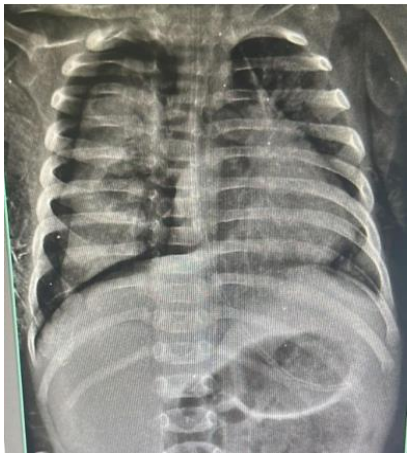

Figure 4. Radiographic image for Case 4.

Case 5

A 2-month-old male with a history of high-grade fever and Staphylococcus aureus sepsis was referred for persistent respiratory distress. Laboratory parameters showed elevated

inflammatory markers. Imaging revealed a left-sided cystic lesion.

Thoracotomy demonstrated necrotizing pneumonia with loculated pneumothorax. Left lower lobectomy was performed. The child completed antibiotic therapy and recovered without recurrence.

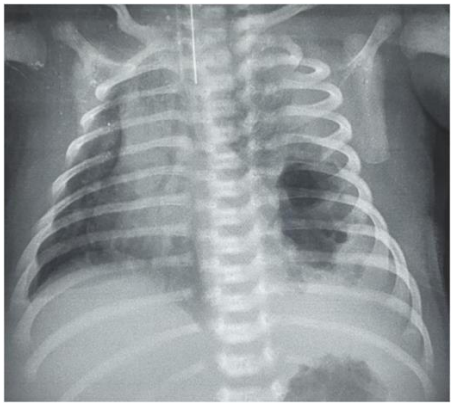

Figure 5. Radiographic image for Case 5.

Table 1 compares the main features of the cases presented.

Parameter	Case 1	Case 2	Case 3	Case 4	Case 5
Age (months)	8	2	7	6	2
Etiology	Pleuropulmonary blastoma	RSV infection	Influenza A infection	Congenital bullous disease	Necrotizing pneumonia
Side	Right	Left	Right	Right	Left
Management	Bullectomy (VATS)	Intercostal chest drain	Intercostal chest drain	Bullectomy (VATS)	Left lower lobectomy
Persistent air leak	Yes	No	No	Yes	Yes
Outcome	No recurrence at follow-up	Recovered; no recurrence	Recovered; no recurrence	Recovered; no recurrence	Recovered; no recurrence

DISCUSSION

This case series highlights the heterogeneity of infantile spontaneous pneumothorax. Three of five cases were infection-related, supporting existing literature identifying viral and bacterial infections as common precipitants in infancy [7, 9]. Increased intrathoracic pressure combined with inflammatory parenchymal injury may predispose to air leak. Persistent air leak in Case 1 led to the diagnosis of PPB, a rare but important differential diagnosis [8]. Similar cases have been reported in which PPB initially presented as a tension pneumothorax [8]. Early surgical intervention facilitated definitive diagnosis.

Congenital bullous disease and necrotizing pneumonia, as observed in our series, are recognized structural causes requiring operative management [6,11]. Recurrence rates of pediatric SP have been reported up to 30% when underlying lesions are present [4,12]. Minimally invasive techniques such as VATS offer improved recovery and reduced morbidity [10]. Recent advancements include genetic testing for DICER1 mutations in suspected PPB cases [13]. Future directions include multicenter pediatric registries to develop standardized management protocols and better define recurrence risk [14].

CONCLUSION

Infantile spontaneous pneumothorax is frequently secondary to infectious, congenital, or neoplastic pathology. Persistent or recurrent air leak warrants advanced imaging and early surgical consultation. Recognition of etiologic diversity is essential for timely diagnosis and optimal outcomes.

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