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**Persistent acquired lobar overinflation complicating bronchopulmonary dysplasia**

Abstract Persistent acquired lobar overinflation (PALO) may complicate bronchopulmonary dysplasia (BPD). From infants admitted to the regional neonatal intensive care unit or who had been followed up at the chronic lung disease clinic in Liverpool over a 6.5-year period, 11 children with BPD and PALO were identified and details of their neonatal and subsequent outcome obtained. Their median gestational age was 29 weeks (range 24–33) and median birth weight was 1317 g (range 676–1968 g). All had received ventilatory support for severe neonatal respiratory distress syndrome for a median of 26 days (range 5–86). The median age the acquired lobar overinflation was detected was 82 days (range 45–424 days). Nine patients required continued neonatal or paediatric intensive care re-admission for deteriorating respiratory function. Six children have subsequently died at a median age of 9.5 months (range 6.5–20). Five patients underwent bronchoscopy, four suggesting the presence of bronchomalacia. Three patients had ventilation-perfusion scans all showing that the overinflated lobe had no mismatch defect unlike other areas of the lung.

**Conclusion** The place of specific therapies for persistent acquired lobar overinflation is unclear. Surgery to remove the overinflated lobe in such cases may be inappropriate and the outcome of this complication of bronchopulmonary dysplasia appears to be poor.

**Key words** Bronchopulmonary dysplasia · Lobar overinflation

**Abbreviations** 
BPD bronchopulmonary dysplasia · PALO persistent acquired lobar overinflation · PIE pulmonary interstitial emphysema

Introduction

A complication of bronchopulmonary dysplasia (BPD) is acquired lobar overinflation [1]. Diagnosis is made radiologically (Fig. 1) and pathogenesis probably includes barotrauma, oxygen toxicity and lung immaturity [4, 7, 11]. Compensatory overinflation could occur secondary to contralateral lung collapse, but it has not been demonstrated by regional lung function testing [13]. Partial intraluminal obstruction, for example by endobronchial granulomas and bronchial stenosis, may also play a role in the pathogenesis of acquired lobar overinflation [11]. In some infants bronchomalacia (dynamic collapse of the airways occurring principally during expiration) may contribute [5, 15].

Decompression of the overinflated lobe has been attempted by using steroids [12], high-frequency oscillatory ventilation [8, 14] independent lung ventilation [9] and physiotherapy. Lobectomy has been performed when other treatments have failed [1]. There little is known regarding the outcome of persistent acquired lobar overinflation (PALO) which is unresponsive to
treatment. We report here our experience of infants with PALO who have been managed in our unit.

Patients and methods

Radiological records of infants admitted to the regional NICU at the Liverpool Women’s Hospital, or who attended the chronic lung disease clinic at the Royal Liverpool Children’s Hospital between 1 April 1991 and 1 October 1997 were searched and infants with BPD and PALO identified. BPD was defined as a requirement for supplemental oxygen for at least 28 days and beyond 36 weeks gestation. PALO was defined as overinflation appearing in a previously radiologically normal lobe where the medial boundary of the lobe crossed the midline and the abnormality persisted for more than 1 month. Details of the neonatal and subsequent outcome of these infants were obtained from their medical records. Results of any respiratory investigations that had been performed were also recorded.

Results

From approximately 180 infants with BPD seen in the study period, 11 cases of BPD with PALO were identified. Their median gestation was 29 weeks (range 24–33 weeks) and their median birth weight was 1317 g (range 676–1968 g). The median maximum ventilatory peak inspiratory pressure used in the neonatal period was 32 cm H₂O and ten infants received a maximum inspired oxygen concentration of 1.0. All infants received surfactant and eight of the mothers received antenatal steroids. The median length of ventilation was 26 days (range 5–86 days). Eight infants developed pulmonary interstitial emphysema (PIE) in the neonatal period and two developed pneumothoraces (one bilateral).

The median age when the acquired lobar overinflation was detected was 82 days (range 45–424 days). This affected the right side in five cases. Treatments used after diagnosis included systemic steroids, mucolytics, physiotherapy, selective bronchial intubation and high frequency oscillatory ventilation.

Five children had a flexible fibre-optic bronchoscopy performed, four revealing severe dynamic airways collapse on inspiration, which was more severe on the overinflated side suggesting bronchomalacia. The airway collapse could be attenuated by increasing the end expiratory pressure in the ventilatory circuit (sometimes up to 20 cm H₂O being required). Three children (all of whose bronchoscopies revealed bronchomalacia) had ventilation-perfusion scans showing normal ventilation together with some perfusion of the affected lobe with mismatch defects in the other (collapsed) lobes (Fig. 2). Four children had computerised thoracic tomography enabling the overinflated lobe to be identified with certainty.

One child died on the NICU aged 11 months. Two children were transferred from the NICU to paediatric wards where their respiratory function deteriorated.

![Fig. 1 Chest radiograph of an infant with chronic lung disease showing persistent overinflation of the left upper lobe with herniation across the midline](image1)

![Fig. 2 (a) Tc99 perfusion lung scan of the infant in Figure 1 demonstrating some perfusion of the left upper lobe (overinflated on the plain radiograph), normal perfusion of the left lower lobe and impaired perfusion of the right upper lobe. (b) 81mKr ventilation lung scan demonstrating good ventilation of the left lung. There is a ventilation defect in the right lung](image2)