

Bronchopulmonary Dysplasia in a Premature Infant Case Report and Literature Review

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Abstract

Background: Bronchopulmonary dysplasia is an important cause of morbidity and mortality in premature infants. The aim of this study is to present a premature, extremely low birth weight infant with bronchopulmonary dysplasia.

Method: A review of the case records of a child with recurrent respiratory distress and the relevant literature.

Results: A preterm, extremely low birth weight baby (birth weight was 0.8 Kg), delivered by emergency caesarian section for previous caesarian section and prolonged rupture of fetal membranes at 27 weeks gestational age. She had spontaneous breathing at birth (APGAR scores were 8 in one minute and 10 in 5 minutes). She developed respiratory distress with cyanosis and became oxygen dependent from the second week of life. Examination revealed severe dyspnoea with grunting respiration, tachypnoea, cyanosis and crackles in the lung fields. Chest X-ray showed hyperinflation, right lower zone patchy consolidation with obliteration of the costophrenic angle. Echocardiography was however normal. She was successively managed with intermittent oxygen, dexamethasone, salbutamol and antibiotics (ceftriaxone). She was nursed in the incubator for 3 months. There was no episode of apneic attack throughout admission. She responded to treatment and was discharged home on intermittent oxygen therapy and nebulisation. The weight on discharge was 1.6kg. At 6 months of age, she is still having recurrent respiratory distress and supplemental oxygen at home. She is regular to follow up with recurrent episodes of wheeze requiring admissions.

Conclusion: Bronchopulmonary dysplasia should be suspected in a premature extremely low birth weight infant with early recurrent respiratory distress.

Key words: Bronchopulmonary dysplasia, prematurity, extreme low birth weight.

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Introduction

Bronchopulmonary dysplasia (BPD) is a form chronic lung disease that occurs most often in premature newborns who had severe lung disease at birth, such as respiratory distress syndrome, particularly in those who needed treatment with a ventilator for more than a few weeks after birth.¹ The condition affects approximately 10% and 40% of very low birth weight (less than or equal to 1,500 grams) and extremely low birth weight (less than or equal to 1,000 grams) infants, respectively.² The delicate tissues of the lungs can become injured when the air sacs are over-stretched by the ventilation or by high oxygen levels. As a result, the lungs become inflamed and additional fluid accumulates within the lungs. Full-term newborns that have lung disease (such as pneumonia) occasionally develop bronchopulmonary dysplasia.² Although a few newborns with very severe bronchopulmonary dysplasia die even after months of care, most newborns survive. Over several years, the lung injury heals. However, later these children are at increased risk of developing asthma and viral pneumonia.²

The need for improved survival has increased the prevalence of BPD, especially in small infants who may have been exposed to oxygen and positive pressure ventilation or in utero infection such as chorioamnionitis. This case report highlights clinical manifestation, diagnosis and treatment of bronchopulmonary dysplasia in an extremely low birth infant.

Case report

B.O is a preterm, extremely low birth weight baby (birth weight was 0.8 Kg), delivered by emergency caesarian section for previous caesarian section and prolonged rupture of fetal membranes at 27 weeks gestational age in a 6 six bedded private hospital in the southern part of Port-Harcourt metropolis. She had spontaneous breathing at birth (APGAR scores were 8 in one minute and 10 in 5 minutes). She developed respiratory distress with cyanosis and became oxygen dependent from the second week of life.

Examination revealed severe dyspnoea with grunting respiration, tachypnoea, cyanosis and crackles in the lung fields. She also had tachycardia and tender hepatomegaly. Chest X-ray showed hyperinflation, right lower zone patchy consolidation with obliteration of the costophrenic angle. Echocardiography was however normal. She was successively managed with intermittent oxygen, dexamethasone, salbutamol and ceftriaxone. She was transfused with sedimented blood cells at a packed cell volume of 20%. She was nursed in the incubator for 3 months. There was no episode of apneic attack throughout admission. She responded to treatment and was discharged home on intermittent oxygen therapy and nebulisation. The weight on discharge was 1.6kg. At 6 months of age, she is small for age (weight=4.5kg), still having respiratory distress and supplemental oxygen therapy at home. She is regular to follow up with repeated episodes of wheeze. She has had five previous hospital admissions following onset of wheeze and difficulty in breathing.

Discussion

Bronchopulmonary dysplasia (BPD) is a form of chronic lung disease that develops in preterm neonates treated with oxygen and positive-pressure ventilation (PPV).¹ BPD is most common in the most immature neonates born at 22-32 weeks' gestational age.³ These patients frequently weigh <1000 g at birth. Our patient was an extremely low birth preterm (birth weight= 0.8Kg) and was delivered at a gestational age of 27 weeks and became oxygen dependent on the second week of life.

She never had need for positive-pressure ventilation.

Injury to the lung as a result of oxygen toxicity, positive pressure ventilation and chorioamnionitis can result in clinically significant pulmonary dysfunction.^{2,4} This can interfere with alveolarization (septation), leading to alveolar simplification with a reduction in the overall surface area for gas exchange. Our patient was exposed to both intrauterine infection and oxygen therapy. It is possible that a combination of intrauterine infection and oxygen therapy with background respiratory distress syndrome may have been responsible for the occurrence of BPD in our patient.

Maternal cervical colonization and/or colonization in the neonate with *Ureaplasma urealyticum* has been implicated in the development of BPD.⁵ Viscardi and colleagues found that persistent lung infection with *U. urealyticum* may contribute to chronic inflammation and early fibrosis in the preterm lung, leading to pathology consistent with clinically significant BPD.⁵ Schelonka and colleagues summarized findings from 23 studies of *U. urealyticum* and concluded that infection with this organism is associated with increased rates of BPD.⁶

Infants with BPD have abnormal findings on physical examination, chest radiography, pulmonary function testing, and histopathology examination.⁷ Persistence of these abnormalities can be associated with an increased risk of BPD. In our series the abnormal physical examination findings were cyanosis, tachypnea, chest retractions, nasal flaring, and grunting. Chest radiographic findings were abnormal in our patient (consolidation on the right lung field with loss of costophrenic angle). Chest radiographs in BPD may demonstrate decreased lung volumes, areas of atelectasis and hyperinflation, pulmonary edema (PE), and pulmonary interstitial emphysema (PIE).⁷ Hyperinflation or interstitial abnormalities on chest radiograph appears to be correlated with the development of airway obstruction later in life.⁷ Chest radiograph in our patient showed bilateral patchy opacification with minimal right pleural effusion.

However there was no evidence of lung hyperinflation or interstitial abnormalities in our patient and this means that our patient may have less tendency of developing air way obstruction later in life. Arterial blood gases may reveal acidosis, hypercarbia, and hypoxia (with increased oxygen requirements). Blood gases estimation was not done for our patient because of lack of facility however our patient had evidence arterial desaturation (cyanosis).

Most recently, CT and MRI studies of infants with BPD have provided detailed images of the lung.⁸ High-resolution CT may detect radiographic abnormalities not readily identified with routine chest radiography.⁸ Echocardiographic assessment is an extremely valuable tool in confirming pulmonary hypertension and cor pulmonale which results from injury to the pulmonary circulation and contribute to the morbidity and mortality associated with severe BPD.⁹ In this series our patient had clinical evidence of cor pulmonale (tachypnea, tachycardia, tender hepatomegaly) but echocardiogram done was normal.

The diagnosis of bronchopulmonary dysplasia is made in the premature newborn who has received ventilation for a prolonged time and who has signs of respiratory distress and a prolonged need for supplemental oxygen.¹⁰

In our series diagnosis was based on the fact that she was born preterm extreme low birth weight with recurrent respiratory distress and prolonged need for supplemental oxygen.

Management of BPD is aimed at maintaining adequate oxygenation while trying to avoid barotraumas and oxygen toxicity. Oxygen is a potent pulmonary vasodilator that stimulates the production of nitric oxide (NO) which causes smooth muscle cells to relax by activating cyclic guanosine monophosphate.¹¹ Our patient responded to oxygen therapy. Other modes of treatment include administration of Steroids to improve pulmonary mechanics, bronchodilators which is particularly helpful when there is evidence of air trapping and use of broad spectrum antibiotics. These modes of treatment were beneficial to our patient. Also an infant with BPD requires increased calories to grow. Transfusion of packed RBCs may increase oxygen-carrying capacity in preterm infants who have anemia (hematocrit < 30%).¹² Our patient was transfused which significantly improved patients clinical status post transfusion.

Conclusion

Bronchopulmonary dysplasia is still a common cause of morbidity among ELBW infants and should be highly suspected in premature infants with early recurrent respiratory distress.

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