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Radiological Analysis of Bronchopulmonary Dysplasia*

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〈국문초록〉

기관지폐이형성증의 방사선 소견에 관한 연구

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유 은 주·연 경 모

기관지폐이형성증(Bronchopulmonary dysplasia)의 흉부방사선 소견은 그 시기에 따라 각기 다르게 나타나며 또한 폐렴과 같이 중복되어 보이므로 진단이 용이하지는 않다. 따라서 흉부방사선소견을 분석하여 특징을 알아보는데 목적이 있다. 기관지폐이형성증으로 추정되는 10명의 환자를 대상으로 출생시 체중, 조산의 정도, 산소 치료의 형태 및 기간과 폐음영의 시간 경과에 따른 변화의 양상 및 정도를 분석하였다. 임신 기간은 26.4주에서 31.3주(평균 28.5주)였으며 출생 체중은 825g에서 1.60kg(평균 1.16kg)이었다. 원인별 분석에서 초자막질환 4명, 무호흡 5명(신생아 무호흡 2명, 패혈증 2명, 신생아 가사 1명), 그 외 기타 원인이 있고 모두 기관삽입으로 산소(100%) 치료를 하였다. 산소 치료 기간은 12일에서 105일(평균 44일)이었다. 방사선 소견의 변화는 mechanical ventilation 시작후 대개 1－2주부터 나타나 진행을 보였고 18개월 이상의 추적이 가능한 3예에서는 호전되는 양상을 보였다. 주된 방사선 소견은 망상결절형 음영, 비정상적인 폐문주위와 간질의 선흔성 음영증가, 간질성 폐기종, 기포양, 폐기종 변화 등이 있으며 또한 폐렴합병에 의한 폐침윤, 폐엽무기폐, 기흉등이 같이 관찰되었다. 따라서 조산아에서 호흡곤란증으로 인하여 산소치료를 하는 경우 폐침윤이 계속될 때 기관지폐이형성증을 생각하여야 한다.

Index Words : Infants, newborn, repiratory system
Lung, abnormalities

Introduction

With improvements in medical management and increased survival of premature and low birth weight babies, new forms of chronic lung abnormality is frequently recognized.

In 1967, Northway et al. described a chronic lung disease that they named bronchoqulmonary dysplasia, basing their observation on newborn infant who received assisted ventilation for respiratory distress syndrome[1-2]. The definition or deagnostic criteria of the bronchoqulmonary dysplasia was changed over the past two decades[3-6].

With importance and increasing incidence of the bronchopulmonary dysplasia, we intended to describe the characteristics of the disease category through our experience by analyzing

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radiographic findings and describe the clinical characteristics.

Materials and methods

We retrospectively reviewed the chest radiographs of 10 infants, who were clinically diagnosed by bronchopulmonary dysplasia during November, 1988 to October, 1989.

They were diagnosed by following criteria (similar to those of Tooley) ; any premature infants who at the age of 30 days still requires supplemental oxygen and who also requires mechanical ventilation soon after birth, had a persistent abnormal chest X-ray. The exception was an infant who died at 13 days of age and in whom bronchopulmonary dysplasia was confirmed by autopsy.

We also collected the clinical data, such as birth weight, gestational age, Apgar score at 1 min. and at 5 min., maximum inspiratory pressure, duration of mechanical ventilation, maximum Fi02, duration of supplemental oxygen and presence of other clinical condition such as PDA (Table 1).

The infants were of 5 males and 5 females and the mean birth weight was 1160g (range from 825g to 1600g) and mean gestational age was 199 days (range ; 185-219 days).

In the 3 infants, chest X-ray follow-up was done over 2 years and lung biopsy was done in one patient and autopsies were performed in two of the three dead infants.

Results

On analysis of initial cause for the respiratory assistance, there are hyaline membrane disease in 4 cases, apnea in 5 cases and right lung hypoplasia with congestive heart failure in one case. In five cases of apnea, the apnea was due to apnea neonatorum 2 cases, sepsis 2 cases and birth asphyxia one case (Table 2). The method of the respiratory care was mechanical ventilation accompanied by the high concentration exygen therapy in all patients.

Initial chest X-rays of four hyaline menbrane disease patients revealed diffuse haziness with air-bronchogram and diffuse granular density,

Table 2. Cause of Initial Respiratory Insufficiency

Disease	No. of cases
Hyaline membrane disease	4
Apnea	5
Neonatal apnea	2
sepsis	2
Birth asphyxia	1
Rt. lung hypoplasia+heart failure	1
Total	10

Table 1. Clinical Data in Infants of BPD

case	sex	Bt.Wt. (g)	Gest.age (days)	Apgar 1min.	score 5min.	PIP (cmH_2O)	day's mech ventilator	max. FiO_2	day's of O_2	RDS	PDA
1	F	1040	190	4	5	20	5	0.8	58	-	
2	M	1150	190	0	2	20	53	0.6	58	-	+
3	F	1050	196	6	7	15	5	0.5	25	mild	+
4	M	1600	212	5	6	25	25	0.7	39	severe	
5	M	1540	219	7	8	25	10	0.8	37	severe	
6	F	1140	194	6	7	15	20	0.6	23	-	
7	M	1160	189	7	8	16	27	0.6	50	-	
8	F	870	185	7	9	18	17	0.6	32	-	
9	M	1230	207	6	8	40	57	0.8	105	-	+
10	F	825	210	2	5	40	12	1.0	12	severe	+
mean		1160	199	5	6.5	23	23.1	0.7	44		

but in the infants without RDS has no specific X–ray finding.

The interval from initiation of the ventilator therapy to initial X–ray change of broncopulmonary dysplasia was 3–25 days (mean 10 days) and the age of initial appearance of BPD was 7–30 days(Table 3). The case that the patient came to our hospital at 4 months of age with obscure previous clinical history was excluded.

The most common radiological findings were increased perihilar streaky density(8/10), emphysematous change (7/10) and other findings such as bubbly or small cystic change (3/10) and reticular or granular density (2/10) appeared (Table 4).

The interstitial emphysema was found in early stage of 3 cases but it was thought to be the complication of the therapy of RDS.

The associated other lung lesions were pneumonia in 4 cases, atelectasis in 4 cases and pneumothorax in 1 case. And other associated neonatal abnormalities were germinal matrix hemorrhage, retrolental ophthalmopathy of premature, patent ductus arteriosus, periventricular leukoencephalopathy and complex heart disease (Table 5).

In the 3 patients with long term follow–up, and they required frequent readmission with recurrent pneumonia but the lung abnormalities

Table 3. Time Sequences of Mechanical Ventilation and X–ray Change

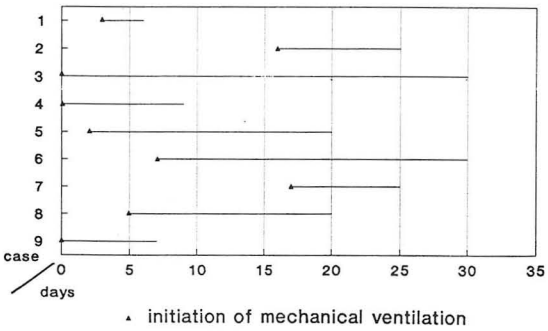

Table 4. Radiologic Findings of the Bronchopulmonary Dysplasia

X–ray findings	No. of cases (n=10)
Perihilar streaky density	8
Emphysematous change	7
Bubbly of small cystic appearance	3
Reticular and granular density	2

Table 5. Associated Clinical Abnormalities

Diseases	No. of cases
Germinal matrix hemorrhage	4
Retrolental ophthalmopathy of premature	4
Patent ductus arteriosus	4
Periventricular leukoencephalopathy	2
Complex heart disease	1

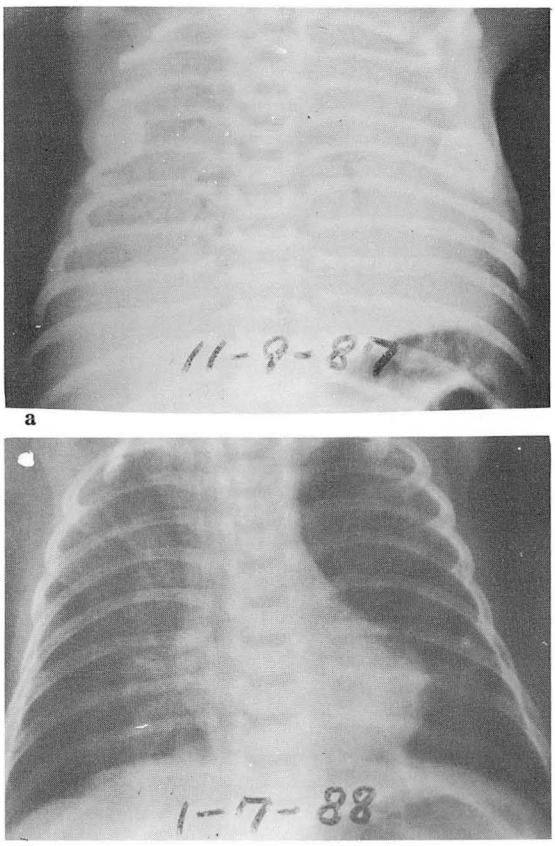

Fig. 1. Case 1. a. Initial chest X–ray showed severe hyaline membrane disease appearance. b. Follow–up after 2 months, demonstrated perihilar streaky density and hyperaeration.

were gradually improved.

Discussion

Since it's original description of Northway and collegues, bronchopulmonary dysplasia is thought to be a chronic lung disease of premature infants that was superimposed on the healing phase of hyaline membrane disease, and in the definition of the disease an abnormal chest X–ray was crucial component[1–6]. When BPD was first described, it was attributed to the adverse effects of oxygen on the immature lungs of premature infants who were being kept alive by mechanical ventilation[1,7,8]. BPD progressed through 4 stages as follows; stage I appearing identical to uncomplicated RDS, stage II being a period of pulmonary parenchymal opacity, stage III by cystic appearance of the lungs due to emphysematous foci and stage IV by strands of pulmonary fibrosis and increased lung volume[1–2]

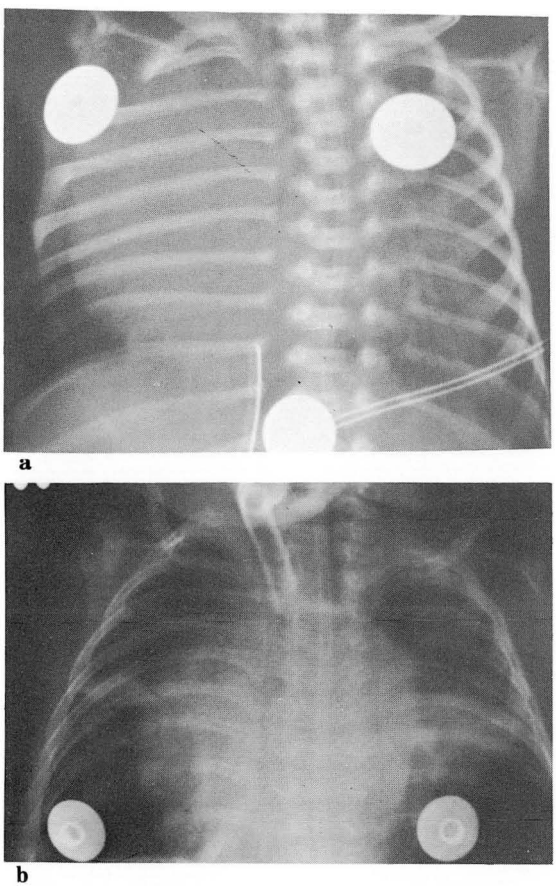

Fig. 2. Case 2. a. Initially this infant had no hyaline membrane disease but right lung hypoplasia and required long–term mechanical ventilation. In follow–up study (b) persistent streaky infiltration, segmental collapse and hyperaeration developed.

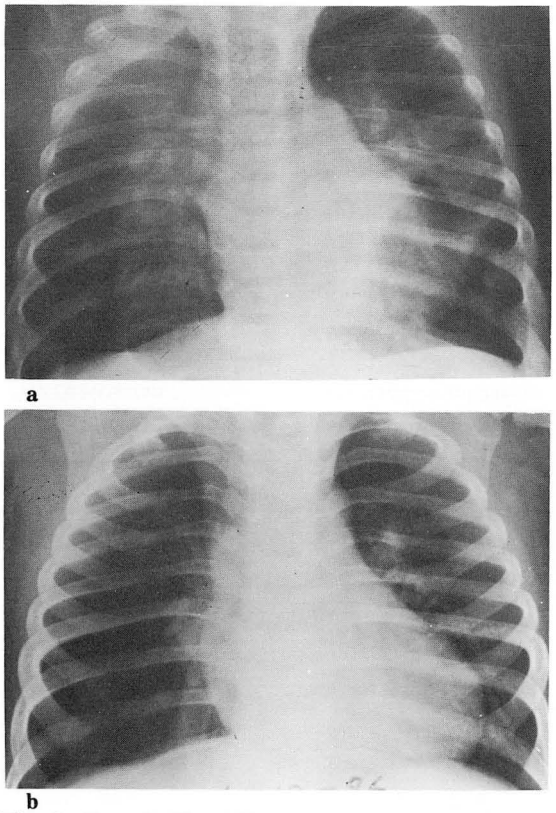

Fig. 3. Case 3. Chest X–ray at 6 months of age(a) showed stage IV BPD and markedly improved pulmonary infiltration in 15 months later(b).

It is well known that the classical description is not standard anymore and other authors suggested that the radiological feature and evolution of clinical BPD often varies from Northway's original description. And others reported the appearance of BPD noted in patients with wide variety of initial condition instead of hyaline membrane disease[9–10]

In our study with 10 premature babies, over

the half of the cases had not hyaline membrane disease but the needed the extensive ventilator care with high oxygen concentration. And the serial change of the chest X–ray was not followed the Northway's original description and most of our patients developed major features of BPD stage IV include considerable hyperaeration and very nonhomogeneous lungs with perihilar irregular streaks of density without passing through the early clinical or raiographic stage.

There are many reported series about high incidence of patent ductus arteriosus in infants with BPD. Brown et al. suggested that the presence of the PDA significantly increased the incidence of BPD in those infants at risk[11].

The prognosis or mortality of infants with BPD varies in different reports[12–14]. In the cases with the long term follow–up, the survivor tend to be improved gradually but the longest period of the follow–up is about 2 years and there is no detailed clinical information of long term outcome.

The diagnosis of the BPD in the acute phase has been based on radiographic criteria, but sometimes these are difficult to diagnose adequately. A variety of conditions simulate the BPD, particularyly on a single radiograph. The Wilson–Mikity syndrome may confused with BPD, possibly because both this syndrome and BPD tend to occur late in the course of premature infants, but the clinical course and the pathologic findings are different. Patients with Wilson–Mikity syndrome generally have an initial benign course and insidious radiologic abnormality and are not dependent to high oxygen or ventilator as BPD.

Similarly children with cystic fibrosis and Hamman–Rich syndrome has different clinical history and progression. And pulmonary lymphangiectasia is also very rare disease and characteristically causes respiratory difficulty at birth in a term infant and presents with a radiographic picture resembling stage III BPD. And other conditions could be differentiated with BPD because they reveal different clinical presentation[1,3].

Conclusively in premature infants with high oxygen and ventilator therapy the chronic persistent pulmonary abnormality with the sequence of preceding films highly suggests the possibility of bronchopulmonary dysplasia and the adequate management must be done.

REFERENCES

1. Northway WH, Rosan RC, Porter DY. Pulmonary disease following respirator therapy of hyaline membrane disease. N Engl J Med 1967 ; 276 : 357–368
2. Northway WH, Rosan RC. Radiographic features of pulmonary oxygen toxicity in the newborn : bronchopulmonary dysplasia. Radiology 1968 ; 91 : 49–58
3. Edwards DK. Radiographic aspect of bronchopulmonary dysplasia. J Pediatr 1979 ; 95 : 823–829
4. Bancalari E, Abdenour GE, Feller R. Bronchopulmonary dysplasia : clinical presentation. J Pediatr 1979 ; 95 : 819–823
5. Hyde I, English RE, Williams JD. The changing pattern of chronic lung disease of prematurity. Arch Dis Child 1989 ; 64 : 448–451
6. Heneghan MA, Sosulski R, Baquero JM. Persistent pulmonary abnormalities in newborns : the changing picture of bronchopulmonary dysplasia. Pediatr Radiol 1986 ; 16 : 180–184
7. Stern L. The role of respirators in the etiology and pathogenesis of bronchopulmonary dysplasia. J Pediat 1979 ; 95 : 867–869
8. Northway WH, Petriceks R, Canty E. Maturation as a factor in pulmonary oxygen toxicity : a preliminary report. J Pediatr 1979 ; 95 : 859–864
9. Escobedo MB, Gonzalez A. Bronchopulmonary dysplasia in the tiny infants. Clin Perinatol 1986 ; 13 : 315–325
10. Edwards DK, Jacob J, Gluik L. The immature

lung : Radiologic appearance, course and complications. AJR 1980 ; 135 : 659–666

11. Brown ER. Increased risk of bronchopulmonary dysplasia in infants with patent ductus arteriosus. J Pediatr 1979 ; 95 : 865–868
12. Mortensson W, Lindroth M. The course of bronchopulmonary dysplasia. Acta Radio 1986 ; 27 : 19–22
13. Mortensson W, Lindroth M, Jonsson B, et al. Chest radiography and pulmonary mechanics in ventilator treated low birth weight infants. Acta Radiol 1983 ; 24 : 71–79
14. Edwards DK, Dyer WM, Northway WH. Twelve year's experience with bronchopulmonary dysplasia. J Pediatr 1977 ; 59 : 839–846