

Clinical Characteristics of the Epidemic *Mycoplasma pneumoniae* Pneumonia Outbreak in 2003~2004

Hye-Oak Kwon, M.D., Shin-I Park, M.D. and Jun-Ho Lee, M.D.

*Department of Pediatrics, Bundang CHA General Hospital,
College of Medicine Pochon CHA University, Sungnam, Korea*

= Abstract =

Purpose : A wide, epidemic outbreak of *M. pneumoniae* pneumonia occurred throughout Korea in late 2003. Compared with previous years, the 2003 outbreak resulted in more severe cases and in an increased incidence of extrapulmonary symptoms and/or complications. We compared the clinical characteristics for *M. pneumoniae* pneumonia of 2003 to those of the past years.

Methods : One hundred six children diagnosed with *M. pneumoniae* pneumonia by serologic tests at Bundang Cha General Hospital between Aug 2003 to April 2004 were enrolled. Medical records were reviewed retrospectively, for clinical, laboratory and radiological aspect as well as complications. The pleural effusions of 3 patients who underwent thoracentesis were also analyzed.

Results : The duration of fever, cough, rhinorrhea, and sore throat was 8.2 ± 4.7 , 22.1 ± 4.8 , 8.4 ± 2.1 , 4.3 ± 1.2 days, respectively. The incidence (percentage) and duration of abdominal pain, vomiting, diarrhea, headache, skin rash, arthralgia was 5.1 ± 2.5 (21.9%), 3.4 ± 2.1 (17.1%), 4.3 ± 1.8 (16.2%), 3.5 ± 2.1 (14.4%), 5.5 ± 0.7 (5.9%) and 4.6 ± 1.3 days (4.9%), respectively. The mean duration of admission and treatment were 7.4 ± 4.3 days and 21.6 ± 11.1 days. Higher values of CRP and ESR on admission were positively correlated with the duration of fever and length of admission. The findings of pleural effusion were similar to those seen in TB pleurisy. Complications, including myocarditis (2 cases), arthritis (3 cases), vasculitis (5 cases), asthma (3 cases), ARDS (1 case), and DIC (2 cases) were observed in 14.1% of patients.

Conclusion : We found a number of characteristics of *M. pneumoniae* pneumonia among cases from late 2003 that were different from those of previous years. This outbreak resulted in more severe cases and in an increased incidence of extrapulmonary symptoms and/or complications. A multicenter study is needed to verify the changes in clinical characteristics observed during the 2003 outbreak from previous ones.

Key Words : *Mycoplasma pneumoniae* pneumonia, Epidemic outbreak

본 논문의 요지는 2004년 제54차 대한소아과학회 추계학술대회에서 포스터 발표하였음.

책임저자 : 이준호, 포천중문외과대학교 분당차병원 소아과

Tel : 031)780-5237, Fax : 031)780-5239, E-mail : Naesusana@yahoo.co.kr

Introduction

Mycoplasma pneumoniae is a respiratory pathogen that primarily causes bronchitis and pneumonia as well as pharyngitis, croup, and bronchiolitis.

Among school-aged children, *M. pneumoniae* is the most common causative organism of community-acquired pneumonia, and *M. pneumoniae* pneumonia is prevalent among even younger children who begin daycare at an earlier age^{1,7)}. While the sporadic spread of *M. pneumoniae* pneumonia occurs each year, epidemic outbreaks occur every 4~7 years. In Korea, epidemics of *M. pneumoniae* are known to occur every 3~5 years and data on the characteristics of these epidemics have been widely published. According to reports^{6,7)} from metropolitan areas prior to 1999, the occurrences of *M. pneumoniae* pneumonia occurred at 3-year intervals with durations of 1 year. The majority of studies showed that *M. pneumoniae* infections have a benign, uncomplicated clinical course and few complications. During late 2003 and early 2004, a nation-wide outbreak of *M. pneumoniae* infection occurred. Compared with the previous outbreaks, this one resulted in more severe cases and in an increased incidence of extrapulmonary symptoms and/or complications. Therefore, a retrospective study was undertaken to determine the clinical features of patients presenting to our hospital during the outbreak.

Materials and Methods

1. Subjects

A retrospective analysis of the medical records of 106 patients (Group 2) diagnosed with *M. pneumoniae* pneumonia and admitted to Bundang Cha General Hospital between Aug 2003 and April 2004 was done. Clinical characteristics were compared with 42 patients (Group 1) diagnosed with *M. pneumoniae* pneumonia from Jan 2002 to Dec 2002.

2. Diagnostic criteria

The diagnoses were confirmed by serological tests of mycoplasma antibody titers, which were measured using a Serodia-Myco II gelatin particle agglutinin test kit (Fujirebio Co., Japan), as an indirect hemagglutination test. The stated diagnostic criteria required that all of the following were present: ① respiratory symptoms, and ② either a single mycoplasma antibody titer rise to 1:320 or greater at the time of admission, or paired samples showing at least a 4-fold rise over a 1~2 week period.

3. Clinical, radiological and laboratory measurements

Medical records were reviewed retrospectively for: ① the duration of fever, respiratory symptoms, admission, and treatment, ② the incidence of extrapulmonary symptoms, and ③ the occurrence of complications.

Laboratory data such as CRP, ESR, leukocyte count, and mycoplasma antibody titers from blood samples taken both at the time of admission and follow up were evaluated. Radiological variables included the site of pulmonary infiltration shown on chest X-rays and the incidence of pleural effusion.

The pleural effusions of 3 patients who underwent thoracentesis were analyzed. In addition, our analysis included data from chest CT scans performed on 4 patients who had continuous rales on auscultation, over a 3 month period.

4. Statistical analysis

Data were expressed as mean \pm standard deviation. Statistical analysis was performed using SPSS software ver. 10.0 and Student's t-test. A *P*-value <0.05 was considered significant.

Results

1. Clinical manifestations

During this period, 78% of patients were children aged between 1 and 5 years, with the peak incidence at 4 to 5 years old. The mean age of enrolled patients was 50 ± 10 months, and the male to female ratio was 1.3 : 1. The outbreak was concentrated in the late fall and winter. These findings did not differ from those of previous studies. The patients were febrile in 83.5% of cases at the time of admission, and the mean duration of fever was 8.2 ± 2.7 days. Respiratory symptoms involved cough (96.2%), rhinorrhea (48.6%), sore throat (39.4%), and respiratory difficulty (12.3%) with durations of 22.1 ± 4.8 days, 8.4 ± 2.1 days, 4.3 ± 1.8 days, 3.8 ± 3.4 days, respectively (Table 2). The incidence of respiratory difficulty seemed to be higher and the duration of cough appeared to be longer than the previous year. Though macrolide was continuously prescribed in patients with a protracted cough of over 2 week's duration until the cough subsided, the extension of treatment duration did not seem to shorten the duration of cough. In 73.5% of patients,

extrapulmonary symptoms were observed, including abdominal pain (21.9%), vomiting (17.1%), diarrhea (16.2%) and the mean duration was 5.1 ± 2.5 days, 3.4 ± 2.1 days, 4.3 ± 1.8 days, respectively (Table 2). The appearance of skin eruption was variable and involved macules, maculopapular rash, urticaria, and petechiae. The mean duration of hospitalization was 7.4 ± 4.3 days, and the mean duration of treatment was 21.6 ± 11.1 days. All patients were treated with either roxithromycin (n=92) or clarithromycin (n=14). 17% of patients were initially treated with beta-lactam penicillin and/or third cephalosporin, and the prescription of macrolide was added after the diagnosis of *M. pneumoniae* pneumonia. Those without initial treatment of beta-lactam were started with macrolide. On auscultation, crackles (87.8%) and wheezing (21.5%) were audible, and decreased breath sounds were observed in 20.3% of the patients. We compared these data with those from the preceding year. The clinical and laboratory features are broadly similar. However, we found both the severity and duration of the symptoms to be much greater than those of preceding year (Table 1).

Table 1. Comparison of Clinical Features in Group 1 vs Group 2

Data	Group 1* (n=42)	Group 2† (n=106)	P-value
Prevalence‡	6.3% (42/660)	16.4% (106/645)	0.009
Mean age(year)	4.3 ± 2.7	4.1 ± 2.5	0.097
Admission days	4.5 ± 2.5 days	7.4 ± 4.3 days	0.043
Duration of fever	4.5 ± 3.1 days	8.2 ± 2.7 days	0.027
Treatment days	10.2 ± 3.6 days	21.6 ± 11.1 days	0.018
CRP (mg/dL)	3.3 ± 1.5	7.6 ± 3.8	0.039
Pleural effusion	4.7%	15.0%	0.015
Atelectasis	2.3%	14.9%	0.010
Extrapulmonary symptoms‡	19.0%	72.6%	0.014
Gastroenteritis	14.4%	17.9%	0.087
Hepatitis	2.3%	12.5%	0.028
Complications‡	7.1%	15.0%	0.030

*Patients admitted with *M. pneumoniae* pneumonia in 2002, †Patients enrolled in our study with *M. pneumoniae* pneumonia since late 2003, ‡Prevalence of *M. pneumoniae* pneumonia, §, ¶ Prevalence of data P value <0.05 means significant, statistically

Table 2. Frequency of Clinical Symptoms (N=106)

Symptoms	Numbers of patients (%)	Duration (days)*
Cough	102 (96.2)	22.1±4.8
Rhinorrhea	52 (48.6)	8.4±2.1
Sore throat	42 (39.4)	4.3±1.2
Diarrhea	34 (16.2)	4.3±1.8
Abdominal pain	23 (21.9)	5.1±2.5
Vomiting	18 (17.1)	3.4±2.1
Headache	15 (14.4)	3.5±2.1
Respiratory difficulty	13 (12.3)	3.8±3.4
Chest pain	9 (8.5)	2.1±2.5
Skin rash	6 (5.9)	5.5±0.7
Arthralgia	5 (4.9)	4.6±1.3

*Mean±standard deviation

2. Laboratory findings

At the time of admission, patients had a mean WBC count of $9,020 \pm 5,037 \text{ mm}^3$, a mean ESR of $57 \pm 35 \text{ mm/hr}$, and a mean CRP of $7.6 \pm 3.8 \text{ mg/dL}$ (Table 3).

Higher value of CRP and ESR upon admission were positively correlated with the duration of fever and length of admission but *Mycoplasma* antibody titers at the time of admission did not correlate with the duration of fever or with the duration of admission (Table 4). The pleural effusions of 3 patients in whom thoracentesis was performed were analyzed (Table 5). The color of pleural effusion was serosanguinous, and all profiles, except for protein and LDH were comparable to transudates. The protein level of pleural effusions ranged from 3.5 to 4.5 g/dL and caused hypoalbuminemia in the patients. The mean serum albumin level in patients with pleural effusion was 3.25 g/dL, and the mean serum protein was 4.05 g/dL.

3. Chest radiological findings

Pulmonary infiltration on chest X-ray was demonstrated in 88.7% of the patients; unilateral (62.2%), bilateral (33.0%), or concomitant with lobular consol-

Table 3. Laboratory Findings (N=106)

Finding	Number of patients (%)	Mean±SD*
WBC count(mm^3)		$9,020 \pm 5,037$
<5,000	4 (3.7)	
5,000~10,000	45 (43.2)	
10,000~15,000	50 (47.6)	
15,000~20,000	4 (3.7)	
>20,000	3 (2.8)	
Neutrophilia (>70%)	78 (73.5)	
ESR [†] , reactive (mm/hr)	79 (74.6)	57 ± 35
CRP [‡] , reactive (mg/dL)	71 (67.5)	7.6 ± 3.8

*Mean±standard deviation, [†]Erythrocyte sedimentation rate, [‡]C-reactive protein

Table 4. Relation between Laboratory Findings and Clinical Course

Finding	Admission days	Duration of fever
CRP (mg/dL)		
≤1.0	5.3 ± 1.2	3.2 ± 1.8
>1.0	8.3 ± 2.8 (<i>P</i> =0.015)	5.3 ± 2.7 (<i>P</i> =0.032)
ESR (mm/hr)		
≤20	5.5 ± 1.7	3.1 ± 2.9
>20	8.2 ± 2.7 (<i>P</i> =0.026)	5.2 ± 3.8 (<i>P</i> =0.043)
<i>Mycoplasma</i> antibody titer		
1 : 320~640	6.3 ± 3.1	2.7 ± 2.1
≥1 : 1,280	7.3 ± 4.8 (<i>P</i> =0.064)	4.1 ± 3.9 (<i>P</i> =0.056)

P value <0.05 means significant, statistically

idation (31.0%), atelectasis (14.9%), or pleural effusion (15.0%) (Table 6).

Patients with pleural effusion had a higher incidence of complications compared to those without pleural effusion. Chest CT scans were performed in 4 patients who had protracted cough and crackles or wheezing on auscultation 3 months after admission. All showed atelectasis, pleural thickening and adhesion. Two patients showed findings consistent with bronchiolitis obliterans, including bronchial wall

Table 5. Laboratory Findings of Pleural Fluid

	Case 1	Case 2	Case 3	Mean
PH	7.5	7.0	7.5	7.33
Color	Yellow	Yellow	Clear to yellow	—
Protein (g/dL)	4.5	3.8	3.5	3.93
LDH* (mg/dL)	4,758	1,718	1,379	2,618
ADA† (mg/dL)	29	83	38	50
WBC (/mm ³)	210	1,450	210	626.66
Glucose (mg/dL)	129	100	113	114
Gram stain	—	—	—	—
AFB stain	—	—	—	—
Culture	—	—	—	—
Tapping day‡	5 days	3 days	6days	4.6 days

*Lactic dehydrogenase (LDH), †Adenosine deaminase (ADA), ‡Means Hospital days

Table 6. Radiological Findings on Chest X-ray (N=106)

Site	Number of patients (%)
Unilateral	66 (62.2)
Right lobe	41 (38.6)
Upper	10 (9.4)
Middle	7 (6.6)
Lower	21 (19.8)
Parahilum	3 (2.8)
Left lobe	25 (23.6)
Upper	5 (4.7)
Lower	18 (16.9)
Parahilum	2 (1.9)
Bilateral	35 (33.0)
Atelectasis	15 (14.9)
Pleural effusion	16 (15.8)

thickening, adhesion and attenuation of pulmonary vessels, mosaic perfusion, and subsegmentally localized bronchiectasis.

4. Complications

Complications, including myocarditis (2 cases), arthritis (3 cases), vasculitis (5 cases), asthma (3 cases), ARDS (1 case), and DIC (2 cases), were observed in 15.0% of the patients (Table 7). We experienced some unusual complications related to *M. pneumoniae* pneumonia such as asthma, PIGN, coronary aneurysm and arthritis.

Discussion

Extrapulmonary manifestations of *M. pneumoniae* pneumonia are uncommon, with cases described as single reports or small series^{3,4}, because *M. pneumoniae* invades and multiplies in the bronchial epithelial cells¹. A variety of mechanisms have been suggested to explain the involvement of distant organ systems. Potential mechanisms include metastatic infection, autoimmunity, toxin generation, microthrombosis and altered host immunity^{4,5}.

In our study, we found some characteristics of *M. pneumoniae* pneumonia that were similar to those of previous years⁶⁻⁸. Compared with previous years^{6,7}, there were more cases requiring admission and the use of medication for longer periods due to prolonged fever, protracted cough or continuous rales on auscultation. There was also an increased incidence of extrapulmonary symptoms involving the skin, pleural cavity, bone marrow, G-I tract and joints, and of complications such as asthma, nephritis, arthritis, and vasculitis. Some of the complications that occurred in our study were extremely severe and unique, and to our knowledge, these complications have not been previously reported. However, there were no deaths in our study. In one

Table 7. Comparison of Frequencies of Complications in Group 1 vs Group 2

Disease	No of patients (%)		P-value
	Group 1* (n=42)	Group 2† (n=106)	
Pulmonary	3 (7.0)	37 (35.4)	0.032
Pleural effusion	2 (4.7)	16 (15.0)	
Atelectasis	1 (2.3)	15 (14.9)	
Asthma	0 (0.0)	3 (2.8)	
Bronchiolitis obliterans	0 (0.0)	2 (1.8)	
ARDS‡	0 (0.0)	1 (0.9)	
Extrapulmonary	6 (19.0)	54 (72.6)	0.014
Gastroenteritis	4 (14.4)	19 (17.9)	
Hepatitis	1 (2.3)	13 (12.5)	
Vasculitis	0 (0.0)	5 (4.7)	
Hemolytic anemia	0 (0.0)	4 (3.8)	
IDA [§]	1 (2.3)	4 (3.8)	
Arthritis	0 (0.0)	3 (2.8)	
Aseptic meningitis	0 (0.0)	2 (1.8)	
DIC	0 (0.0)	2 (1.8)	
Myocarditis	0 (0.0)	2 (1.8)	

*Patients admitted with *M. pneumoniae* pneumonia in 2002, †Patients enrolled in our study with *M. pneumoniae* pneumonia since late 2003, ‡Acute respiratory distress syndrome, §Iron deficiency anemia, ||Disseminated intravascular coagulation
P value 0.05 means significant, statistically

case, a 4-year-old boy was admitted with cough and gross hematuria. Chest X-ray showed pneumonic infiltration. The Mycoplasma antibody titer was 1 : 10,240 and C3 was 47 mg/dL. Urinalysis revealed blood (+), protein 2 (+), RBC (many/HPF), and WBC (10~30/HPF). An ultrasonogram of the kidney showed swelling and increased echogenicity in both kidneys. In other case, a 3-year-old boy was admitted with a persistent fever, cough and dyspnea. Chest X-rays showed pneumonic consolidation, accompanied by a profuse amount of pleural effusion. The Mycoplasma antibody titer was 1 : 40,960. Mild cardiomegaly was detected on a follow-up chest X-ray, and echocardiogram showed mild coronary dilatation and pericardial effusion. We excluded a case of concomitant Kawasaki disease because the patient's fever pattern was not remittent, was controlled with antipyretics, and other clinical features did not correspond to diagnostic criteria of Kawasaki

disease.

We compared the data collected in late 2003 to data from the preceding year (Table 1). In comparison with patients diagnosed with *M. pneumoniae* pneumonia at our hospital in 2002, there were remarkable increases in the incidence of mycoplasmal pneumonia, extrapulmonary symptoms, and complications in late 2003. In addition, clinical courses, including total febrile period, admission period and treatment period were significantly longer than those in 2002. In our survey, the estimated frequencies of mycoplasma pneumonia, extrapulmonary symptoms and complications were 16.4%, 73.5% and 15.0%, respectively, compared to those of 6.3%, 19.0% and 7.1%, respectively, in 2002.

Compared with previous studies, the incidence of extrapulmonary manifestation was remarkably higher in our study. Pyun et al.⁷⁾ reported an incidence of 25% in 1997 and Lee et al.⁸⁾ an incidence of 17%

in 1993. In our study, the most common extrapulmonary manifestation was gastrointestinal complication, occurring in 30.4% of patients. Elevated liver enzymes were observed in 12.5% of patients. Hepatic dysfunction in these patients was transitory, and recovery of normal liver function correlated directly with the resolution of the mycoplasma respiratory disease¹⁰. The pathogenesis of self-limiting hepatitis may be attributed to a direct cytolytic effect mediated by the infecting mycoplasma, or to an immunological autoimmune disorder resulting from the production of heterophil antibodies, etc.^{4, 10} In patients with persistent rales on auscultation, the incidence of extrapulmonary symptoms was higher, but this correlation could not be explained.

In evaluating laboratory results, we found that neither the level of the initial mycoplasma antibody titer at admission, nor the follow-up titer correlated with the duration of fever, length of admission, duration of medication, incidence of extrapulmonary symptoms or associated complications. However, the higher values of CRP and ESR at admission positively correlated with both the duration of fever and length of admission. There were no further positive correlations among the variables we evaluated.

We found that 33.0% of patients showed bilateral pulmonary infiltration on chest X-ray, a percentage that was much higher than 17% and 13% in previous years^{6, 8}. Pulmonary infiltrations in *M. pneumoniae* pneumonia patients were shown unilaterally in 70~90% of cases and bilaterally in 10~30% of cases and were more likely to occur in the sequence of right lower lobe, left lower lobe, and right upper lobe^{11, 12}. The prevalence of right lower lobe involvement can be attributed to exudate shift in the alveoli due to gravity and the anatomical disadvantage of the right bronchus, which is straighter and shorter than the left bronchus¹¹.

Chest HRCT was performed in 4 patients who had continuous crackles on auscultation over a 3-month period, even though the severity of crackles

had improved over time. In 2 of these patients, bronchiolitis obliterans was observed and was subsegmentally localized in small areas in the previously infiltrated lobe. Compared with previous years^{8, 9}, more patients in our study had pleural effusion (15.0%), though *M. pneumoniae* pneumonia is generally accompanied by small pleural effusion in approximately 20% of cases^{11, 13}. In analysis of the pleural effusion, the protein level, LDH and WBC counts corresponded to exudate, while the other values corresponded to transudate. Such findings are similar to that seen in TB pleurisy. The values of ADA in pleural effusions ranged from 29 to 83 in our study, and the value for 1 patient extended into the territory of suspected TB pleurisy. The only way to differentiate TB pleurisy is by collectively considering the Mantoux test, TB contact history, AFB staining, TB culture, and TB PCR^{14, 15}. Compared to patients without pleural effusion, *M. pneumoniae* pneumonia patients with accompanying pleural effusion are known to have a longer duration of illness, a higher incidence of complications, and a higher probability of co-infection with viral agents, such as the adenovirus and parainfluenza virus^{16~18}. In 9% of patients with persistent cough and severe continuing asthma symptoms, various viral studies were performed to check concomitant viral infection. Respiratory syncytial virus was found in one case.

M. pneumoniae infection is known to possibly exacerbate asthmatic symptoms and play a pathogenic role in asthma¹⁹. In our study, asthma was newly developed in 3 cases. The exacerbation of previously-diagnosed and treated asthma was observed in 5 cases. Possible causal mechanisms of mycoplasma-induced airway inflammation and hyperresponsiveness have been investigated, including increased Th2 responses and inflammatory neuropeptides^{19, 20}.

Complications of *M. pneumoniae* pneumonia largely fall into two categories those involving the respiratory system and those that are non-respiratory. Those with respiratory system involvement include

pleural effusion, lung abscess, Swyer-James syndrome¹³⁾, emphysema, bronchiolitis obliterans²¹⁾, bronchiectasis, etc. Those that are non-respiratory in nature include congestive heart failure, acute myocardial infarction, myocarditis²²⁾, pericarditis, Stevens-Johnson syndrome, encephalitis, aseptic meningitis, peripheral neuritis, cerebellar ataxia, transverse myelitis, Guillain-Barré syndrome, cranial nerve palsy, etc.^{13, 23)}. 33% of patients with CNS complications suffer permanent sequelae²³⁾. Among patients with CNS complications in our study, fortunately only 2 patients had aseptic meningitis and both recovered with no sequelae. According to reported studies, the occurrence of coronary aneurysm and PIGN, as complications of *M. pneumoniae* pneumonia, are rare. We believe that such complications implicate an autoimmune-mediated mechanism, such as that of vasculitis. The recent demonstration of tuberculin anergy and depressed T- and B-cell lymphocyte function during the course of *M. pneumoniae* infection suggests the possibility that immunosuppression may contribute to the pathogenesis of extrapulmonary complications^{4, 24)}.

We found a number of characteristics of *M. pneumoniae* pneumonia among cases from late 2003 that were different from those of previous years^{7~9)}. Furthermore, we propose the following hypotheses to explain why this outbreak was more clinically severe and accompanied by more frequent extrapulmonary symptoms and complications than outbreaks in previous years: First, it is likely that this epidemic outbreak was caused by a different subtype of *M. pneumoniae* from that of previous outbreaks. According to one report²⁵⁾, Group I (91.7%) was more prevalent than group II (8.3%) with a three-year cycle of epidemic outbreak from 1997 to 2002 in Korea. To date, there is little published data available to compare the immune responses, disease severity, and exchange phenomena between the two groups. Second, this epidemic outbreak may have occurred concomitantly with other viral infections, such

as adenovirus, influenza, parainfluenza, and rhinovirus. Third, the appearance of serious autoimmune diseases resulted from impaired and altered immunity in the host. That is, such an altered immune response could allow for escape of *M. pneumoniae* from the respiratory tract infection, the development of autoantibodies, prolonged infection, and perhaps the activation of a quiescent infection. Further studies are needed to substantiate the above hypotheses.

The findings in our study are subject to some limitations. First, this study was performed retrospectively. Second, case ascertainment was conducted only in our hospital. Third, determination of the beginning and end of the outbreak was not possible with the available data.

In conclusion, *M. pneumoniae* is generally thought to cause mild disease of the respiratory tract and uncomplicated pneumonia in affected children. However, serious pulmonary and extrapulmonary complications such as reported in our study may occur at any times. Knowledge of the less common pulmonary and systemic manifestations is important for differential diagnosis and institution of proper, early antimicrobial treatment and appropriate control measures including use of chemoprophylaxis during outbreaks of acute respiratory illness. A multicenter study is needed to verify the changes in clinical characteristics observed during the 2003 outbreak from previous outbreaks.

한 글 요 약

2003년 하반기에 유행한 *Mycoplasma pneumoniae* 폐렴의 특징에 대한 고찰

권혜옥 · 박신이 · 이준호

포천중문외과대학교 분당차병원 소아과

목 적 : 2003년 하반기 우리나라에서 전국적으로 마이코플라즈마 폐렴의 폭발적인 유행을 보였다.

그러나 예년과는 달리 심한 임상경과를 밟거나, 합병증과 폐외 증상을 동반하는 경우가 많이 관찰되었기에 저자들은 본원의 경험을 토대로 2003년 유행했던 마이코플라즈마 폐렴의 임상양상에 대해서 고찰해 보고자 한다.

방 법 : 2003년 8월부터 2004년 4월까지 분당차 병원 소아과에 폐렴증상으로 입원한 환아들 중, 입원 후 검사한 혈청 마이코플라즈마 항체가가 1:320 이상이거나 1주 간격으로 시행한 항체가가 4배 이상 증가가 있었던 환아 106명을 대상으로 후향적 고찰을 하였다.

결 과 : 총발열기간은 평균 8.2 ± 2.7 일, 입원 후 발열기간은 평균 5.3 ± 2.0 일이었다. 호흡기 증상으로는 기침(96.2%), 콧물(48.6%), 인후통(39.4%), 호흡곤란(12.3%) 등의 순이었으며 지속기간은 각각 평균 22.1 ± 4.8 일, 8.4 ± 2.1 일, 4.3 ± 1.2 일, 3.8 ± 3.4 일 등의 순이었다. 기침이 3개월까지 가는 경우도 소수에서 관찰되었으나, 치료기간과는 상관관계가 없었다. 비호흡기증상으로는 복통(21.9%), 구토(17.1%), 설사(16.2%), 두통(14.4%), 피부발진(5.9%), 관절통(4.9%) 등의 순이었다. 입원기간은 평균 7.4 ± 4.3 일이었으며 총치료기간은 21.6 ± 11.1 일이었다. 합병증으로 파종성 혈관 내 응고증(2명), 심근염(2명), 관절염(3명), 혈관염(5명), 천식(3명), 급성호흡부전(1명) 등이 관찰되었다.

결 론 : 2003년 하반기 우리나라에서 유행했던 마이코플라즈마 폐렴은 예년과는 달리 심한 임상경과를 보였고, 적지 않게 합병증을 동반하였다. *Mycoplasma pneumoniae* 아형의 종류에 따라 임상경과가 심해질 수 있는지는 아직 확실하지 않다. 단지, 다른 바이러스 감염과 동시 감염되는 경우 바이러스성 폐렴의 증세를 악화시키는 것으로 알려져 있다. 이번 유행과 다른 바이러스와 공동감염 관련여부는 본 연구에선 확인할 수 없었다. 소아에서 마이코플라즈마 폐렴이 심한 임상경과를 밟을 수도 있다는 것을 알아야 하겠다.

References

- 1) Bertman R, Kliegman R, Jenson H. Nelson Textbook of Pediatrics, 17th ed. Philadelphia : WB Saunders Co. 2004:990-2.
- 2) Jacobs E. Mycoplasma pneumoniae virulence factors and the immune response. Rev Med Microbiol 1991;2:83-90.
- 3) Marrie TJ. Community-acquired pneumonia. Clin Infect Dis 1994;18:501-13.
- 4) Fernald GW. Immunologic mechanisms suggested in the association of M. pneumoniae infection and extrapulmonary disease : a review. Yale J Biol Med 1983;56:475-9.
- 5) Feigin R, Cherr J. Textbook of Pediatric infectious disease, 3rd ed. Philadelphia : WB Saunders Co. 1992:1866-90.
- 6) Hong JY, Nah SY, Nam SG, Choi EH, Park JY, Lee HJ. Occurrence of Mycoplasma pneumoniae pneumonia in Seoul, Korea, from 1986 to 1995. J Korean Pediatr Soc 1997;40:607-13.
- 7) Pyun BY, Kim HH, Chung JT, Lee JS. A study as epidemiologic and clinical aspect of mycoplasma pneumoniae pneumonia during the last 5 years. Pediatr Allergy Respir Dis 1998;8:240-7.
- 8) Lee EK, Hong YJ, Lee MI, Ahn DH, Sohn KC. A clinical study on mycoplasma pneumoniae pneumonia. Pediatr Allergy Respir Dis 1993;3:11-9.
- 9) Kim BY, Lee HS, Kim IK, Choi CH, You KH. Clinical consideration between in type of pneumonia and cold agglutinin titer, and mycoplasma antibody titer caused by mycoplasma pneumoniae in children. J Korean Pediatr Soc 1993;36:959-67.
- 10) Squadrini F, Lami G, Pellegrino F, Pinelli G, Bavieri M, Fontana A, et al. Acute hepatitis complicating Mycoplasma pneumoniae infection. J Infect 1988;16:201-2.
- 11) Putamen CE, Curtis AM, Simenone JF, Jensen P. Mycoplasma pneumoniae : Clinical and roentgenographic patterns. AJR 1975;124:417-22.
- 12) Light RW. Pleural diseases, 4th ed. Philadelphia : Lippincott Williams and Wilkins. 2001:42-86.
- 13) Lind K. Manifestations and complications of Mycoplasma pneumoniae disease : a review.

1) Bertman R, Kliegman R, Jenson H. Nelson Textbook of Pediatrics, 17th ed. Philadelphia :

- Yale J Biol Med 1983;56:461-8.
- 14) Cassell GH, Cole BC. Mycoplasma as agents of human disease. N Engl J Med 1981;304:80-9.
 - 15) Klockars M, Kleemola M, Leinonen M, Koskela M. Serum adenosine deaminase in viral and bacterial pneumonia. Chest 1991;99:623-6.
 - 16) Chan ED. Fulminant mycoplasma pneumonia. West J Med 1995;162:133-42.
 - 17) Waris ME, Toikka P, Saarinen T, George RB, Zinskind M, Rasch J, et al. Diagnosis of Mycoplasma pneumoniae pneumonia in children. J Clin Microbiol 1998;36:3155-9.
 - 18) Shah DC, Muthiah MM. Adult respiratory distress syndrome due to mycoplasma pneumoniae. Postgrad Med J 1996;72:241-2.
 - 19) Biscardi S, Lorrot M, Marc E, Moulin F, Boutonnat-Faucher B, Helibronner C, et al. Mycoplasma pneumoniae and asthma in children. Clin Infect Dis 2004;38:1341-6.
 - 20) Johnston SL, Pattemore PK, Sanderson G, Mogabgab WJ, Forsgren M, Tunevall G, et al. Community study of role of viral infections in exacerbations of asthma in 9~11 year old children. BMJ 1995;310:1225-9.
 - 21) Prabhu MB, Barber D, Cockcroft DW. Bronchiolitis obliterans and mycoplasma pneumonia. Respir Med 1991;85:535-7.
 - 22) Sands MJ, Satz JE, Turner WE, Scoff LA. Pericarditis and perimyocarditis associated with active mycoplasma pneumoniae Infection. Ann Int Med 1977;86:544-8.
 - 23) Koskiniemi M. CNS manifestations associated with Mycoplasma pneumoniae infections : summary of cases at the University of Helsinki and review. Clin Infect Dis 1993;17:52-7.
 - 24) Sabato AR, Cooper DM, Thong YH. Transitory depression of immune fuction following Mycoplasma pneumoniae infection in children. Pediatr Res 1981;15:813-6.
 - 25) Kim SS, Kang H, Ahn BM, Lee WW, Kim ER, Kim SY, et al. Study of exchange phenomenon of mycoplasma pneumoniae in children from 1997~2002. Korean J Pediatr 2004;47:24-30.