[Case Reports]

A 22-YEAR-OLD WOMAN WITH FULMINANT CHLAMYDIA PNEUMONIAE PNEUMONIA

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Abstract: Chlamydia pneumoniae (C. pneumoniae) is a common pathogen of community-acquired pneumonia. The clinical features of infection caused by C. pneumoniae are usually mild and it does not progress into respiratory failure in young people. We describe a healthy, immunologically intact, 22-year-old woman with severe respiratory failure caused by C. pneumoniae accompanied by aspergillosis. The infection rapidly progressed and required mechanical ventilation. C. pneumoniae infection should be taken into account when treating patients with rapidly progressive pneumonia even in immunocompetent young adults.

Key words: Severe respiratory failure, aspergillosis, Chlamydia pneumoniae

INTRODUCTION

Majority of Chlamydia pneumoniae (C. pneumoniae) infections are mild1–3 and patients with severe C. pneumoniae pneumonia have mostly underlying diseases2–7. We describe a 22-year-old healthy woman with severe acute pneumonia caused by C. pneumoniae accompanied by aspergillosis.

CASE REPORT

A 22-year-old French woman, who had no complaints two days before, admitted to our hospital complaining general fatigue, high fever (39.4°C) and dyspnea at 9:00 on October 22. She had a bronchial asthma but had suffered no attack for last five years without corticosteroids. Coarse crackles were audible in bilateral lower lung fields. A chest X-ray (Fig. 1A) and computed tomography (CT; Fig. 2) showed...
Fig. 1. Chest radiograms (A) on admission (October 22, 1998) and (B) 17 hours later (October 23)
diffuse alveolar and interstitial infiltration shadow in both lung fields and bilateral pleural effusion. Erythrocytes sedimentation rate was 34 mm/hr, C-reactive protein 28.2 mg/dl, and white blood cell count 19,400/µl with neutrophilia (94.6%). Arterial blood gas analysis revealed hypoxia with 53.2 mmHg of PaO₂ and hypocapnia with 33.2 mmHg of PaCO₂. Electrocardiogram and ultrasonic echocardiogram were within normal limits.

We suspected *Mycoplasma, Chlamydia*, or Legionellosis and mixed bacterial infections, and started empiric antibiotic combination therapy with tetracycline (minocyclin; 200 mg/day) and carbapenem (panipenem; 2 g/day). Despite administrations of aminophylline, β-stimulant and oxygen, her hypoxia progressively worsened in several hours. We administered 1,000 mg of methylprednisolone, but PaO₂ further decreased to 45.1 mmHg at 22:30. We started mechanical ventilation at 22:50 with 100% FiO₂ and positive end-expiratory pressure (PEEP; 10 cmH₂O), and combined antifungal drug (fluconazole; 200 mg/day). However, at 2:30 on October 23, her PaO₂ decreased to 34.3 mmHg and a chest radiogram revealed an extension of the infiltration shadow to almost all lung fields (Fig. 1B). Both sputum and blood cultures for bacteria and mycobacterium were negative and a microscopic
examination of sputum revealed numerous neutrophils but no pathogenic microorganisms. She was given another dose of 1,000 mg of methylprednisolone, 20 mg of furosemide and 1 mg of epinephrine. After these treatments, PaO₂ turned to increase, and was 89.5 mmHg (100% FiO₂ and 10 cmH₂O of PEEP) at 9:20 on the same day. Thereafter, she quickly recovered. Artificial ventilation stopped on October 24, administration of antibiotics and antifungal drugs was discontinued on November 10, and the dose of corticosteroid was gradually decreased and stopped on November 17. All laboratory findings and a chest radiogram were normal on December 22.

We diagnosed the pneumonia caused by C. pneumoniae infection complicated with aspergillosis based on the following findings. IgA against C. pneumoniae was negative (0.34, negative<0.90) by enzymelinked immunosorbent assay; (ELISA)⁸⁻¹⁰ on October 22 but positive (1.78) on November 10. The rise of IgA greater than 1.00 was compatible with diagnostic criteria for acute C. pneumoniae infection⁹. IgG against C. pneumoniae was positive both on admission (0.92, negative<0.90) and on November 10 (0.90). Aspergillus antibody was negative on admission but positive on November 10. β-D-glucan, which is a marker of mycosis¹¹, was 105 pg/ml (normal<20) on admission and decreased to 7.4 pg/ml by November 4. Cryptococcus antigen, Candida antigen, antibodies to Trichosporon, Legionella, Mycoplasma, Cytomegalovirus and Influenza virus were all negative. Total IgE was high (820 U/ml) but specific IgE against Aspergillus was negative. CD4-positive lymphocyte count and CD8-positive lymphocyte count were normal on November 10.

DISCUSSION

Acute respiratory diseases caused by C. pneumoniae are usually not severe¹⁻⁷ but some cases of severe respiratory failure due to C. pneumoniae have been sporadically reported⁶,⁷,¹²⁻¹⁸. While almost patients with severe illness had underlying diseases²⁻⁷,¹²⁻¹⁶, three cases in healthy young adults were reported⁶,¹⁷,¹⁸. All of these cases developed multiorgan dysfunction syndrome or encephalitis and two of them did not recover completely. Compared with these cases, clinical course in our patient was more rapidly progressed but she quickly recovered.

Dalhoff K et al. described extrapulmonary rheumatologic involvement was seen in immunocompetent patients with C. pneumoniae infection compared with in immunocompromised cases¹⁹. In our patient, corticosteroid seemed to be effective and serum IgE was high, suggesting that an immunological reaction to C. pneumoniae would play a role on the development of severe respiratory failure. In addition, mixed infection of C. pneumoniae and aspergillosis might also have contributed to severe illness. A recent study described patients with C. pneumoniae pneumonia complicated with Streptococcus pneumoniae infection revealed more severe manifestations than patients with C. pneumoniae infection alone⁶.

In conclusion, C. pneumoniae infections should be considered as one possible
cause when treating patients with rapidly progressive severe pneumonia even in immunocompetent young adults, and corticosteroid pulse therapy would be effective in this situation.

REFERENCES
