

A case of a solitary mass shadow caused by *Mycoplasma pneumonia*

Shigehiro Tanaka¹⁾, Toru Aizawa¹⁾, Miho Yotsumoto¹⁾, Yoshio Koyanagi¹⁾,
Minoru Tokoro¹⁾, Kazuto Hirata²⁾, Hiroshi Fujiwara³⁾ and Shigeo Fujimoto³⁾

¹⁾*School of Letters Department of Health and Sports Sciences,
Mukogawa Women's University, Nishinomiya 663-8558, Japan*

²⁾*Department of Respiratory Medicine, Graduate School of Medicine,
Osaka City University, Osaka 545-8585, Japan*

³⁾*Department of Sport Medicine, Graduate School of Medicine,
Osaka City University, Osaka 545-8585, Japan*

Abstract

Mycoplasma pneumonia is a well-known cause of community-acquired pneumonia, and has a variety of appearances on radiographs of the chest. A 31-year-old man with a solitary mass on chest radiograph with dry cough visited our hospital. Transbronchial lung biopsy from the upper lung showed type II pneumocyte hyperplasia. We treated him with clarithromycin given the low probability of *Mycoplasma pneumonia*, resulting in improvement. *Mycoplasma pneumonia* can appear as solitary mass on chest radiographs.

Introduction

Diagnosis of a solitary mass shadow on chest radiographs is a common clinical problem. Neoplasm and tuberculosis are well-known causes of solitary mass shadows, and *Streptococcal pneumonia* can also exhibit a spherical mass¹⁾. On the other hand, *Mycoplasma pneumonia* (*M. pneumonia*), a community-acquired pneumonia, and has a variety of appearances on radiographs of the chest²⁾ and exhibits a variety of clinical features³⁾, but has few consistent pathological findings. We experienced the case of solitary mass shadow on chest radiographs.

Case report

A 31-year-old male visited our hospital be-

cause of dry cough and right-sided chest pain. He had been well until 10 days previously and had no history of smoking. His temperature was 36.7°C, and his pharynx was slightly injected without tonsillar exudates, his pulse was 72 beats/min, his blood pressure was 120/80 mm Hg, and no other abnormal findings were obtained on physical examination. Chest radiography (Fig. 1.) and computed tomography (Fig. 2-A,B) revealed a solitary mass in the right upper lung without mediastinal lymphadenopathy. Six months before at a regular health checkup, the result of these examinations were normal.

Abnormal blood examination findings included a white blood cell count of 10630/ μ l, c-reactive protein of 3.5mg/dl, and erythrocyte sedimentation rate of 40mm/hour. Serum liver and kidney function tests were normal. Bacterial culture of

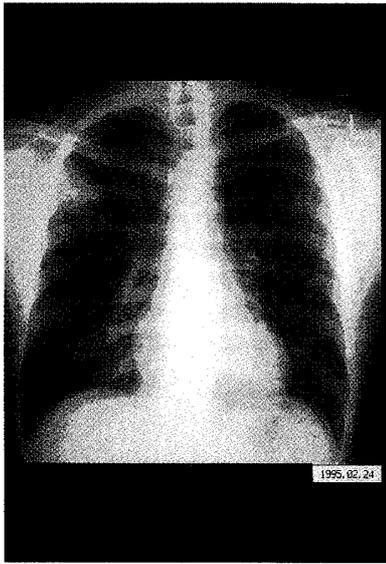


Fig. 1. Chest radiograph on initial visit revealing a mass shadow in the right upper lung

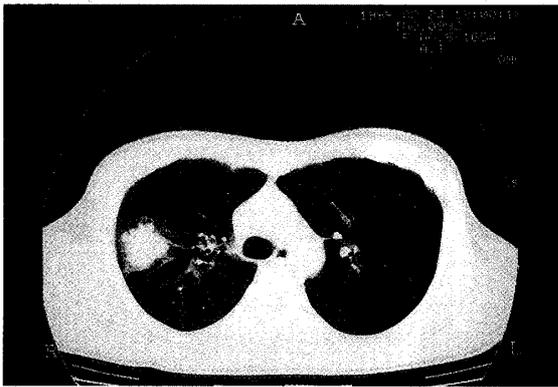


Fig. 2. A. Computed tomograph of the chest revealing a mass in the right upper lung. : pulmonary window

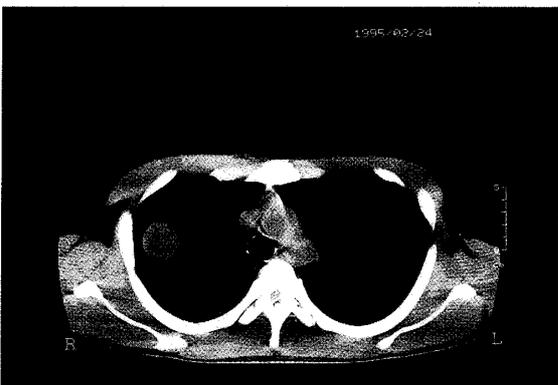


Fig. 2. B. Computed tomograph of the chest revealing a mass in the right upper lung. : mediastinal window

his sputum revealed normal flora. Repeated examination of his sputum by cytology and acid-fast staining revealed neither malignancy nor *Mycobacterium*. Examination of tissue from transbronchial tumor biopsy revealed that the alveolar walls were slightly thickened and had lymphocytic infiltrates without malignant cells or granulama (Fig. 3. A).

Almost all of the lining cells are stained with epithelial membrane antigen (Anti-Human Epithelial Membrane Antigen: DAKO A/S Grostrup, Denmark; Fig. 3. B) and anti-surfactant apoprotein A (Anti-Human Surface Apoprotein A: DAKO Japan Co., Ltd. Kyoto; Fig. 3. C).

Bacterial pneumonia could not be ruled out, and he therefore underwent antibacterial therapy with sparfloracin for ten days, but his cough and chest pain continued. At that time, it was strongly suspected that the mass shadow on his chest radiograph was caused by malignancy or tuberculosis. On the 16th day from his first visit to the hospital, clarithromycin was started since *M. pneumonia* and *Chlamydia pneumonia* were considered unlikely. After a week, his mass shadow was smaller, and it disappeared after a month due to treatment with clarithromycin for a total of twenty days (Fig. 4.).

Chlamydia IgA titer was < 8 . Passive hemagglutination (PHA)⁴⁾ titers to *M. pneumonia* were 160 (normal: < 40) on the 22nd day after first visit to the hospital, 40 on the 36th day, and < 40 on the 77th day, the cold hemagglutination titer was 64 on the 22nd day, 64 on the 36th day, and 8 on the 77th day, and c-reactive protein was $< 0.1\text{mg/dl}$ on the 36th day.

Discussion

Rose and Ward¹⁾ reported 21 cases of round pneumonia; in 9 of 17 cases in which culture was performed, *Streptococcus pneumonia* was obtained, while in the other 8 the results of culture were unknown. Round pneumonias, which are not rare, are thought to occur when an infiltrate featuring predominantly alveolar inflammation and exudation spreads from a small peripheral

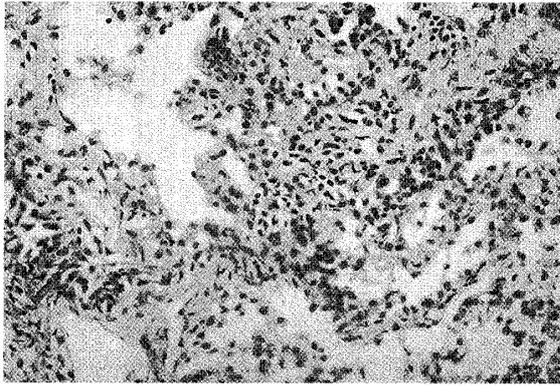


Fig. 3. A. Transbronchial lung biopsy from right S3 segment showing that the alveolar walls are slightly thickened and have lymphocytic infiltrates (Haematoxylin and eosin; original magnification $\times 200$)

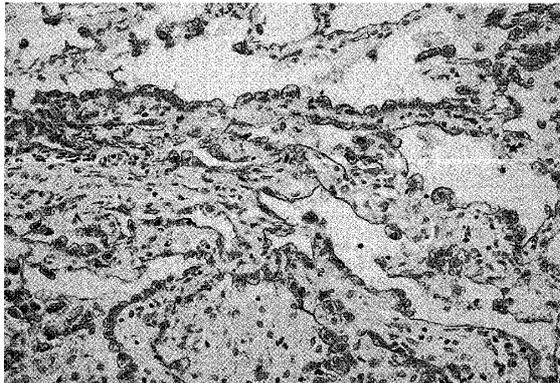


Fig. 3. B. Almost all of the lining cells are stained with epithelial membrane antigen (original magnification $\times 200$)

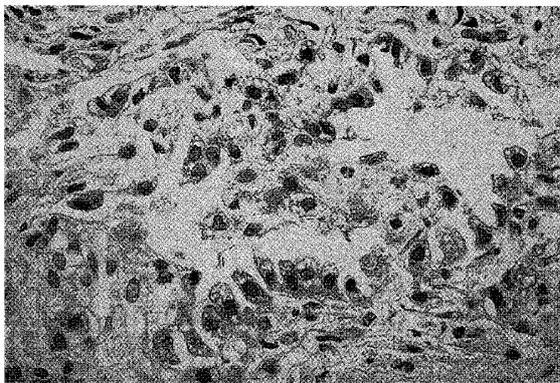


Fig. 3. C. Almost all of the lining cells are stained with anti-surfactant apoprotein A (original magnification $\times 400$).

alveolar locus centrifugally through the communicating channels, the pores of Kohn and channels of Lambert, accounting for an initial, nonsegmental distribution and a spherical shape^{1,5}). This description probably applies to *M. pneumoniae* as well. Radiographically, *M. pneumoniae* are confluent, patchy, nodular, or mixed, and they have no distinctive radiographic features²). In younger patients, *M. pneumoniae* can also cause lymphadenopathy²). *M. pneumoniae* occurs in patients with a wide age range from childhood to adulthood from 20 to 70 years of age, confirming the ability of *Mycoplasma* to produce disease in all age groups^{3,6}). *M. pneumoniae* with cough, fever, and chest radiographical abnormality is the cause of community-acquired pneumonia in 10–20% of all adult pneumonias^{2,3,6}). Serologic diagnosis of *Mycoplasma* infection requires a fourfold increase in complement fixation test results for paired serum samples or a titer of higher than 64 for a single sample^{2,6}).

On the 23rd day after his first visit to the hospital, the mass shadow on his chest radiograph was smaller, and the PHA titer was determined, given the probability of *M. pneumoniae*.

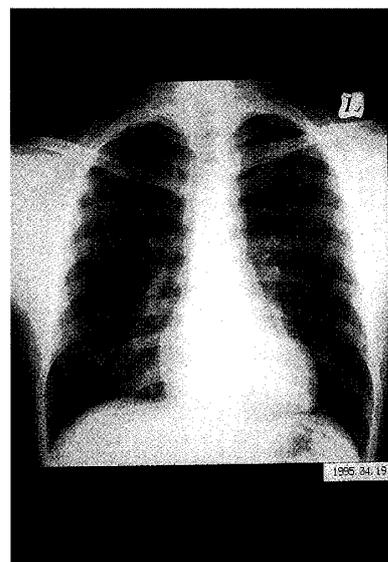


Fig. 4. Chest radiograph showing improvement of the mass shadow in the right upper lung.

We were unable to isolate the organism from throat specimens or sputum. The patient was followed up, and PHA titers demonstrated that he had *M. pneumoniae*. We used clarithromycin after sparfloxacin therapy, taking into consideration the efficacy of these agents for *Chlamydia pneumoniae*⁷⁾ and *M. avium*⁸⁾. In the treatment of *M. pneumoniae*, sparfloxacin may be effective⁹⁾, although in present case it did not show clear efficacy, while clarithromycin and erythromycin are equally effective⁶⁾. We made effort to detect specific DNA from species of bronchial biopsies by the polymerase chain reaction methods using specific primers of the *M. pneumoniae*. However, we could not find out the specific DNA from species kept in formaline solution. So we speculate that radiographical round formation (mass shadow) is allergic reaction.

It has been reported that the pathological findings of *M. pneumoniae* are bronchiolitis obliterans^{10),11)} interstitial pneumonitis^{10),11)}, or, in severe cases, diffuse alveolar damage and disseminated intravascular coagulation^{11),12)}. Furthermore, it has also been reported in electron microscopic studies that type II pneumocyte hyperplasia is found on open lung biopsy in patients with *M. pneumoniae*¹¹⁾. In normal lung tissue, type I and type II pneumocytes are stained with epithelial membrane antigen¹³⁾, and type II pneumocytes are stained with anti-surfactant apoprotein A restricted to the cytoplasm in the perinuclear region¹⁴⁾. Apoprotein A is synthesized in type II pneumocytes and associated intracellular and extracellular lamellar bodies¹⁵⁾. In our case, on transbronchial biopsy almost all hyperplastic type II pneumocytes were stained with anti-surfactant apoprotein A, which may inhibit plasma protein leakage¹⁶⁾ or stimulate inflammatory cytokine and immunoglobulin production¹⁷⁾. It has been reported that ECP derived from activated eosinophils might play a role in the pathogenesis of *M. pneumoniae*¹⁸⁾. So this case of a mass shadow in *M. pneumoniae* may be due to allergic reaction. However, our pathologic

findings are nonspecific, and further study is needed to clarify why cases of *M. pneumoniae*, especially those which prove fatal, exhibit a variety of pathological features.

M. pneumoniae infection can exhibit a variety of clinical manifestations and radiographical appearances. In rare cases, it can cause mass shadows on chest radiographs.

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References

- 1) Rose, R.W., Ward, B.H., *Radiology*, **106**, 179-182(1973)
- 2) Finnegan, C.C., Fowles, S.J., White, R.J., *Thorax*, **36**, 469-472(1981)
- 3) Murray, H.W., Masur, H., Senterfit, L.B., Roberts, R.B., *Am. J. Med.*, **58**, 229-242 (1975)
- 4) Sorodoc, G., Stoian, N., Ghyka, G.R., Peulescu, P., Osman, J., Puca, D., Caruntu, H., Toma, E., Dumineca, A., *Rev. Roum Med.-Virol.*, **27**, 51-53(1976)
- 5) Hershey, C.O., Panaro, V., *Arch. Intern. Med.*, **148**, 1155-1157(1988)
- 6) Cassell, G.H., Drnec, J., Waites, K.B., Pate, M.S., Duffy, L.B., Watson, H.L., McIntosh, J.C., *J. Antimicrob. Chemother.*, **27**(suppl), 47-59(1991)
- 7) Cooper, M.A., Baldwin, D., Matthews RS, Andrews JM, Wise, R., *J. Antimicrob. Chemother.*, **28**, 407-413(1991)
- 8) Fernandes, P.B., Hardy, D.J., Mcdaniel, D., Hanson, C.W., Swanson, R.N., *Antimicrob. Agents Chemother.*, **33**, 1531-1534(1989)
- 9) Kaku, M., Ishida, K., Irifune, K., Mizukane, R., Takemura, H., Yoshida, R., Tanaka, H., Usui, T., Tomono, K., Suyama, N., Kofa, H., Kohno, S., Hara, K., *An-*

- timicrob. Agents Chemother.*, **38**, 738-741 (1994)
- 10) Koletsky, R.J., Weinstein, A.J., *Am. Rev. Respir. Dis.*, **122**, 491-496(1980)
 - 11) Rollins, S., Colby, T., Clayton, F., *Arch. Pathol. Lab Med.*, **110**, 34-41(1986)
 - 12) Scully, R.E., Mark, E.J., McNeely, W.F., McNeely, B.U., *N Engl. J. Med.*, **326**, 324-336(1992)
 - 13) Nagata, N., Dairaku, M., Sueishi, K., Tanaka, K., *Am. J. Clin. Pathol.*, **88**, 552-559 (1987)
 - 14) Singh, G., Katyal, S.L., *Am. J. Pathol.*, **101**, 51-62(1980)
 - 15) Possmayer, F., *Am. Rev. Respir. Dis.*, **138**, 990-998(1988)
 - 16) Yukitake, K., Brown, C.L., Schlueter, M.A., *Pediatr. Res.*, **37**, 21-25(1994)
 - 17) Kremlev, S.G., Phelps, D.S., *Am. J. Physiol.*, **267**, L712-L719(1994)
 - 18) Yano, T., Saito, S., Arikawa, K., Kitazato, Y., Koga, H., Kumazawa, J., Honda, J., Oizumi, K., *Kansenshougaku-Zasshi*, **75**, 36-41(2001)