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CASE STUDY

Mycobacterium avium-intracellulare pleuritis with massive pleural effusion

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Mycobacterium avium-intracellulare *pleuritis with massive pleural effusion. Y. Okada, Y. Ichinose, K. Yamaguchi, M. Kanazawa, F. Yamasawa, T. Kawashiro. ©ERS Journals Ltd 1995.*

ABSTRACT: Atypical mycobacterial infection is seldom accompanied by pleural involvement. We report a very rare case of *Mycobacterium avium-intracellulare* pleuritis with massive pleural effusion.

The patient was a non-immunocompromised 35-year-old Japanese male with insidious onset of fever, chest pain and anorexia. The pleural effusion gradually resolved with empirical antimycobacterial treatment, leaving considerable pleural adhesion and thickening.

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Although the clinical features of atypical mycobacterial infection (AMI) resemble those of tuberculosis, pleural effusion is rare in cases of AMI [1, 2]. We report a case of *Mycobacterium avium-intracellulare* complex (MAC) pleuritis with massive pleural effusion. This has never been reported in non-immunocompromised patients.

Case report

The patient was a 35-year-old Japanese businessman. He was admitted to Keio University Hospital because of massive pleural effusion in the left thorax. Three months before admission he experienced general malaise, low grade fever, and discomfort in the left chest. One month before admission he noticed dull pain in the left lower chest.

The patient was slightly ill-nourished. His temperature was 38.6°C, blood pressure was 120/80 mmHg, and the pulse was regular, 84 beats·min^{-1}. He breathed regularly, 16 breaths·min^{-1}, without difficulty. Respiratory sounds were remarkably diminished on the left side. On chest percussion, there was dullness on the left side below the frontal second intercostal space. No symptoms or signs suggesting immunodeficiency were noted.

The erythrocyte sedimentation rate was 25 mm·h^{-1}. C reactive protein (CRP) was strongly positive. White blood cell count was 4.8×10^{9}·L^{-1} (4,800·cells·mm^{-3}). Fibrinogen was increased, 477 mg·dL^{-1}. Total protein was 7.0 g·dL^{-1}, with decreased albumin (55%) and increased γ-globulin (22%) fractions. Human immunodeficiency virus (HIV) antibody was negative. A purified protein derivative (PPD) skin test was positive. Repeated microbiological examinations of sputum were negative.

On chest roentgenogram, massive pleural effusion was observed in the left thorax (fig. 1). The effusion was yellowish clear with specific gravity 1.021. Cytological examination of the effusion did not show malignant cells.

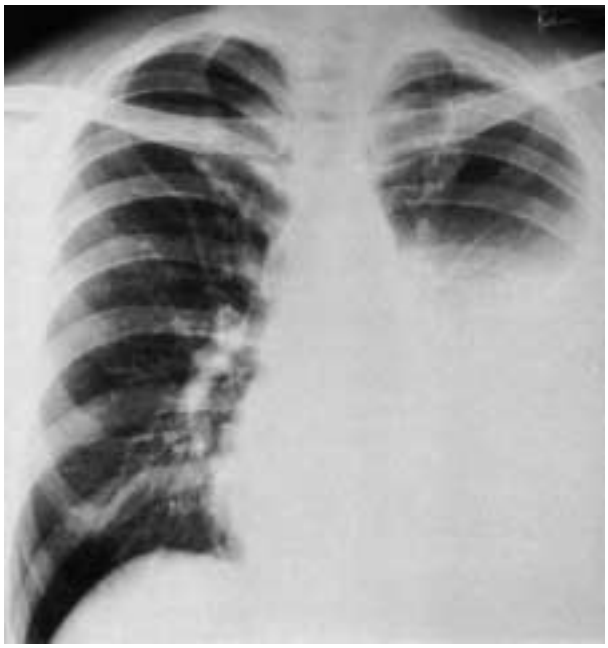

Fig. 1. – Posteroanterior chest roentgenogram at the time of admission. There was massive pleural effusion in the left thorax. Apparent intrapulmonary lesion was not recognized.

Biochemical analysis of the effusion was performed: protein 5.4 g·dL^{-1}, lactate dehydrogenase (LDH) 1,954 U·L^{-1} (serum LDH 285 U·L^{-1}), Rivalta reaction positive, and glucose 4 mg·dL^{-1}. Microbiological examinations, staining and culture of the effusion, were negative except for the mycobacterial culture.

Based on the clinical findings, tuberculous pleuritis was strongly suspected and the treatment was started with isoniazid 300 mg·day^{-1}, rifampin 450 mg·day^{-1} and streptomycin 750 mg, twice weekly. Subsequently, the laboratory reported to us that many colonies of mycobacterium were cultured from the effusion sample. The growing microorganism was later identified as MAC (table 1) [3]. The strain was resistant to most of the anti-tuberculous drugs *in vitro*; however, it was sensitive to high concentrations of streptomycin, isoniazid and ethionamide. Gradual improvement in the clinical features was observed. The patient was discharged free of symptoms after 4.5 months admission followed by a further 3 months treatment with unchanged regimen at the out-patient clinic, without recurrence. On chest roentgenogram, the pleural effusion was completely absorbed, although considerable adhesion and thickening remained (fig. 2).

Table 1. – Results of microbiological investigations

Temperature range for growth	22–42°C
Growth rate	slow (> 2 weeks)
Photochromogen	(-)
Colony pigmentation	(-)
Niacin test	(-)
Catalase - 68°C	(+)
Tween-80 hydrolysis - 1 week	(-)
Arylsulphatase - 3 days	(-)
Nitrate reduction	(-)

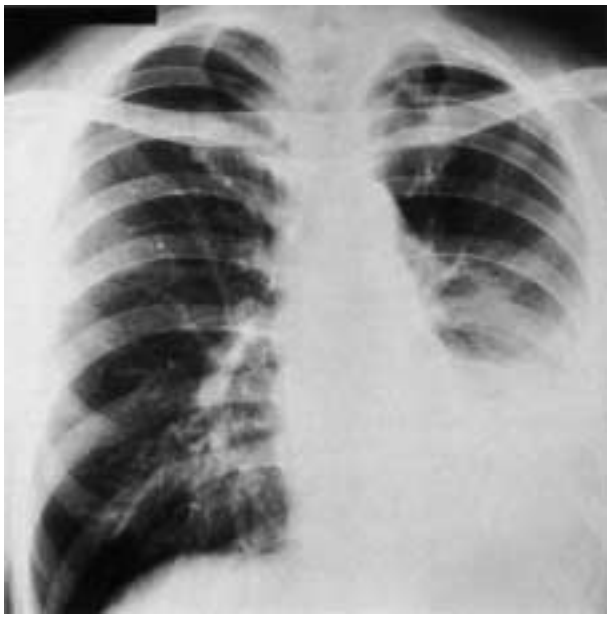

Fig. 2. – Posteroanterior chest roentgenogram at the time of discharge. Pleural effusion had been absorbed, but considerable adhesion and thickening of the pleura remained. The mediastinum was shifted to the left.

Discussion

It is generally assumed that when an atypical mycobacterium is isolated from a closed space, it is responsible for the observed pathological changes. However, an atypical mycobacterium can be isolated from pleural effusion unrelated to AMI, such as in congestive heart failure and metastatic cancer [4]. In the present case, the patient did not have such an underlying disease. The clinical features of this case were typical of pleuritis. Because we detected only MAC from a closed space, *i.e.* from the pleural space, and did not detect any pathogenic microorganism at all from sputum, we diagnosed the condition of this patient as pleuritis caused by MAC. Coexistence of immunodeficiency, such as acquired immunodeficiency syndrome (AIDS), was excluded. The prognosis was good. Pleural involvement without HIV infection was reported in a patient with disseminated MAC infection [5]. However, primary MAC pleuritis without disseminated infection is extremely rare. One probable explanation of the unusually massive retention of pleural effusion in the present case is that the patient had not been treated for approximately 3 months from the onset of the pleuritis. The elevated LDH and low glucose in the effusion suggested a strong and prolonged inflammatory process that might cause pleural thickening. The positive result of the PPD skin test is not inconsistent with MAC infection. It is accounted for by the following facts; 1) most Japanese have received bacille Calmette-Guérin (BCG) vaccination and, therefore, show a positive PPD reaction; and 2) atypical and tuberculous mycobacteria have a shared surface antigen that can cause a cross-reaction of the PPD test [6].

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