

Severe mycoplasma pneumonia

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Holt, S., Ryan, W. F., and Epstein, E. J. (1977). Thorax, 32, 112-115. Severe mycoplasma pneumonia. A patient who developed a protracted illness following severe mycoplasma pneumonia is described. The acute phase of the infection was complicated by myocarditis and haemolytic anaemia. The respiratory symptoms abated and lung function tests improved with the administration of systemic and inhaled corticosteroids.

Mycoplasma pneumoniae, first isolated in 1944 by passage in laboratory animals and chick embryo, has been implicated as an important cause of respiratory infection, sometimes producing a wide variety of non-respiratory syndromes (Eaton *et al.*, 1944; Lambert, 1969). This organism usually causes mild self-limiting respiratory disease and has been shown by serological studies to have a wide geographical prevalence (Hayflick and Chanock, 1965).

Only about 10% of patients with mycoplasma infection will develop major respiratory disease, the features of which are not distinct enough to allow an accurate diagnosis without recourse to serological studies (Mufson *et al.*, 1961). However, unlike other causes of primary atypical pneumonia, untreated mycoplasma pneumonia may give rise to a prolonged respiratory illness with persistent radiographic shadows and respiratory symptoms (Mufson *et al.*, 1961). Complications of mycoplasma infection tend to be uncommon, few severe infections having been described.

A patient with severe mycoplasma pneumonia, complicated by myocarditis and haemolytic anaemia, is described. The infection gave rise to prolonged respiratory disability which responded to treatment with corticosteroids.

Case report

A 50-year-old housewife was admitted with a three-day history of cough, productive of a small amount of mucoid sputum, breathlessness, general malaise, and fever. Examination revealed a pyrexia of 40°C, and there were signs of consolidation in both lower zones of the lung fields. The pulse was 80 per minute and regular, blood-pressure 120/80 mmHg, and cardiac auscultation

was normal. Treatment was started with ampicillin, 500 mg six-hourly by intramuscular injection. On admission the haemoglobin was 11.5 g/dl, white cell count $6.2 \times 10^9/l$ with 90% polymorphs and an ESR of 92. A chest radiograph (Figure) demonstrated patchy consolidation in both lower zones of the lung fields with a small effusion at the left base.

Three days after admission the patient's general condition deteriorated with clinical and radiological evidence of increased consolidation in both lung fields and extension to the right upper zone. Streptomycin, 0.5 g by intramuscular injection twice daily, was added to the treatment with apparent slight improvement. Five days after admission the haemoglobin was recorded at 11 g/dl, white cell count $11.6 \times 10^9/l$ with 90% polymorphs. The serum showed a complement fixation antibody titre of 1 in 1280 against *Mycoplasma pneumoniae*. Cold agglutinins were present and reported as follows:

- with adult 0 positive red cells 1 in 1000
- with patient's own red cells 1 in 1000
- with 0 positive cord red blood cells 1 in 32.

The direct Coombs' test was positive at 1 in 500.

These results indicated mycoplasma pneumonia with cold agglutinin disease of anti-I specificity. Tetracycline was begun in a dose of 1 g orally every six hours, and the ampicillin was discontinued on the sixth day after admission. Her general condition improved over the next 48 hours, but again deterioration occurred on the eighth day when she developed central cyanosis, tachypnoea, and a pyrexia of 39°C. Blood gases were reported as pH 7.42, P_{CO_2} 6.5 kPa, and P_{O_2} 5 kPa with a bicarbonate of 29.4 mmol/l. Improvement occurred with controlled oxygen therapy. On the 10th day she complained of bilateral pleuritic

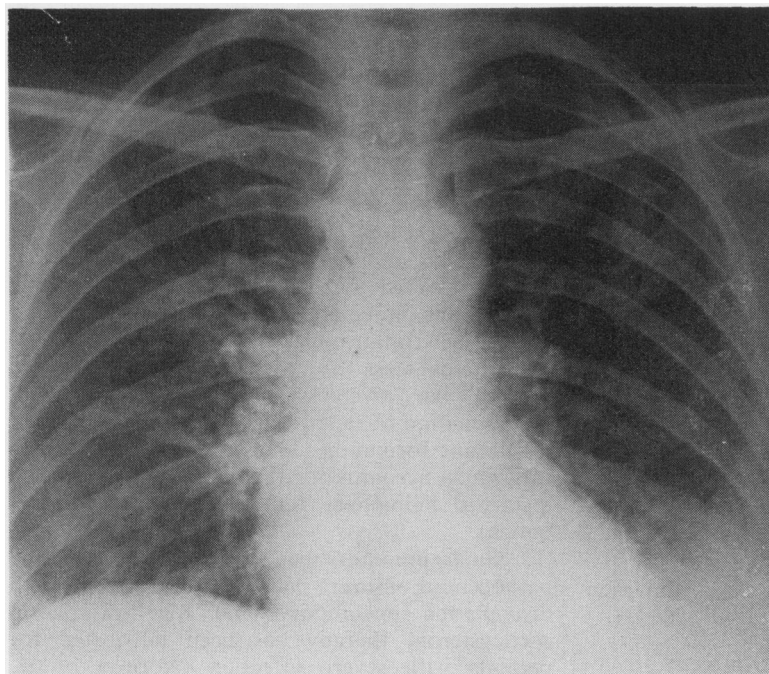


Figure Chest radiograph showing patchy consolidation at both lung bases with a small effusion at the left base.

chest pain and developed a pleural friction rub over both lung fields. A chest radiograph showed persistent bilateral lower zone consolidation. However, over the next three days, with continued supportive therapy, she made a good improvement with a return of the blood gases to normal. Sputum and a throat swab failed to grow any organism in a wide range of primary and secondary tissue cultures.

On the 17th day after admission an electrocardiogram showed sinus rhythm at a rate of 80 per minute with generalised ST-segment arching and T-wave inversion in leads I, II, III, aVL, aVF, and V1 to V6. These ECG abnormalities had resolved within one week; no pericardial friction sounds were heard and there was no rise in transaminase enzymes. On the 25th day, the haemoglobin had fallen to 10 g/dl with 3% reticulocytes, and the red cells showed auto-agglutination.

She maintained good progress and was mobilised and allowed home after 49 days in hospital. On discharge she still had an irritant non-productive cough, breathlessness on moderate exertion, and coarse crepitations in both lower zones of the lung fields.

FOLLOW-UP STUDIES

At the outpatient clinic one month after discharge

from hospital she complained of a non-productive cough and dyspnoea. Examination revealed coarse crepitations over both lower lung fields. Haemoglobin was 13.2 g/dl, white cell count $8.7 \times 10^9/l$, and ESR 20 mm. A further one month's course of tetracycline, 500 mg every six hours, was prescribed. At her next monthly attendance she continued to complain of a troublesome cough disturbing her sleep, breathlessness when walking quickly, and intermittent bouts of wheezing. Examination demonstrated coarse inspiratory crepitations and rhonchi at both lung bases with a short wheeze on forced expiration. Respiratory function tests showed a mild restrictive impairment of ventilatory capacity with a small lung volume and reduced transfer factor (see Table). A chest radiograph revealed fine shadowing in both lower lung fields and pleural thickening at the left base.

In view of these findings a persistent 'alveolitis' was suspected and treatment was started with Becotide inhalations, 2 four times daily, and prednisolone, 5 mg once daily. Over the ensuing two months the cough and dyspnoea were relieved and lung function tests demonstrated some improvement (see Table). The steroid therapy was gradually tailed off with no recurrence of her symptoms. The patient remained well with clear lung fields on examination 12 months after the

Table Respiratory function tests

	After onset of illness		
	15 weeks	23 weeks	1 year
Ventilatory capacity			
Forced vital capacity (litres)	2.7	2.95	3.0
Forced expiratory volume (l s) (litres)	1.95	2.3	2.3
FEV as % of VC	72%	78%	77%
Max. voluntary ventilation (l/min)	74	88	83
Lung volumes (litres)			
Vital capacity	2.6		2.85
Inspiratory capacity	2.0		2.2
Expiratory reserve volume	0.6		0.65
Functional residual capacity	1.42		2.6
Residual volume	0.82		1.95
Total lung capacity	3.42		4.8
RV/TLC ratio	24%		41%
Transfer factor (kPa)	1.73		3.3
	(55% normal)		(107% normal)

onset of the illness. A chest radiograph then demonstrated residual pleural thickening at the left costophrenic angle and respiratory function tests showed further improvement (see Table).

Discussion

Mycoplasma pneumoniae infection usually gives rise to a mild respiratory illness and, in many cases, the attack may be subclinical (Lambert, 1969). Serological studies have shown a high incidence of complement-fixing antibodies against mycoplasma in the population. In one survey it was estimated that 19% of the population in southern England may possess such antibody (Lambert, 1968). When mycoplasma pneumonia is complicated by the development of cold agglutinins, as in this patient, then the clinical picture tends to be that of major respiratory involvement. Occasional severe, complicated infections have been described, mainly in patients with evidence of depressed immunity (Foy *et al.*, 1973). The present case report is of interest because of the occurrence of two uncommon complications during the acute phase of major respiratory disease and the persistence of respiratory symptoms during convalescence.

It has been observed that a small proportion of patients with *Mycoplasma pneumoniae* infection may continue to complain of cough and dyspnoea with associated impairment of gas transfer factor (Jones, 1969). On the whole, symptoms of mycoplasma pneumonia disappear early in the convalescent period, but physical signs and radiographic abnormalities may persist. In a study of 109 cases of mycoplasma pneumonia, 22% of

patients had abnormal radiographs one month after the onset of illness, but these had cleared within a further six weeks (Mufson *et al.*, 1961).

Because *Mycoplasma pneumoniae* infection is rarely fatal, opportunities to study pathological changes in the lungs have been few. The capacity for producing prolonged respiratory illness, as in our patient, with impairment of ventilatory capacity and gas transfer factor suggests the presence, in some cases, of a persistent pneumonitis. Observed changes in the lungs during mycoplasma infection include lymphocyte and plasma cell infiltration with purulent exudation, and microabscess formation of bronchiolar distribution. An alveolitis may also occur with desquamation of septal cells, oedema, and hyaline membrane formation (Jones, 1969) and it may be this which accounts for the protracted illness and disturbed pulmonary function tests in our own patient.

Cellular immunity has been implicated in the pathogenesis of severe pneumonitis in patients with mycoplasma pneumonia, and for this reason corticosteroid therapy has been advocated for patients with severe infection (Noriega *et al.*, 1974). This may provide an explanation for the good therapeutic response that was obtained by their use in our patient.

Haemolytic anaemia and myocarditis are well recognised but uncommon consequences of mycoplasma infection (Worlledge and Blajchman, 1972; Lewes *et al.*, 1974). The severity of haemolysis may be wide but is clinically apparent in less than 5% of patients (Jones, 1969). In this patient only a mild asymptomatic haemolytic anaemia occurred. It is also of interest that myocarditis with marked ECG changes was present without cardiac symptoms. It has been suggested that *Mycoplasma pneumoniae* infection may be a common cause of symptomless myocarditis (Lewes *et al.*, 1974).

Although chest radiographic findings tend to be more marked in mycoplasma pneumonia as compared with other causes of primary atypical pneumonia, no distinctive radiological features of mycoplasma infection can be identified that will allow an accurate aetiological diagnosis (Mufson *et al.*, 1961). Severe bilateral involvement of the lungs with extension to the upper lobes, as occurred in this present case, is uncommon in mycoplasma pneumonia and reflects the severity of this infection. The presence of residual pleural thickening is also of interest and has rarely been described. Mufson *et al.* (1963) reported a patient similar to this case in whom pleural thickening

was present on the right side one year after the onset of infection.

Tetracycline is well established in the treatment of *Mycoplasma pneumoniae* infection and has been shown to accelerate clinical recovery and radiographic clearing (Kingston *et al.*, 1961). The role of corticosteroids in the management of fulminant infections (Noriega *et al.*, 1974) and protracted convalescent illness, as in this present patient, has yet to be defined. However, the good therapeutic response obtained by inhalational and low-dose systemic corticosteroid treatment suggests a possible role for these drugs in the management of persistent respiratory disability following mycoplasma pneumonia.

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