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 6.2×10⁹/1 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 11.6×10⁹/1 with 90% polymorphs. The serum showed a complement fixation anti-body 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, Pco₂ 6.5 kPa, and Po₂ 5 kPa with a bicarbonate of 29.4 mmol/l. Improvement occurred with controlled oxygen therapy. On the 10th day she complained of bilateral pleuritis.
Severe mycoplasma pneumonia

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 8·7×10⁹/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
Ventilatory capacity
Lung
Respiratory transfer factor
dence of complement-fixing antibodies
demonstrated residual pleural
southern England
Mycoplasm,a pneumoniae infection
cases,
mycoplasma in the
tinins,
(Lambert, 1968). When mycoplasma
was
and
of
case
sent
been
of
during convalescence.
pneumonia
continue
associated
period,
(Jones, 1969).
graphic abnormalities
cases
of
impairment
sent
19% of the
population.
many
complain
of
of
major
respiratory illness.
Mycoplasma pneumoniae infection maybe
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 com-
mon cause of symptomless myocarditis (Lewet
et al., 1974).

Although chest radiographic findings tend to be
more marked in mycoplasma pneumonia as com-
pared with other causes of primary atypical
pneumonia, no distinctive radiological features of
mycoplasma infection can be identified that will
allow an accurate aetiologcal 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

**Table Respiratory function tests**

<table>
<thead>
<tr>
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<th>After onset of illness</th>
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<tbody>
<tr>
<td></td>
<td>15 weeks</td>
</tr>
<tr>
<td>Ventilatory capacity</td>
<td></td>
</tr>
<tr>
<td>Forced vital capacity (litres)</td>
<td>2·7</td>
</tr>
<tr>
<td>Forced expiratory volume (l/s)</td>
<td>1·95</td>
</tr>
<tr>
<td>FEV as % of VC</td>
<td>72%</td>
</tr>
<tr>
<td>Max. voluntary ventilation (l/min)</td>
<td>74</td>
</tr>
<tr>
<td>Lung volumes (litres)</td>
<td></td>
</tr>
<tr>
<td>Vital capacity</td>
<td>2·6</td>
</tr>
<tr>
<td>Inspiratory capacity</td>
<td>2·0</td>
</tr>
<tr>
<td>Expiratory reserve volume</td>
<td>0·6</td>
</tr>
<tr>
<td>Functional residual capacity</td>
<td>1·42</td>
</tr>
<tr>
<td>Residual volume</td>
<td>0·82</td>
</tr>
<tr>
<td>Total lung capacity</td>
<td>3·42</td>
</tr>
<tr>
<td>RV/TLC ratio</td>
<td>24%</td>
</tr>
<tr>
<td>Transfer factor (kPa)</td>
<td>1·73</td>
</tr>
<tr>
<td></td>
<td>(55%</td>
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<td></td>
<td>normal</td>
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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 inci-
dence 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 agglu-
tinins, 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
depressed immunity (Foy and Lambert, 1968). 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 radi-
ographic 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 that
presence, in some cases, of a persistent pneumo-
nitis. Observed changes in the lungs during
mycoplasma infection include lymphocyte and
plasma cell infiltration with purulent exudation
and microabscess formation of bronchial distri-
bution. An alveolitis may also occur with
desquamation of septal cells, oedema, and hyalling
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 myco-
plasma infection (Worledge and Blajchman,
1972; Lewes *et al*., 1974). The severity of haemo-
lysis 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
in patients with marked ECG changes was present without
cardiac symptoms. It has been suggested that
*Mycoplasma pneumoniae* infection may be a com-
mon cause of symptomless myocarditis (Lewet
et al., 1974).
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.

**References**


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